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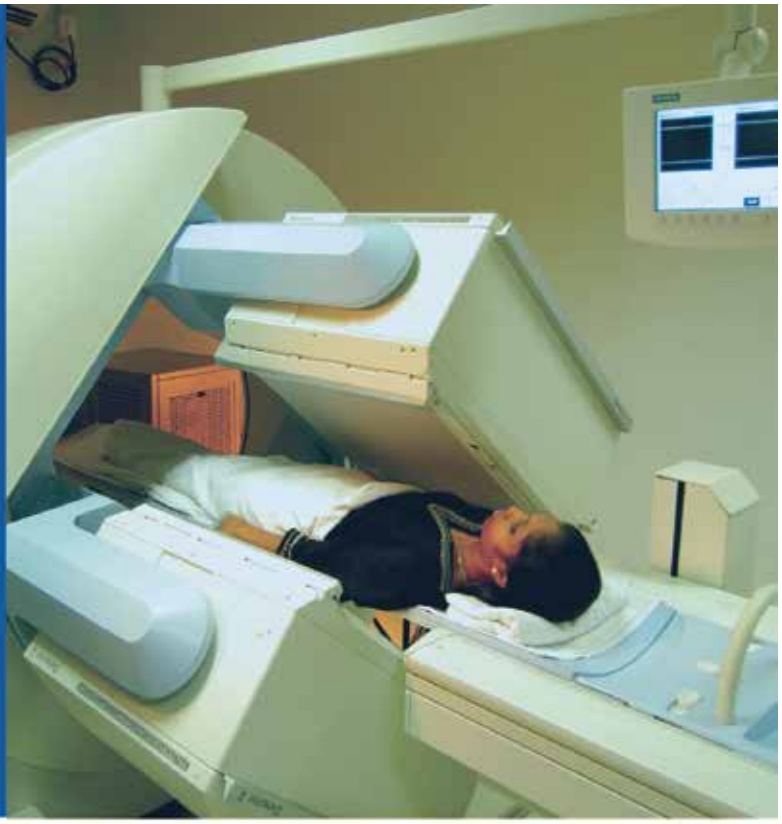

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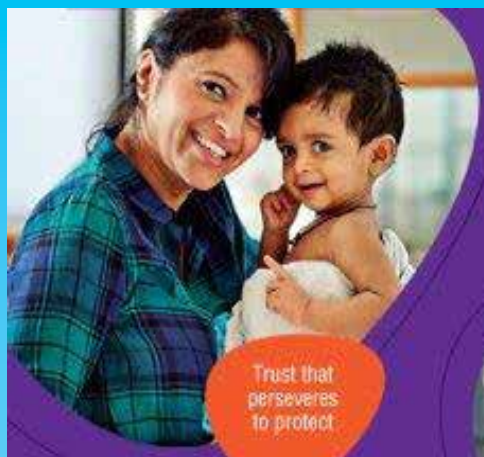

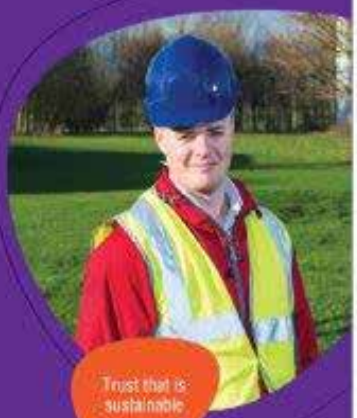

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AUGMENTIN 156 mg suspension: Bottles of powder for the preparation of four thousand suspension.

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Indication

AUGMENTIN is an antibiotic agent with a notably broad spectrum of activity against the commonly occurring bacterial pathogens in general practice and hospital. The β-lactamase inhibitory action of clavulanic extends the spectrum of amoxicillin to embrace a wider range of organisms including many resistant to other β-lactam antibiotics.

AUGMENTIN should be used in accordance with local official antibiotic-prescribing guidelines and local susceptibility data.

AUGMENTIN oral preparations for three times daily dosing are indicated for short-term treatment of bacterial infections in the following cases.

Upper respiratory tract infections (including ENT) e.g. tonsillitis, sinusitis, otitis media.

Lower respiratory tract infections e.g. acute exacerbation of chronic bronchitis, lobar and bronchopneumonia.

Genito-urinary tract infections e.g. cystitis, urethritis, pyelonephritis.

Skin and soft tissue infections, e.g. boils, abscesses, cellulitis, wound infections.

Bone and joint infections e.g. osteomyelitis.

Dental infections e.g. dentoalveolar abscess.

Other infections e.g. intra-abdominal sepsis.

Susceptibility to AUGMENTIN will vary with geography and time (see Pharmacological Properties, Pharmacodynamics for further information). Local susceptibility data should be consulted where available, and microbiological sampling and susceptibility testing performed where necessary.

Infections caused by amoxicillin -susceptible organisms are amenable to AUGMENTIN treatment due to its amoxicillin content. Mixed infections caused by amoxicillin -susceptible organisms in combination with AUGMENTIN -susceptible β-lactamase producing organisms may therefore be treated with AUGMENTIN.

Dosage and Administration

Usual dosages for the treatment of infection in Adults and children over 12 years

Mild to Moderate infections: One AUGMENTIN 375 mg tablet 3 times a day. Severe infections: One AUGMENTIN 625 mg tablet 3 times a day. Where the 625 mg tablet is not available, a dose of two AUGMENTIN 375 mg tablets 3 times a day may be taken. Therapy can be started parenterally and continued with an oral preparation.

Children:

The usual recommended daily dosage is 25mg/kg/day* to divided doses every eight hours. The table below presents guidance for children. Under 1 year 25 mg/kg/day, for example 7.5 kg child would require 2 ml AUGMENTIN 156 mg suspension 3 times a day. 1-4 years 5 ml AUGMENTIN 156 mg (10-18 kg) suspension 3 times a day. Over 5 years 5 ml AUGMENTIN 312 mg (18-40 kg) suspension 3 times a day.

In more severe infections the dosage may be increased up to 50 mg/kg/day in divided doses every eight hours.

* Each 25 mg AUGMENTIN provides 20 mg amoxicillin and 5 mg clavulanic.

AUGMENTIN 375 mg and 625 mg tablets are not recommended in children of 12 years and under.

Dosage in dental infections (e.g. dentoalveolar abscess)

Adults and children over 12 years: One AUGMENTIN 375 mg tablet 3 times a day for five days.

Contraindications

AUGMENTIN is contra-indicated in patients with a history of hypersensitivity to beta-lactams, e.g. penicillins and cephalosporins.

AUGMENTIN is contra-indicated in patients with a previous history of AUGMENTIN-associated jaundice/hepatic dysfunction.

Warnings and Precautions

Before initiating therapy with AUGMENTIN, careful enquiry should be made concerning previous hypersensitivity reactions to penicillins, cephalosporins, or other allergens.

Serious and occasionally fatal hypersensitivity (anaphylactoid) reactions have been reported in patients on penicillin therapy. These reactions are more likely to occur in individuals with a history of penicillin hypersensitivity (see Contraindications).

Interactions

Concomitant use of probenecid is not recommended. Probenecid decreases the renal tubular secretion of amoxicillin. Concomitant use with AUGMENTIN may result in increased and prolonged blood levels of amoxicillin but not of clavulanate.

Concomitant use of allopurinol during treatment with amoxicillin can increase the likelihood of allergic skin reactions. There are no data on the concomitant use of AUGMENTIN and allopurinol.

Pregnancy and Lactation

Reproduction studies in animals (mice and rats) with orally and parenterally administered AUGMENTIN have shown no teratogenic effects. In a single study in women with pre-term, premature rupture of the fetal membrane (pPROM), it was reported that prophylactic treatment with AUGMENTIN may be associated with an increased risk of occurring necrotising enterocolitis in neonates. As with all antibiotics, use should be avoided in pregnancy, especially during the first trimester, unless considered essential by the physician.

AUGMENTIN may be administered during the period of lactation. While the completion of the risk of sensitization associated with the excretion of trace quantities in breast milk, there are no detrimental effects for the infant.

Overdose

Gastrointestinal symptoms and disturbance of the fluid and electrolyte balances may be evident. Gastrointestinal symptoms may be treated symptomatically with attention to the water electrolyte balance.

Amoxicillin crystalluria in some cases leading to renal failure has been observed (see Warnings and Precautions).

AUGMENTIN can be removed from the circulation by haemodialysis.

Version number: GDS21/IPI/2

Date of issue: 30 January 2015

Date of preparation: 05/06/2015

For more information please refer the full product information.

AUGMENTIN is a trademark of the GlaxoSmithKline group of companies.

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Source : IMS TSA MAT FEB'15. **1.** Current Allergy & Clinical Immunology 2001;14:14-16. **2.** Drugs 2000; 59(2): 301-321. **3.** Expert Opin. Drug Metab. Toxicol. 2009; 5(7):813-822.

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President's Message

It is with great pleasure I take this opportunity to be able to express my sincere appreciation to the IMPA in publishing the 2017 Journal (Volume 11) again this year.

I hope the IMPA would be able to publish this journal annually and if possible more frequently.

I wish to thank the editor Prof. Joel Fernando along with the members of the editorial board for having devoted much of their valuable time and efforts in compiling this journal which supports IMPA members to receive and express their opinion on several topics of significance specially in the field of medicine.

I wish to thank all those who have submitted articles for this journal. I also wish to thank our administrative officer Ms. Champa Silva for her untiring efforts in coordinating all the work required in printing this journal.

I appreciate the efforts of our printer AK2PRO for obliging us always in producing this excellent journal.

Finally I thank the sponsors and advertisers for all the support and assistance provided to publish this journal and wish the IMPA the very best in all the future activities.

Dr.A.H.A.Hazari

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Content

WITH BEST COMPLIMENTS

FROM

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Editorial

Private sector is taking an increasingly important role in delivery of health services. Private sector provided fifty percent of outpatient care and five percent of inpatient care in the year 2000. The share of inpatient care is relatively small in comparison to the public sector ninety five percent. However it is a well known fact that patients visiting public sector hospitals are frequently requested by specialists to obtain drugs and laboratory services from the private sector. The impact of current operations of the private sector on the public sector needs to be studied.

Health policies implemented by governments in the nineties gave due place to promoting the private sector. Specialist based private hospital services were encouraged hopefully expecting to ease the overburdened government hospital sector. However there has been no reduction in the use of government hospital services. Instead it has opened up new opportunities in the private sector to provide direct access to government specialists without a proper referral system. Patients pay more for a distorted specialist dominated primary care. The role of government in private sector health service development must extend beyond mere promotional activities. The government must give direction to private sector growth to achieve health policy goals that will improve equitably the health of the entire population. Growth of the private sector does not diminish the overriding importance of the government and public sector as determining agents of national health policy. Private sector growth poses a challenge to the public sector. Existing policy framework, information systems and organizational structures of the ministry of health do not yet respond fully to the challenges and opportunities offered by an expanding private sector.

Persons seeking private sector care in general expect a better service than what is provided by the public sector. Patients value the freedom to choose services in the private sector. Patients need service related information to guide them in making their choices. It is the responsibility of the private sector to provide such information direct to the public as well as to the health ministry for monitoring purposes.

Growth of the private sector has posed many challenges for itself. Generating the required Human Resources, monitoring standards, containing cost escalations and addressing issues related to access and affordability are some key challenges facing the private sector.

Penetration of transnational tertiary care corporations and their impact on locally operated tertiary care facilities in both private and public sectors need to be evaluated. Areas for collaboration among public, private and transnational providers need to be identified and developed. Measures required for private sector to have its own stewardship for quality assurance, fairness in pricing of services and provision of relevant information for service regulators and users need to be urgently addressed.

Regulating the health sector is a function of government. Currently only the private sector is regulated and that too is restricted to registration and data collection processes. Regulations for quality, standards, accredition etc. are yet to be implemented. Patients are exposed to dangers of obtaining health services from unregulated facilities both in public and private sectors. The government is responsible for securing the rights of patients to receive services of guaranteed quality. Health care provided by both public and private sectors need to be better regulated.

Prof.I.Joel Fernando

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Pre Diabetes, does this exist?

A.L.P. de S Seneviratne

Diabetes is a major and growing public health challenge which threatens to overwhelm medical services. Type 2 diabetes confers significant morbidity and mortality, most notably with target organ damage to the eyes, kidneys, nerves and heart. The magnitude of cardiovascular risk associated with diabetes is best illustrated by its position as a coronary heart disease risk equivalent. Complications related to neuropathy are also vast, often working in concert with vascular abnormalities and resulting in serious clinical consequences such as foot ulceration. Increased understanding of the natural history of this disorder has generated the potential to intervene and halt pathological progression before overt disease ensues, after which point management becomes increasingly challenging

Although the pathogenesis of Type 2 Diabetes Mellitus (T2DM) is complex, the central mechanism is impairment in the action of insulin (insulin resistance), combined with inadequate secretion of insulin itself. Investigators have proposed a multistage model of the development of T2DM This follows an initial period of insulin resistance which is compensated for by increased insulin secretion from functional β-cells and increased β-cell mass, which work to maintain glycemic levels. With chronic over activity, there is a stage of stable adaptation, in which the β-cells are unable to fully compensate. This will initially manifest as prediabetes – Impaired Glucose Tolerance (IGT), characterized by postprandial hyperglycemia and/or impaired fasting glucose (IFG), evidenced by mild fasting hyperglycemia. High hepatic insulin resistance is typically seen in IFG, with almost normal values in skeletal muscle whilst in patients with IGT, the main site of insulin resistance is muscle, with only small changes in liver sensitivity. β-cell dysfunction is seen in both IFG and IGT. These two markers of defective glucose metabolism form the basis of the prediabetes state although the clinical relevance of their mechanistic differences is uncertain. The following stage is decompensation with rapid rise in glucose levels

In a normal person fasting blood glucose from venous blood is less than 100mg/dL (6.0m.mol/L.) If you consider HbA1c, it should be below 5.7%. The American Diabetic Association has released the following criteria to consider prediabetes state or impaired fasting glucose or glucose tolerance.

FBS - 100mg – 125mg/dL (6.1 – 6.9m.mol/L)

Postprandial blood glucose 140mg – 199mgp/dL (7.8 – 11.0 m.mol/L)

HbA1c 5.7 – 6.4%.

Prediabetes includes the concepts of impaired fasting glucose (IFG) and impaired glucose tolerance (IGT). In 2005 Wen et al reported on an 11-year follow-up of 36 000 persons with IFG (fasting glucose levels between 6.1 and 6.9 mmol/L) had significantly increased risk of mortality

related to cardiovascular disease (CVD). In a detailed review of the topic, Unwin and colleagues concluded that IFG and IGT (glucose ≥ 7.8 and < 11.1 mmol/L, 2 hours after ingestion of a 75-g oral glucose load) were associated with CVD. Impaired glucose tolerance was more strongly associated with CVD than IFG.

A number of studies have looked at lifestyle and pharmacologic interventions in people with prediabetes to determine if progression to frank diabetes can be prevented. These studies were summarized in a meta-analysis published recently by Gillies et.al in the British Medical Journal 2007. Researchers concluded that, in people with IGT, lifestyle and pharmacologic interventions (various anti obesity agents and oral hypoglycemic agents) are effective in delaying the onset of type 2 diabetes. They did not look at metformin individually, but instead included it with all other oral antidiabetic agents. Metformin is recommended as first-line treatment in diabetes; it is inexpensive compared with the newer drugs, and we believe that a review looking specifically at metformin is important.

The same factors that increase the risk of developing type 2 diabetes, increase the risk of developing prediabetes. These factors include:

- **Weight.** Being overweight is a primary risk factor for prediabetes. The more fatty tissue you have — especially inside and between the muscle and skin around your abdomen — the more resistant your cells become to insulin.

- **Waist size.** A large waist size can indicate insulin resistance. The risk of insulin resistance goes up for men with waists larger than 40 inches and for women with waists larger than 35 inches.

- **Dietary patterns.** Eating red meat and processed meat, and drinking sugar-sweetened beverages, is associated with a higher risk of prediabetes. A diet high in fruits, vegetables, nuts, whole grains and olive oil is associated with a lower risk of prediabetes.

- **Inactivity.** The less active you are, the greater your risk of prediabetes. Physical activity helps you control your weight, uses up glucose as energy and makes your cells more sensitive to insulin.

- **Age.** Although diabetes can develop at any age, the risk of prediabetes increases after age 45. This may be because people tend to exercise less, lose muscle mass and gain weight as they age.

- **Family history.** Your risk of prediabetes increases if you have a parent or sibling with type 2 diabetes.

- **Race.** Although it's unclear why, people of certain races — including African-Americans, Hispanics, Native Americans, Asian-Americans and Pacific Islanders — are more likely to develop prediabetes.

- **Gestational diabetes.** If you developed gestational diabetes while pregnant, you and your child are at higher risk of developing prediabetes. If you gave birth to a baby who weighed more than 9 pounds (4.1 kilograms), you're also at increased risk of prediabetes.

- **Polycystic ovary syndrome.** This common condition — characterized by irregular menstrual periods, excess hair growth and obesity — increases women's risk of prediabetes.

- **Sleep.** People with a certain sleep disorder (obstructive sleep apnea) have an increased risk of insulin resistance. People who work changing shifts or night shifts, possibly causing sleep problems, also may have an increased risk of prediabetes or type 2 diabetes.

Other conditions associated with prediabetes include:

- High blood pressure
- Low levels of high-density lipoprotein (HDL) cholesterol, the "good" cholesterol
- High levels of triglycerides — a type of fat in your blood

When these conditions occur with obesity, they are associated with insulin resistance.

In overweight adults up to 22.6% have been shown to have prediabetes, and approximately 5–10% of individuals with prediabetes will progress to diabetes in a year. Further, current estimates predict 472 million people having prediabetes worldwide by 2030. There is significant discrepancy in rates of progression, with some population based observational studies reporting that 55–60% of individuals with IFG at baseline having normal fasting plasma glucose (FPG) at 10 years follow up]. This is dependent on the population studied, ethnicity, obesity and other cardiovascular risk factors present. Those individuals with dysglycaemia, combined with dyslipidemia, hypertension and obesity appear to be most at risk of developing diabetes and subsequent CVD. Importantly, the risk also increases for those with higher prediabetes FPG values, leading to the proposal that these measures be regarded as continuous rather than discrete variables, with the development of diabetes itself viewed as a continuous process. Of note, IFG is more prevalent in men than women.

Some institutions have avoided the term 'prediabetes' to highlight the fact many individuals will not progress to diabetes. WHO has suggested 'intermediate hyperglycemia', whilst an ADA commissioned panel preferred 'high risk state of developing diabetes'. There is overlap between the various parameters of prediabetes and although the reproducibility of diagnostic criteria is lower than that for T2DM, the predictive value is higher than that of individual risk factors, with the combination of IFG and IGT more predictive than each parameter alone. To date, no diabetes prediction tool has been universally accepted, but it is likely the most effective model will consist of an initial 'prescreen' based on routine clinical values, followed by a more detailed assessment for certain at-risk individuals (e.g. overweight) with laboratory measures.

Fasting hyperglycemia, post-load glucose and HbA1c have been shown in multivariable adjusted analyses to be predictive of vascular mortality independent of vascular risk factors such as obesity, blood pressure and lipid profile. As well as predicting the development of diabetes, prediabetes itself is associated with increased risk of disease. Epidemiological studies, such as the Australian Diabetes and Lifestyle (AusDiab) project, have shown clearly that target organ damage precedes the diagnosis of T2DM, with both renal and retinal damage seen in patients with IGT. A number of other population studies have confirmed that individuals with IFG and IGT have increased risk of developing macro vascular disease; around a third of patients with coronary artery disease (CAD) have abnormal oral glucose tolerance test (OGTT), and 22% with acute and 14% with stable coronary heart disease have newly diagnosed T2DM.

There is a particularly strong association between prediabetes and autonomic neuropathy, with consistent reports of disordered parasympathetic function and sensory neuropathy. Diabetic autonomic neuropathy can affect a number of organ systems, with cardiovascular symptoms including resting tachycardia, orthostatic hypotension and silent myocardial ischemia, and cardiovascular autonomic neuropathy is significantly associated with overall mortality. Diabetes is also the most common cause of chronic peripheral neuropathy; chronic hyperglycemia with associated metabolic abnormalities, redox imbalance, dyslipidemia and advanced glycation end products (in conjunction with microvascular disease) are some of the multifactorial insults contributing to nerve pathology in diabetes. Pain and autonomic symptoms are typical. Therapies to treat diabetic neuropathy are still being actively pursued and range from aldose reductase inhibitors to antioxidants and angiotensin-converting enzyme (ACE) inhibitors. Patients with idiopathic small fibre neuropathy and diabetes or IGT have been shown to exhibit corneal changes, which can be detected and quantified by corneal confocal microscopy. This potentially powerful diagnostic tool may provide the earliest and most sensitive test to detect small fibre damage in diabetes and prediabetes. Importantly, the corneal changes appear to be graded with severity of disease, permitting quantification and monitoring of therapeutic interventions.

Can you prevent diabetes in prediabetic subjects?

Weight loss improves outcomes in T2DM and delays or prevents progression from IGT/IFG to diabetes. Epidemiological data from the Framingham Study has shown that sustained weight loss in overweight individuals can have a primary preventative effect on the incidence of T2DM, with a moderate weight loss of approximately 4 kg protective against progression to T2DM in at-risk patients. Consequently, many strategies aimed at preventing T2DM have focused primarily on weight loss, which has made it difficult to determine the independent effects of weight loss, dietary changes and exercise due to their combination in a lifestyle intervention arm of many studies. The largest study to date was conducted by the Diabetes Prevention Program (DPP) research group. This involved 3234 nondiabetics (ethnically and racially diverse) individuals with IGT of mean age 51 years and BMI 34 kg/m2 randomized to placebo, metformin or lifestyle intervention. The lifestyle intervention group were expected to maintain a weight reduction of 7% through dietary means and physical activity, performing at least 150 minutes of moderate intensity activity per week. Lifestyle intervention was associated with an impressive 58% reduction in the incidence of diabetes compared with placebo and superior to metformin. Indeed, for every 1 kg reduction in weight there was an associated 16% reduction in incidence of diabetes. Follow-up studies have confirmed that this preventative or delaying effect persists at 10 years. Post hoc analysis also confirmed the extreme propensity of gestational diabetes mellitus (GDM) on progression to diabetes, with a 71% higher incidence than those without a history of GDM (despite similar glucose levels at entry). Women with GDM randomized to metformin benefited from a 50% reduction in incidence of diabetes, compared with 14% in those without GDM.

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National Hospital of Sri Lanka

A legend in patient care – 150 years of illustrious service to the nation

Dr. Joe Fernando

Introduction

In 2014 a grateful Sri Lankan nation saluted this illustrious national institution and commemorated 150 years of dedicated service and recalled the pivotal role it has played in the day to day life of the people of this country. Few institutions in Sri Lanka can boast of such a unique record of service impacting so beneficially on the lives of the community for 150 long years. Throughout this period the National Hospital of Sri Lanka (NHSL) has stood steadfastly in times of peace as well as in times of strife. It has withstood all the ravages that the country underwent on more than one occasion. Thus it is incumbent on every citizen to preserve, foster and develop this national institution for posterity. The British rulers who occupied the maritime provinces in the 19th century endeavored to provide some form of health care to the indigenous population, probably to protect their garrisons from pestilential diseases, and also through some degree of sympathy. With the above in view the Civil Medical Department was created in 1858 to cater to the needs of the indigenous population. This indeed was a landmark in the history of the medical services in this country. In keeping with the above the first hospital was established in Prince Street, Pettah. Pettah hospital had 100 beds. Soon the demand at the Pettah Hospital outstripped the facilities, and the outcome was the establishment of the General Hospital with 200 beds at the present site, in 1864.

Medical care during the time of the Sinhalese Kings

Medical care is not a new phenomenon to Sri Lanka. Ancient chronicles such as 'Mahawansa' where the history of its people is recorded, reveals that provision of health care services was considered a major responsibility of the state during the times of the Sinhalese kings. The concept was that the state whether represented by the King or the Prime Minister was duty bound to look after the health of the people. This developed as part of the Buddhist Ethos, which stresses the duties of the rulers towards the ruled. The healthcare system that existed before the advent of the colonizing powers was entirely indigenous and included Ayurveda. During four centuries of colonial rule this system gradually lost its identity as an island wide organization although the practice of indigenous medicine was kept alive by passing on the system from father to son.

The main seats of learning were the monasteries and temples, and naturally a considerable number of Buddhist monks were famous Ayurvedic Physicians. Furthermore, some of the kings were well known for healing the sick. Among the many kings who were famous for treating the sick was king Buddhadasa. Anuradhapura and Polonnaruwa were in succession capitals of Sri Lanka during the glorious periods of its civilization. The ruins of ancient cities clearly point to the

existence of large hospitals constructed by the Sinhala kings for treating the sick. Ola leaf manuscripts contain descriptions of diseases and methods of treatment. Invasions by the South Indian kings as well as the Western powers heralded the decline of Ayurveda and other indigenous systems.

Medical care during the colonial era and the establishment of the NHSL

Successive invasions by the Portuguese, the Dutch and the British resulted in the gradual introduction of Western Medicine to this country. During the Portuguese and Dutch periods a few hospitals were built in the maritime provinces, chiefly in Mannar and Galle. However the Western system of medicine became established only during the British period. In the beginning the British were mainly concerned about the health of their garrisons. Nevertheless some degree of attention was paid to the health of the civilian population as well to ensure that the soldiers were spared from contacting pestilential diseases. A landmark in the Western system of medicine was the creation of the Civil Medical Department in 1858. The British also created a 'Native' medical establishment, thereby expanding the Western system throughout the country. Even to this day some of the older hospitals in this country are referred to as civil hospitals. The main task of the native medical establishment was to serve the local population. In 1817 Dr. Charles Farell, Deputy Inspector General of Hospitals suggested to the Governor that a general hospital be established for the poor. Accordingly in 1819 the first hospital was set up at Prince Street, Pettah which accommodated 100 beds. With western medicine increasing in popularity the demand increased resulting in outstripping the resources of the hospital. The outcome was the establishment of the present General Hospital Colombo (GHC) with 200 beds in 1864. It was located at Longden Place which was later named Kynsey Road in 1900, in recognition of the contribution made by Sir W.R.Kynsey, the former Principal Civil Medical Officer. It is noteworthy that at that time Mutwal was the residential area of Colombo, and the hospital was built at Longden Place so as to be in the countryside. By 1864 the hospital had 21 wards. The wards were connected by long corridors and the roofs were thatched. Some of the wards were named Seamen's wards, Planters' ward, Matapan Ward, Merchants' Ward & Gnanasekaram Ward. There were also other wards – European surgical, accident, native surgical, native medical, female surgical, dysentery, diarrhoea, etc. A physician & a surgeon looked after the patients.

In 1870 under very fortuitous circumstances the Colombo Medical School commenced training of doctors. The first principal of the medical school was Dr. Loos. A fee of sterling 2 pounds was charged from the students at the beginning of each term. The declared objective of establishing a medical school was to impart to the native youth of the country a practical, sound and safe knowledge of medicine and surgery. At the outset, the school commenced in a block of buildings of the General Hospital. In 1876 Mudliyar Samson Rajapaksha generously donated the land on which the present buildings stand. The buildings were put up by donations from Sir Charles Henry De Soysa and Muhandiram Samson Fernando. In 1880 the status of the medical school was elevated to that of a college. The General Medical Council of Great Britain recognized the licentiate L M S in Medicine & Surgery, granted by the College which was registrable in Great Britain. When

the University of Ceylon was established in 1942 the diploma was converted to the degree MBBS.

In 1942 several developments which were favourable to the newly established hospital in general and the medical profession in particular took place. The Ceylon branch of the British Medical Association was established in 1887. De Soysa Lying in hospital was built and donated to the government by Sir Charles Henry De Soysa, and was opened in 1879. Lady Havelock Hospital, later named Lady Ridgeway Hospital was opened in 1885. In 1900 the Bacteriological Institute (Pasteur Institute), now the MRI was opened, again a donation by Sir Charles Henry de Soysa. The first head of the Bacteriological Institute was Dr. Sir Marcus Fernando. The aforesaid institutions were responsible in no small measure to the development of General Hospital as an excellent patient care institution and a teaching hospital. The Ceylonese doctors who succeeded the British medical personnel were very eminent personalities. The Principal Chief Medical Officer always had a very high regard for the local medical professionals, so much so that Sir West Ridgeway, Governor in his address to the Ceylon Medical College in 1903 remarked that 'Ceylon' is proud of its medical services and justly proud and it has been a great pleasure for me to be associated with the medical services of Ceylon, and Ceylonese I hope it will remain.

Nursing Care

Of the many professions allied to medicine, nursing is the most important. In ancient times the monks themselves attended on patients. The Vinayapitaka contained the code of conduct for Buddhist Monks which exhorted on them to attend on the sick brethren. General Hospital Colombo was the first hospital to deploy female nurses. In the beginning they were British nurses from United Kingdom. A qualified matron from UK was sent to establish a nursing school in the General Hospital Colombo in 1878. Although the services of nurses were appreciated by all, there had been difficulties in recruitment due to various prejudices. Since the government had difficulties in recruiting nurses, the government had appealed to the Catholic Church for help. Thus the Franciscan Missionaries of Mary agreed and the first batch of Franciscan sisters arrived in 1886. They took over the nursing of 200 patients in the wards at General Hospital. The sisters were accommodated at St. Peters House in General Hospital. Due to the shortage of beds in the hospital an annex was opened at Ragama, where the elderly and the chronically ill were accommodated. Some of the nuns also worked at the Ragama annex. In 1912 an out break of cholera occurred at the Ragama quarantine camp and the patients who went to Ragama from General Hospital were taken back to General Hospital. Even to this day the medical wards of General Hospital are referred to as the Ragama section.

General Hospital in the 20th century

The 20th century in general and the latter half of that century in particular could be considered a favourable period for the General Hospital in spite of two world wars in 1918 & 1938. Even though the British were pre occupied with the war efforts, development of the General Hospital continued unabated. In 1913 the govt. announced that a rapid development programme would commence. Furthermore due to the high cost of land it was decided that all new buildings would be multi-

storied. During the first half of the century, more and more specialties were introduced which facilitated improving patient care as well as teaching.

With World War II coming to an end, the distant dream of independence was now at the doorstep of this country. As expected Sri Lanka gained independence in 1948. With renewed vigour and enthusiasm the country was on its way to rapid development in all areas, in particular, health and education. The impressive multistoried building facing Regent street, was constructed in 1956. During this period a whole range of hitherto unavailable specialties were introduced. Neurosurgery, Cardiology, Thoracic surgery, Ear, Nose & Throat surgery, and Neurology etc. The bed strength of the hospital had increased several fold by now. A nurses' training school was established, and lay nurses took over the nursing duties. The government decided to discontinue the services of the Catholic nuns. With a view to fast tracking development of GHC, the Ministry of Health created the Colombo Group of Hospitals which comprised the General Hospital Colombo, De Zoysa Maternity Hospital, Eye Hospital, Castle Street Hospital for Women & the Colombo South Hospital. The Colombo Group was placed in charge of a very experienced medical administrator – Dr. Malinga Fernando who later was appointed Director General & later Secretary Health.

Decentralization of General Hospital Colombo (GHC)

In 1977 the Ministry of Health appointed a committee to examine and report on further development of this all important institution. The terms of reference among others included the role of GHC in the national context, being the largest patient care institution in the country, its role as an important teaching hospital, basic & post basic training of nursing personnel. The committee was of the firm view that GHC should function as a special decentralized unit of the Ministry of Health with a separate budget, and a senior medical administrator designated superintendent to be in-charge of the institution assisted by a deputy. The Ministry of Health, accepting the recommendation of the committee, decided to implement the recommendation, with effect from 1st January 1978.

This indeed was a far sighted move in the right direction. Accordingly the author was appointed as Superintendent while Dr. Lucian Jayasuriya was appointed the Deputy. Soon after decentralization, the new management was able to implement several development programmes. A long felt need of the hospital was a medical & surgical intensive care unit. The Surgical Intensive Care Unit was setup adjoining the operating theaters A, B & C. Dr. Thistle Jayawardane senior consultant anaesthetist was the prime mover in establishing this unit. The management of the hospital with the fullest cooperation of the physicians was able to obtain a grant of Rs. 5 million from late Mr. Moosajee to establish the Medical Intensive Care Unit. Probably these two units were the very first intensive care units in the country.

The neurology unit was housed in ward 43 with a very limited number of beds. There was an urgent need to develop a separate neurology unit in the teaching hospital. The senior consultant neurologist Dr. J.B. Peiris held discussion with the management to construct an up –to-date neurology unit with donations from various philanthropists. To this end a meeting was held with several

invitees where the spouse of late President Premadasa was the Chief Guest. At this meeting a number of volunteers agreed to provide donations and a considerable amount was collected towards this project. Ven. Vipassi, the Chief Buddhist priest of the hospital contributed very generously towards this project. Within two years the unit was completed which indeed was a boon to the General Hospital.

The accident service which was housed in the rear section of the Victoria Memorial Eye hospital was totally refurbished & a critical care section was also set up with funding from the Bank of Ceylon. A separate operating theatre in close proximity to the main theatres was opened with a gift from the Nawaloka Group of Companies. A branch of the Bank of Ceylon and a sub post facing Regent street were opened.

Creation of the Ministry of Teaching Hospitals

In 1983 for some hitherto unexplained reasons, the Ministry of Health was divided and a separate Ministry of Teaching Hospitals was created to manage all the teaching hospitals in Colombo as well as outstations. The new Ministry was in the charge of Mrs. Sunethra Ranasinghe while the rest of the Ministry of Health was under Dr. Ranjith Atapattu. The above decision was taken by the late President J.R. Jayawardana. At that time the common anecdote in medical circles was "Sunethra got the best & Ranjith got the rest" Dr. Lucian Jayasuriya was appointed Director of Teaching Hospitals. Be that as it may the Ministry of Teaching Hospitals was able to carry out several important developmental programmes among which was the construction of a new up to date purpose built accident service facing Regent Street, thus fulfilling a long felt need of the hospital. The Ministry of Teaching Hospitals also established an infection control unit, and augmented the water supply to the hospital. All these important programmes were carried out by the Ministry of Teaching Hospitals thanks to generous grant provided by the Finnish Government. On completion of the above work a sum of Rs. 100 million was left over. By now political changes took place and late President Ranasinghe Premadasa was appointed as President. With this change, the Ministry of Teaching hospitals was abolished and the teaching hospitals were again vested with the Ministry of Health.

The Finnish Authorities suggested that the Rs 100 million that was left over be utilized to refurbish the old medical wards in the Ragama section. When the request came to the Secretary Ministry of Health – the author was occupying this post. He suggested that instead of refurbishing the old wards a new purpose built, multi storied unit be constructed to accommodate several wards which later could be replicated. With the implementation of this suggestion several multistoried units have been constructed to accommodate over 1000 patients.

In 1995 General Hospital was renamed National Hospital of Sri Lanka which is more appropriate considering that it is a national institution serving the entire nation and the streets encircling the hospital has been named as hospital square. The total budget of the National Hospital is approximately Rs. 6 billion, while the institution employs almost 5000 staff of all categories. There are 23 operating theatres and 19 intensive care units. The important role of the National Hospital in the 30 year war with the LTTE cannot be over emphasized. The main

brunt of the casualties was borne by the National Hospital while a lesser number was treated at the Anuradhapural General Hospital. Throughout the period of the war an excellent disaster plan was in operation at the hospital. The accident service was programmed to provide the best possible services to the forces personnel as well as civilian casualties – this indeed was morale booster for the forces personnel.

Conclusion

During the last 150 years National Hospital of Sri Lanka has provided patient care to millions of patients. The greatest tribute that could be paid to this institution is to develop it as a centre of excellence. The responsibility to this end solely rests with the Ministry of Health. The greatness of an institution cannot thrive on the mere memories of its glories past, rather particularly in the case of a public institution such as the National Hospital which we are all proud of, its greatness must necessarily be measured, by credibility enjoyed in the public eye.

Acknowledgement

In the preparation of this article considerable information has been obtained from 'A history of Medicine in Sri Lanka' authored by the one and only medical historian of great repute Dr. Chris Uragoda. He is a well known chest physician, Past President of the Royal Asiatic Society, Fellow of the National Academy of Sciences Sri Lanka, and a Member of the Experts Panel on Tuberculosis.

The writer wishes to express his deep gratitude and thanks Dr.Uragoda.

The writer of this article was the first superintendent of General Hospital, Colombo from 1978 – 1982.

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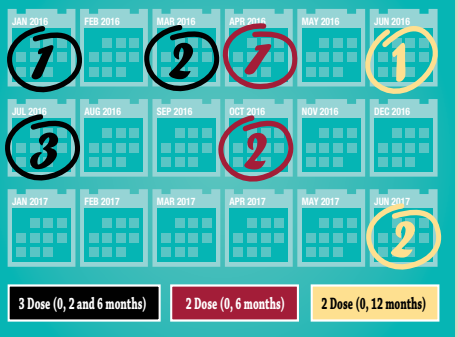

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Palliative Care Association of Sri Lanka

Dr. K. Chandrasekher

The Palliative Care Association of Sri Lanka (PCASL) was formed in 2014 and officially launched in April 2015 under the patronage, guidance and auspices of the College of General Practitioners of Sri Lanka, (CGPSL), an academic body of family physicians which has been at the forefront, over the past four decades. This gained recognition for Family Medicine as an academic discipline in Sri Lanka. Aspects related to social responsibility and accountability are foremost in all the projects conducted by the CGPSL.

Understanding the importance of palliative care, a relatively less heard discipline in Sri Lanka then, but a pressing need for the community at large, was the main reason the CGPSL set up the Palliative Care Subcommittee in 2012.

This Subcommittee signed an MOU with the 'Institute of Palliative Medicine (IPM)' located in Calicut, Kerala, India, which is a World Health Organization (WHO) Collaborating Centre for Community Participation in Palliative Care, to use their expertise to develop palliative care skills and resources in Sri Lanka.

Importance of the Short Certificate Courses in Palliative Care

The Subcommittee, jointly with IPM, commenced two-day certificate courses for doctors in caring for the dying and their grieving families. Six such two-day certificate courses were conducted from 2012-2014. Three more such training programmes have been held after the launch of the PCASL in 2014 bringing the total number of trained doctors to almost 421 both in the state and private sector.

The courses were held in Colombo, Galle, Jaffna, Kandy, Batticaloa, and Anuradhapura. These certificate courses were open to all doctors in Sri Lanka and were attended by general practitioners(GPs) in both the state sector and private sector, consultant physicians, anaesthetists, as well as doctors attached to the Cancer Control Programme of the Ministry of Health.

To understand the importance of such an activity, we would like to take the reader through some important statistical information pertaining to our country. These are as follows:

a) Sri Lanka has a population of about 21.2 million and approximately 112,500 deaths a year. The total number needing palliative care in the country can be estimated to be 60% of all deaths or about 68,000 people a year, with the majority of them dying of non-communicable diseases.
b) With rapid ageing of the population, the highest number of patients needing palliative care will in the future come from the elderly terminally ill. There are very few institutions in the country providing much needed palliative care services.
c) GPs are called upon to provide palliative care to patients who are at home and in

the final stages of their lives. This stage can last several years in almost 90% of the patients. The majority of such patients are afflicted with problems of old age and may be bed ridden. Patients dying due to cardiovascular problems and malignancies also constitute a large segment needing palliative care. Many are stroke victims. Some are in the final stages of dementia, end stage renal disease, terminal chronic obstructive pulmonary disease (COPD), HIV/ AIDS and cerebral palsy.

d) Furthermore, in dealing with chronic illness in patients of all ages (paediatrics to geriatrics), actual home base care is provided by lay persons, who are family members or hired help who have little or no formal medical or paramedical training.

e) Doctors in general practice, both full time and part-time working almost exclusively in the private sector, are responsible for providing 51%, of the primary care in our country.

f) Palliative care is not still an established medical or nursing specialty in Sri Lanka. Education and training in palliative care has been the weakest link in palliative care in the country. Training facilities for health care professionals or community volunteers has not yet been formalized.

Thus, establishing facilities to train doctors and nurses to take care of the incurably ill, chronically bed ridden and dying patients, and training community volunteers are important in this context. With this objective, as a first step, PCASL commenced basic training programmes in palliative care through its short certificate courses as mentioned above.

If Sri Lanka has a pool of trained doctors and nurses capable of educating the families and the community on basic aspects on palliative care, the quality of care in this area can be significantly improved within a short period of time. This is one more reason why the short certificate courses, in palliative care, conducted by the PCASL for primary care physicians assume significance.

The Importance of Home- based Palliative Care

Home-based care forms the backbone of any palliative care management programme. Capacity building of trained volunteers and families under the supervision of medical and para-medical personnel in the community can have an immediate impact in improving the standard of care.

The total number needing palliative care in our country can be estimated to be 60% of all eventual deaths taking place every year in Sri Lanka. As mentioned above, statistics show that only 10% of this number has instant deaths while 90% often suffer a long, lingering death. This can lead to great emotional turmoil and suffering not just in dying patients but also in their loved ones who care for them. Even in modern Sri Lankan society many people die at home.

General practitioners (GPs) are called upon in such instances to provide palliative care to patients who are at home and in the final stages of their lives. This makes it imperative for GPs to be trained adequately in the field of palliative medicine. GPs, however, are not specialist palliative care physicians. They need the guidance and support of specialists in the respective fields when the patients they care for are afflicted with complicated problems and a multidisciplinary approach is needed to alleviate or resolve these. They need to work with specialists and para-medical personnel

as members of a multidisciplinary team to provide palliative care to patients, both in hospital and at home, so that continuity of care as well as coordinated care is ensured. Training GPs would have an immediate impact since they are already managing such patients.

We strongly believe that this would enable us to fulfil the theme for 2016, "Living and dying in pain:-It doesn't have to happen "come true.

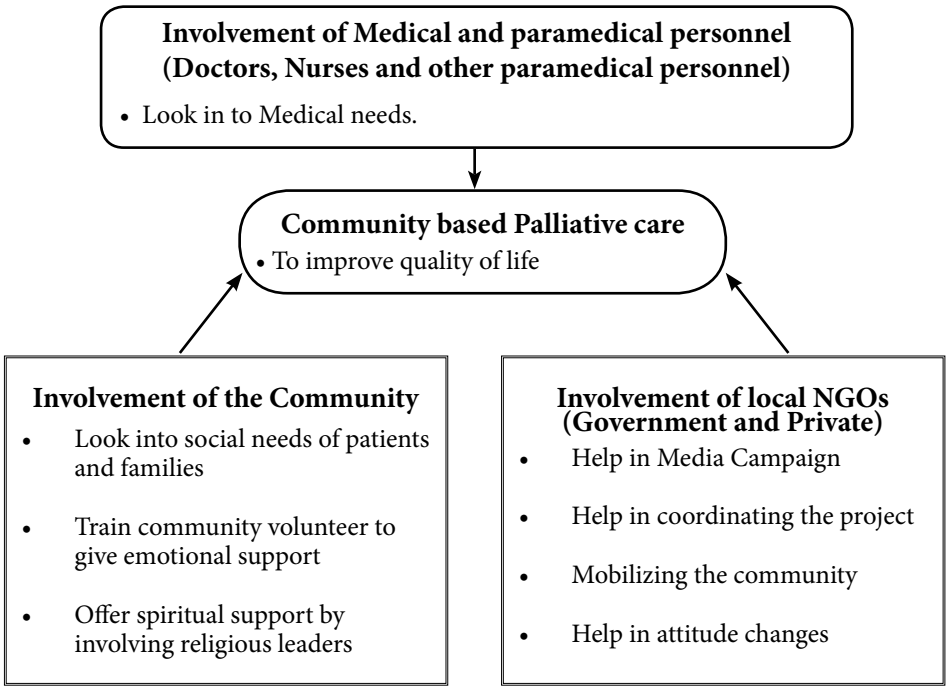
Plans are thus underway to form branches in various parts of the island with community involvement. The first initiative has been taken with the Piliyandala branch of the Palliative Care Association. This is serving as a model project which has been developed, owned, and sustained by the local community. This branch, in the Kesbewa Divisional Secretariat which is functioning for the last one year has been able to extend support to eight patients and their families in the past year. This enabled two patients who were terminally ill with cancer to have a dignified death. The branch has also been able to conduct four awareness programmes for the nursing staff of the Divisional Hospital at Piliyandala as well as for the staff of the Ministry of Health (MOH), Piliyandala. A Continuous Professional Development (CPD) Programme was also held for the doctors in the Piliyandala area on "Pain Management in Palliative Care in General Practice"

Awareness programmes were held for retired officers of the Bank of Ceylon and for community leaders in the area to enhance their understanding of the importance in palliative care. These types of sensitization programmes help to recruit volunteers to join the effort of helping the less fortunate in their community. The good work done by the organization is creating awareness in the community and it is now possible to see that donors are coming forward to provide support and assistance.

One such donor from overseas provided a grant that was used to conduct a training programme for doctors, support some activities of the Piliyandala branch, and also conduct the first oration, since the formation of the organization, on World Palliative Care Day in October 2017. All donations and expenses are audited, and proper accounts are submitted to the donors.

Plans are also being drawn up to run a community project to look after the poor, in need of palliative care, in the community. A multidisciplinary team would look after such patients in their homes and ensure that coordinated and continuous care is provided ensuring they continue to attend the state-run clinics. Such a public health approach is increasingly being seen as a practical solution to ensure better quality and coverage in palliative care without burdening the already over stretched state-run free medical care system. . (Please see diagram)

Sensitization pocket meetings are also being planned to enhance awareness of the importance of palliative care among members in the community, and solicit their participation in community programmes of the above nature so that these can be run sustainably.



Role played by the various segments of the Community in Community based Palliative Care Services.

The Palliative Care Association of Sri Lanka (PCASL) has also been active in convincing the Ministry of Health of Sri Lanka to integrate palliative care into the Ministry of Health's, Sri Lanka Strategic Plan 2016-2025. The draft Palliative Care Policy for Sri Lanka submitted by the PCASL to the Ministry of Health, led the Ministry to appoint a Ministerial Task Force to formulate a National Palliative Care Policy for Sri Lanka.

This in turn led to the formation of the "Palliative Care and End of Life Care Task force" formed by the Sri Lanka Medical Association which is working towards achieving the goals of the Ministry of Health. The PCASL is an important stake holder in this high-powered committee.

In brief, some of the other activities undertaken by the organization are given below

- A sensitization programme on Palliative Care was conducted for over 500 children from Colombo, Kalutara and Gampaha Districts on the World Palliative Care Day in 2015.

- A two day Foundation Course in Palliative Nursing Care was conducted for forty Nurses in 2016.

- The Sri Lanka Medical Association recognizing the services rendered by the PCASL requested the organization to conduct the Post Congress Session on Palliative Care during their 129th Annual Session of Sri Lanka Medical Association.

- A Parallel Session on Palliative Care was conducted by PCASL at the College of Surgeons of Sri Lanka Annual Academic Sessions in 2015.

It is with great satisfaction we would like to share the information that The Asia Pacific Hospice Palliative Care Network has recognised PCASL as the National Organisation in Palliative Care in Sri Lanka.

It is a great honour for us that Palliative care Association of Sri Lanka has been recognised by the Ministry of Health in their National Strategic Framework for Development of Health Services 2016-2025 printed according to instructions of the Sectoral Oversight Committee (sections 43 & 44 of Constitution) of Democratic Socialist Republic of Sri Lanka.

The PCASL is a member of the, International Organisations such as The World Hospice and Palliative Care Alliance and International Association for Hospice and Palliative Care enabling the organization to be represented in International Conferences many a times.

Our Dreams

The intentions of the PCASL are primarily to see that every Sri Lankan doctor in general/family practice has an adequate grounding in palliative care to serve his or her patients when they require such care. The palliative care services as suggested above would complement the free healthcare offered by the state thereby reducing the number of state hospital beds occupied by terminally ill patients.

Sri Lanka has a history of establishing a palliative care centre in Colombo even before the internationally known St. Christopher's Hospice in London. Our hope is to bring back this pride by working together with like-minded individuals and organizations, locally and internationally; empowering GPs by training and strengthening their networks to access multidisciplinary expertise; and sensitizing the community so that patients needing palliative care would receive effective and coordinated care and support. This would ensure that we "do not leave those suffering, behind" which was the theme for this year's World Hospice and Palliative Care Day.

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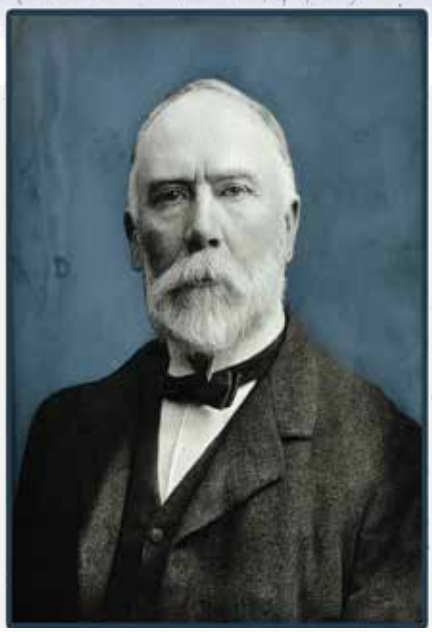
The Question is not how long we live, but how well we live



Sir. James Mackenzie

Sir James Mackenzie FRS (12 April 1853 – 26 January 1925) was a Scottish cardiologist who was a pioneer in the study of cardiac arrhythmias. Due to his work in the cardiac field he is known as a research giant in primary care.

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Excessive Daytime Sleepiness

Dr. Lasantha Heenatigala

The American Academy of Sleep Medicine has defined Excessive Daytime Sleepiness (EDS) as the inability to maintain wakefulness and alertness during the major waking episodes of the day, with sleep occurring unintentionally or at inappropriate times almost daily for at least three months(1). EDS is characterized by persistent sleepiness during the day even after apparently adequate or prolonged nighttime sleep. Hyper-somnolence is another word sometimes used to describe EDS. The ICSD-3 (International Classification of Sleep Disorders 3) defines hyper-somnolence as excessive sleepiness when wakefulness is expected, and hypersomnia as a disorder characterized by hyper-somnolence. Patients often use other terminology to describe EDS, using words like tiredness and fatigue and therefore the doctors have to inquire precisely to differentiate EDS from other types of symptoms.

About 20% of the general population, report a level of day time sleepiness sufficient to interfere with daily activities and it is said to be the most common of sleep-related patient symptoms(2). It is undoubtedly a common complaint in primary care yet underdiagnosed and most sleep/wake disorders including excessive day time sleepiness can be effectively managed in the primary care setting (3). EDS can have serious and diverse consequences such as road traffic accidents, work place accidents, health problems, and poor academic and professional performances.

Table 1- common causes of EDS

Primary Hypersomnias of central origin,

- Narcolepsy,
- Idiopathic Hypersomnia,
- Other rare primary Hypersomnias, (Ex: Kleine-Levin syndrome)

Secondary hypersomnia's

- Sleep disorders
  - Sleep-related breathing disorders
  - Behavioral sleep deprivation
  - Other sleep disorders
    - circadian rhythm sleep disorders, (Jet lag, Shift work disorder, Delayed sleep phase type)
    - sleep-related movement disorders (restless legs syndrome, periodic limb movement syndrome, sleep walking disorder, Nightmare disorder)

- Medication Medical, and Psychiatric conditions,
  - Medication effects Includes prescription, nonprescription, and drugs of abuse
  - Psychiatric conditions (depression, anxiety)
  - Medical conditions (Including Hypothyroidism, head trauma, stroke, cancer, inflammatory conditions, encephalitis, neurodegenerative conditions)[2].

There are a lot of causes for excessive day time sleepiness and would include a lot of physiological, neurological, medical, psychological and social causes. Primary causes of hypersomnia are rare and the secondary causes therefore are more common.

Sleep deprivation is the most common cause for EDS while medication effects, substance use, obstructive sleep apnea (OSA), and other medical and psychiatric conditions also play an important role. (Table 1). EDS is a well-known side effect of certain groups of drugs and therefore medication review of each patient with EDS is important and in these circumstances the treatment would just be the omission of the offending drug. (Table 2). Recreational drugs, such as marijuana and alcohol are sedating and can be the cause for the EDS while withdrawal of stimulant drugs (such as Cocaine and Amphetamines) also may contribute to EDS.

Table 2- common drugs classes causing hyper-somnolence

Anticonvulsants
Antidepressants
Antihistamines
Antihypertensives
Antipsychotics
Antitussives
Dopamine blockers
Opiates
Sedatives

Sleep related breathing disorders are a very common cause of sleep fragmentation and hence EDS. Obstructive sleep apnea (OSA) is the most common and important in this group of disorders all over the world. Its prevalence in middle aged American population is found to be 9% in women and 24% in men and about one fifth of these patients experience EDS. (5) Other disorders in this group includes Hypopnoeas and Upper Airway Resistance Syndrome (UARS).

Medical disorders such as structural brain disease, encephalopathies, stroke, neuro-degenerative disorders, cerebral tumours and multiple sclerosis are examples for medical conditions known to associate with EDS. A variety of medical conditions predisposes the patients to fragmentation of sleep and therefore to EDS. Chronic pain is the most important cause of sleep fragmentation and the others include Asthma, Cardiac failure, Arthritis, Fibromyalgia, GORD, Epilepsy, Urinary dysfunction and Irritable bowel Syndrome. Psychiatric disorders such as depression and Psychogenic hypersomnia also are examples for association with EDS.

Primary sleep disorders include Narcolepsy which is the most common of them with a prevalence rate of 0.03%-0.05% while Idiopathic hypersomnia comes second to it.

A focused medical history and a physical examination, including the search for a possible secondary cause for EDS is important. Detailed information about sleep patterns may be helpful to ensure

that the patient has adequate regular night time sleep and in the detection of sleep deprivation. The patients sleep hygiene should also be looked upon and any corrections should be done accordingly where necessary. Some useful information may be obtained from the spouse or bed partner in the cases of Obstructive Sleep Apnoea, Restless Legs Syndrome and Periodic Limb Movement Disorder and also in parasomnias such as sleep walking, sleep terror disorder and nightmare disorder. Presence of any chronic medical or psychiatric condition and the use of alcohol and recreational drugs may reveal the true reason for EDS.

During the physical examination also some important clinical features may be observed in relation to Obstructive sleep apnoea, neurological, medical, psychiatric conditions. For example, increased BMI and neck circumference, increased age, alcohol use, male gender and anatomical variations of the upper airways are some risk factors for Obstructive Sleep Apnoea. A high Mallampati score (class 3 or 4) is associated with higher chances of OSA.[4].

Though some patients would present with the symptom of daytime sleepiness, most would underestimate their excessive sleepiness and would therefore go unnoticed. For that reason there are validated questionnaires available for the GP's to get subjective information from patients about their day time sleepiness.

The Epworth Sleepiness Scale is a validated, patient-completed assessment of daytime sleepiness that can be used as a screening test [2]. A score of 12 or more on the Epworth Sleepiness Scale is a kind of indication that further evaluation is necessary. The Stanford Sleepiness Scale is another alternative such Questionnaire that can be used in the initial assessment [2].

In addition to the subjective sleepiness scales, some of the physiologic measures of sleepiness also can be used to assess the severity of the problem as well as to come to a specific diagnosis. The doctors can seek the help of a sleep laboratory if he or she feels it is necessary for further evaluation. Overnight oximetry is one of the simplest and, generally, earliest sleep studies that may be conducted. It involves the use of a probe worn on the finger or earlobe that continuously measures oxygen levels and heart rate. Overnight Polysomnography is an important investigation and involves an overnight stay that is monitored by a trained technician. It is usually helpful in the diagnoses of Sleep apnoea, Narcolepsy, Idiopathic hypersomnia, Restless legs syndrome, Periodic Limb Movement disorder and Parasomnias. .

The Multiple Sleep Latency Test (MSLT) is an important investigation that is done when further investigation is required in OSA and also in Idiopathic hypersomnia and narcolepsy. This is a standardized test performed by allowing the patient to take naps on 5 occasions throughout the day. Each nap is separated by 2 hours of wakefulness. During each nap, sleep latency, or the amount of time it takes the patient to fall asleep, is measured with standard polysomnographic technique, and a mean sleep latency time can be established [2].

Maintaining a Sleep diary is another way of obtaining some important data which may be helpful in difficult cases.

If there are features of Narcolepsy (such a history of cataplexy, hypnagogic hallucinations or sleep paralysis) or Idiopathic hypersomnia (a history of long sleep times, sleep drunkenness, automatic behavior and un-refreshing naps) they should be referred to a sleep center for further evaluation and assessment.

Treatment of the EDS obviously depends on the cause of the problem. While managing the patients it is always important to provide them with appropriate advices proactively to prevent any accidents or harm due to the EDS.

If sleep deprivation is the problem, explanation, reassurance and some advice may be what are all necessary. Some advice on life style modification would help some people with shift work disorder or delayed phase disorder. All may benefit by providing information on how to practice good sleep hygiene. The medical and psychiatric problems may be handled appropriately. Patients with OSA may be referred to ENT surgeons or sleep specialists for treatment with positive pressure devices (e.g., CPAP) during sleep which would improve symptoms of daytime sleepiness for most patients.

Most patients may benefit with non-pharmacological measures and only a minority may need therapy with an activating agent such as Modafinil, amphetamines (dextro¬amphetamine, methylphenidate) and pemoline.

It is always worth mentioning that the primary care physician can always seek the advice and support of an appropriate specialist while managing the patients.

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The Reality of The Heroin Epidemic

Dr. H.I. Pathirajamudali

Heroin addiction of long duration causes permanent changes in the brain and brain functions. The symptoms and signs of brain damage could be usually seen after 5 years of continuous use of heroin. Heroin causes changes in the brain structure and how it works. This leads to compulsive and destructive behaviour. After more than 10 years of addiction they are more like animals or vegetables. This is not a moral weakness or a weakness of character. It is a disease and therefore it is a medical problem that needs to be treated. Therfore heroin addicts should go to medical clinics and not to police stations or prisons. Criminalization of heroin addiction which is a disease is a blunder.

Heroin affects the mesolimbic pathway or reward pathway of the brain. Heroin addiction, schizophrenia and depression cause similar changes in the brain. Heroin is also capable of shrinking brain areas that are essential for wise decision making and to withstand momentary urges. This is why many heroin addicts experience relapses. Forgetfulness, personality changes and changes in the sleep patterns are seen in both heroin addiction and Alzheimer's disease. Shrunken brain, dying neurons with tangels and plaques were discovered at postmortems of heroin addicts. These are features of Alzheimer's disease.

Heroin addiction could be cured completely before brain damage occurs. That is during the first five years of addiction. After brain damage has already occured, treatment will prevent further damage to the brain and remove the heroin residues from the fatty tissues in 2 to 3 months. If treatment is not given, then, it will take 6 years for the heroin residues to leave the fatty tissues. When heroin residue is present, relapses occur more frequently. We can deal with a very large number of heroin addicts, if we treat them. A person taking treatment will not lose his job because he can take treatment while working, so that the family does not have to go hungry, because they will not lose the bread winner. A person taking treatment need not mix up with other heroin addicts, thus preventing the formation of groups and networks which lead to crimes.A person taking treatment is not exposed, whereas when he enters prisons or other isolations, people in the vicinity will get to know and the entire family and their home is blacklisted by the residents of the area. With treatment the appearance of heroin addicts soon become normal.When taking treatment, with the amount of money spent to buy heroin for 2 days, they can take treatment for one month. It is a chronic disease of the brain and abstinence alone will not cure it. As with other chronic diseases the earlier the treatment offered in the disease process, the greater the positive outcome.

After treating heroin addicts from all over the country, and talking to their parents and families during the past 30 years, my estimate is that there are more than 450,000 heroin addicts in the country, thus causing an epidemic. Many, who are working at

ground level, say that the number of addicts in the country is much more. I started treating heroin addicts in 1986. I received 200 tablets of Methadone in February 1988 from Civil Medical stores after an inspection of my dispensary by the food and drugs inspectors and other officials. There were a few heroin addicts coming for treatment everyday. The amount of methadone was inadequate and on each occasion when I requested for an increase of the quota issued to me, the authorities came for an inspection of my clinic.

In 1988 there were 15 doctors who obtained methadone from the civil medical stores to treat heroin addicts. By 2009 there were only 8 doctors who treated heroin addicts. Those doctors engaged in the treatment of heroin addicts under the authorization of the Director General of Health Services (DGHS) and with the guidance of other directors. The DGHS is also a member of the Board of Directors of the National Dangerous Drugs Control Board. In a letter dated 22nd February 2009 addressed to a doctor treating heroin addicts, stated as follows "We are informed that you have obtained/purchased 51,000 and 7768 methodone tablets in 2008 and 2009 respectively, which is a controlled substance which has to be used only for approved medical use. In terms of the law and the International Convention it is a narcotic substance. Hence its use is illicit. Please, Let us know the names and addresses of persons on whom you have administered this narcotic substance."

This letter was received after treating more than hundred thousand heroin addicts during past 20 years. In 2008 and until February 2009 there were 9000 names and addresses of patients. It would have been suicidal to have given their names and addresses. It was against the Hipocratic Oath and professional secrecy. Most of the 8 doctors stopped treating heroin addicts. If not for such occurances the number of doctors of 15 in 1988 would have increased to 100 doctors by now.

Methadone is the safest medicine to treat heroin addicts. It has a long duration of action of 24 hours. It occupies opioid receptors thus reducing craving to take heroin. Methadone allows a gradual reduction of dosage, until abstinence occurs and compliance is assured. Addicts on methadone are more likely to have jobs less likely to commit crime and less prone to HIV infections. They will need very little money for their survival. With money spent on heroin for 2 days, they can take treatment for one month. Unfortunately most of the people who could benefit from methadone do not receive it.

We obtained methadone and other medicines from civil medical stores and the medical supplies division (MSD) from 1988 to 2009 without any hindrance. We did not get methadone in 2009 and 2010 because methadone was out of stock. Again we did not receive methadone in 2014. Then again we Did not get methadone from March 2017 to November 2017

The British National Formulary (BNF) recommended 6-12 tablets of methadone per day. Addiction to methadone in as dangerous as addiction to heroin. But with the reduced dosage we give our patients, that is 1/2 to 1 tablet per day, there can never be a single methadone addict.

The real picture of heroin addiction in this country is much darker and gruesome. Unless, atleast 100 doctors volunteer and the Ministry of Health Services supplied the necessary medicines to treat heroin addicts, to get over this disaster, it is likely

that the country would be full of mentally disoriented criminals and we will need many mental asylums to accomodate them. If this continues the country would be infested with beggars and street prostitutes.

Methadone is available in most counties to treat heroin addicts. In 1988 there were only a few thousands of heroin addicts and they could obtain methadone and other medicine from doctors. Now there is an epidemic of more than 450,000 heroin addicts. For each heroin addict one family in completely destroyed. But there is no treatment available. We would like to know why there are so many obstacles and suppressions to treatment of such a massive section of unfortunate youth of our country.

In conclusion, this heroin epidemic is a national calamity. In my opinion it is the most damaging medical problem that this country is facing today. It is the paramount obligation of doctors and other patriotic people to come together, organize and develop ways to save this country from this menace.

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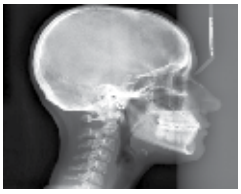

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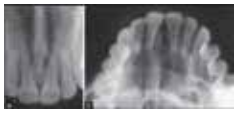

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Healing Plants

Dr. N.A. Kottahachchi

History proves that we had a valuable system of medicine. Sri Lanka is a granary of herbal plants. The more we peep into our gardens or to the neighboring groves, the more we collect enough herbs for any emergency such as for cuts, bleeding aches and pains. We need not borrow any medicines from other sources. All we need to do is to open the old manuscripts and identify the herbal plants and learn their uses. The herbs have been tested and experimented a thousand and one times. Traditional physicians have with them the secrets of these valuable medicines, but they fear to part with them. The reason for this is that when medicine is commercialized the tendency is for it to be adulterated. The most essential factor for the upliftment of the indigenous practice is to enrich the knowledge of students in the use of herbs with the aid of traditional physicians. They have valuable knowledge which cannot be proved scientifically.

Ayurvedic science in Sri Lanka shone at its best during the reigns of King Buddhadasa and King Parakramabahu the Great. During those days every Sinhalese of noble birth was expected to know the Ayurveda besides royalty. This included Buddhist Monks and poets. These physicians attained a high degree of efficiency in both medicine and surgery. Yet they did not work for pecuniary gains. Even the Sinhalese kings among whom there were famous physicians and surgeons who practiced medicine as an act of service to gain merit. In many parts of the island people still have confidence in indigenous treatment. The main reason is that the indigenous physician is available in their hour of need even without State aid and without any facilities. They still continue their valuable traditional medicine. This shows the usefulness of indigenous medicine to relive mankind from suffering.

Dr. John Attygalle in his book, "Materia Medica" gives us his knowledge and experience with native physicians. They have no modern knowledge of antiseptics or aseptic methods in their treatment. Yet the doctor admits how the patients recover without any blood poisoning or other ill effects. The reason for this is that the remedies used possess antiseptic properties as known to western treatment.

It is interesting to read what Robert Knox had to say on this subject. He says, "Here are professed physicians all in general have some skills and are physicians themselves. The woods are the apothecaries, shops where with herbs, leaves and rind of trees and they make all their physic and plasters with which they do notable cures". That is history. Now let us try to go back to those herbs at our doorsteps which will certainly keep us healthier and wealthier too.

At this juncture I will highlight on some details on some herbs that are relevant to Western practice also.

GOTUKOLA (Centella Asiatica)
Current researchers have found that it promotes proper functioning of central

nervous system and brain as it contains Triterpene Saponin compounds. It also promotes skin and hair nutrition and also improves immunity to control cancer. I can be use regularly when a person is suffering from Sinusitis, Catarrh and Bronchitis.

LUNUVILA (Bacope Monnaieri)
Improves cognitive function in healthy adults. It supports mental clarity and assists with concentration and attention span. It also serves as a nerve tonic and supports cardiovascular health. It has antioxidant properties too. In vitro animal studies suggest Lunuvila has neuro-protective effects and is also beneficial during times of stress and anxiety.

WENIWEL (Coscimium Fenestratum)
The creeper is found in the maritime regions in Sri Lanka and in many parts of India. It main use is as a remedy against Tetanus. It is also sued as an ingredient in many medicinal preparations, choornas, pills and kalkas. It is also used in the treatment of diarrhea in both adults and children. Scientific examinations show that plant's chief constituent is Herberine, a bitter alkaloid and is very useful in the treatment of bleeding piles and in case of excessive menstruation. Among many uses, it can be use as an antidote in the form of infusion in the treatment of snake bites.

DELUM (Punica Granatum)
Mostly known as pomegranate this is used in the treatment of sore eyes. Pomegranate is a source of some very potent antioxidants. Pomegranate tree which is said to have flourished in the Garden of Eden, has been excessively used as a folk medicine in many cultures. Fresh juice 85% moisture, 10% total sugars, 1.5% pectin and polyphenols. Pomegranate fruit polyphenols protect against lipid peroxidation in serum by direct interaction of polyphenols with LDL or indirectly by increasing serum. All these anti-oxidative and anti-atherogenic effects of pomegranate polyphenols were clearly demonstrated in vivo in healthy persons and in diabetic patients. Pomegranate juice can be beneficially used in combination with low-dose statins in hypercholesteromic patients.

ASAMODAGAM (Trachyspernum Ammi Spragues)
Asamodagam is rich in thymol. It is carminative, a stimulant and an anti-spasmodic. It is given for flatulence, colic, diarrhea and spasmodic disorders.

AMBARELLA (Spondias Pinnata)
Known as 'hogplum' or 'wild mango' in English, the pulp of this fruit is astringent as it is used as a remedy for dyspepsia. The seasoned green fruit are recommended for diabetic patients. The tender leaves are used as a poultice for old and incurable ulcers. The juice of the bark along with king coconut mil boiled together until oil formed, has been used for burns and it has been proved that the white scars on the skin due to burns disappear by applying his oil.

AVOCADO
One hundred grams of avocado contains 160 grams of K. calories. It also contains vitamins E,K,A,C & B and also it contains zinc, potassium, manganese and iron and a rare herbal chemical Beta Sitosterol. It is an important constituent of avocado. It helps to reduce LDL and to increase HDL. Avacado also helps to improve immunity and to reduce incidence of large ball cancer as it contains Omega Three fatty acids which are ideal for pregnant and lactating mothers.

JAMBOLA

Sponge between the rind and the real fruit contains active ingredient 'quercetin' and 'hesperidin', rare flavanoids used to curb the progress of breast-cancer. It contains a high amount of Vitamin C, low sodium and high potassium as ingredients. Hence it is not recommended for Kinney disease.

IRIVERIYA

Iriveriya is another plant found in gardens. It has been grown successfully as an indoor plant. The book 'Yoga Satakaya' gives details of how to use Iriveriya in various stages of diarrhea, for example, in long standing cases it is a mixture of iriveriya and kalanduru, if the diarrhea is accompanied with loss of blood. The ingredients are iriveriya, kalanduru and unripe Beli Fruit.

EKAVERIYA

(Rauwolfia Serpentina Benith)

In the 17th century, a German botanist used Rauwolfia in the treatment of hypertension, epilepsy and snake poisoning and multifarious diseases.

KAPPARAVELLIYA

Kapparawelliya is a plant worth mentioning. It is very much like Iriveriya plant in appearance and is of the same family. It is very effective in catarrhal afflictions of children. A useful prescription for whooping cough is kapparawelliya, kalanduru and heerassa and red onions taken in equal quantities, pounded and juice extracted. To this extract, an equal quantity is added together with sukiri and brought to the boil until it becomes a thick syrup. The dose is a teaspoonful or two, as required. It has a marked effect in shortening the disease. Care should be taken to ascertain that is prepared fresh every tow of three days.

ASH PLANTAINS

Ash plantain is a very useful plant as every part of it has some medicinal use. The juice extracted from the yam, trunk and the flower is used in decoctions for bleeding piles and female disorders. The ripe plantain is a sure remedy for constipation. An old Ola manuscript give tender ash plantains boiled with their skins is a remedy for cancer and stomach ulcers. In the United States, a syrup made of plantains is said to be very effective in curing coughs and bronchitis. Ash plantains should be cooked with their skins to get the best effects.

BANDAKKA (Abelmosches Esculentus)

Bandakka contains Vitamin B, Folic acid, potassium, fibre and low calories and therefore is ideal for bleeding after pregnancy, heart patients, diabetes. It is also useful in stress and body pains as it induces sound sleep.

MARGOSA (KOHOMBA)

Dr. Gunawardane's work on "Medicinal Plant of Ceylon" mentions margosa as a remedy for falling hair. The seeds are used as an insecticide and as a detergent for washing hair. Kohomba is a fine shady tree and it is mentioned in Ayurveda texts that in ancient times Kohomba trees were methodically planted in front of the house. The smell that comes from the tree is a disinfect and kills germs and helps to purify the air. Therefore plant kohomba trees where air pollution exits. Serves a duel purpose to provide purified air as an anti-pollutant .

ERABADU (Erythrina Variegeta)

This is a large and quickly growing tree with a yellowish grey bark. Leaves are triplicate and bright green in colour. This is a decorative plant and the brilliant scarlet flowers are produced when the branches are bare of leaves, hence the name

'Coral Tree'. This plant is associated with a Hindu mythological episode in which a quarrel occurred between Rakhmini and Satyabhama for the possession of these flowers stolen by Krishna. Its leaves represent the Hindu triad. The middle leaflet is Vishnu. On the right is Brahma with Siva on the left. The tender leaves are cooked with coconut milk as a vegetable for children who are one year old to prevent worm complaints. As an agent that promotes secretion and flow of milk, either the juice or the fresh leaves with king coconut milk can be given to lactating mothers.

The beautiful flowering tree grows in the dry zone and is found in Talawa lands and in low Uva. I also grows in parts of Eastern, Northern and North Central provinces. Many physiological experiments have been done with the bark of the edabadu tree in India and found that this herb acts upon the central nervous system so as to diminish or abolish its functions. When tested upon frogs the electric contractibility of the muscles was diminished and reflex action abolished. The experiments done in 1890 and in 1907 show that erabadu contains a poisonous alkaloid.

CADJU

Cadju belongs to the Anacerdiaceae family. It is an effecive remedy for scurvy caused by lack of fresh food. Cadju 'puhulan' is rich in Vitamin C and the juice is a powerful diuretic. This can be juice in urinary complaints. The kernel of the nut is like the almond and is recommended to diabetic patients. The oil extracted from the shell of the nut contains Cardol and Anacardic acid. This oil is used to remove warts and corns and o treat ringworms. In the ancient days people use Cadju oil to protect timber and books from white ants and other insects.

Herbal Anti diabetics

There are herbals which are known to reduce blood sugar. These include - Madatiya leaves and mellum., Ranawara entire plant, Masbedda leaves, Kowakka and Tebu leaves, Woodapple (Divul) and Beli, Nelli and ambarella, Wenivel , Agunakola , Bark of Kumbuk tree, Japan Batu Kola, Mella Kola mellum, Wal Kotamalli leaves, Sarana, Kiri Anguna kola, Thora Kola, Passion Fruit kola, Wattakka Tender leaves, Thumba Kola, Kohila dalu

MANIOC (Manikot Esculenta)

Anti-cancer chemicals found in manioc especially better for prostrate and urinary bladder

"Mallum" prepared from leaves are very nutritious and the bark contains hydrocyanic glycosides and therefore is must be cooked in an open vessel and also add 'Katuru Murunga' leaves and saffron to curb poisoning from hydrocyanic glycosides. Better to cook in clay pots. Manioc leaves mixed with sulphur are good for skin diseases.

RANAWARA (Cassea Auriculata)

It is a lovely bush which flowers beautifully. This is an ever-green plant. Flowers are large and yellow and grows abundantly throughout the dry zone. Five parts of the ranawara tree such as the leaves, roots, flowers, bark and seeds are commonly used in indigenous treatment. They are used especially in the treatment of diseases in the urinary organs and also in cases of constipation. In the treatment of diabetes, it gives excellent results either as a powder or as a decoction with bee-honey. The infusion of the leaves make a good cool drink. It is used in India for tanning and dyeing. It is chiefly used for heavy hides where colour is not of much importance. It dyes leather to a buff colour. It is also used to modify dyes. In the past the bark was exported from our country inconsiderable quantities for that

purposes as referred in Fredrick Lewis book of plants. The wood is not used as timber but is suitable for handles of small tools.

ADATHODA (Adathoda Visca)
The leaves contain powerful curative values especially for chest diseases. An infusion of fresh adathoda leaves with 02 or 03 tablespoons mixed with 01 teaspoon of ginger juice and bee-honey is very effective for the treatment of coughs accompanied by heavy chest. Smoking is harmful for the lungs, but dried leaves of adathoda (Pawatta) is a cure for asthma.

BELI (Aegle Narmelos)
The roots and the fruits are commonly used in indigenous medicine. Beli root is one of the ingredients in the 'Dasamula Arishta' (10 medicines). Both ripe and unripe beli fruits are used in medicine. The unripe of the half-ripe fruit is an astringent and an aid to digestion. It is used in dysentery and diarrhea. It is a very effective remedy for the irritation of alimentary canal. The ripe fruit is aromatic cooling and a laxative. In the villages of Sri Lanka, people make beautiful walking-sticks and handles for tools.

In Pakistan, the yellow dye obtained from the rind of the unripe fruit is used in calico paintings. An essential oil, Martinelle Oil, is distilled from the rind. The pulp has detergent properties and so it is used in washing clothes and also a varnish.

JAK (Artocarpus Heterophyllus)
It has two verities, soft 'Vela' and hard 'Varaka'. The varaka of the hard jak is more popular than the soft Jak. When tender it is called 'polos'. This is a delicious curry among the Sinhalese and Tamils. In the diet chart of ancient kings this 'polos curry' was never absent. The king's menu was a very balanced diet and it was arranged by educated 'veda ralas' ho served the king. Polos is a curry which is relished by expectant mothers. The curative value of polos is that it is digestive, nutritious and helps to enrich breast milk. 'Varaka' has a different medicinal value. The varaka bark is used for sprains and the leaves are dried and powdered and poured like coffee to be used by diabetic patients.

NIVITI – SPINACH (Basella Alba)
It is a one such vegetable which gives energy and strength. Spinach has come to us from the Middle East. It was grown in the 11th century in Spain and in the 15th century in France. It is rich in protein and helps in digestion. It is a stimulant for all secretions in the stomach, the liver and the pancreas. It is also very rich in minerals. Most people know that is contains lots of iron but according to scientific experiments it also contains phosphorous, iodine and many other elements essential to keep the system in good shape. In the old texts of ayurveda spinach is mentioned as "Sukkra Janaka" which means that it is an aphrodisiac. Spinach is one of the basic medicines for insomnia and insanity. Nivithi generally grows in cold and water-logged places. It may also be grown in pots as an indoor plant.

OLENDER (KANERU)
(Nerium Odorum)
According to Sinhalese 'Materia Medica' this belongs to the same family as 'Araliya'. The plant contains properties from which a kind of rat-poison is manufactured in Pakistan. The roots are more poisonous than the other parts of the plant. Scientific experiments done in India have shown the leaves contain two glycosides, "Nerrin" and "Diedrin". In his book Dr. William Thompson M.D. says that the active properties in the leaves include cardiac glycosides. They help in cases of heart disease and as a circulation diuretic. Oleander was widely used for this purpose in Europe instead of digitalis.

Herbal anti-hypertensives

There are many herbals known to reduce blood pressure. These include - Juice of shoe-flower Juice of beetroot, Fiber rich foods, Ekaveriya, Raufolfia, Uguressa , Beli roots, Rasakinda Babila roots, Nidikumba roots, Thotila roots.

Herbal Anti-Fungals

- Paste of Ehela (Cassia Fistula) applied over parts affected by ring worms.
- Paste of Ath-Thora (Atylosia Trinevrin) mixed with bee-honey or paste of roots with sandalwood is very effective.

The recent valuable article written by Manel Tampoe "Towards an Ayurvedic Renaissance" is an eye-opener to those well wishers who want to nurture Ayurvedic back to revitalized medical system. The large and valuable contributions with indigenous physicians would give to medical knowledge in this country is still awaited.

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Melioidosis in Sri Lanka

Dr Enoka Corea

What is melioidosis?
Melioidosis is a tropical infection caused by the saprophytic soil bacterium Burkholderia pseudomallei. It is found between 20°N and 20°S of the equator. In high endemic areas, such as Thailand, Malaysia and Northern Australia, it is a significant cause of community-acquired pneumonia and sepsis and has a high mortality. Its apparent rarity in other tropical regions may be due to lack of clinical awareness and microbiology services.

How is infection acquired?
Infection is acquired accidentally, during occupational, recreational or lifestyle exposure to soil, mud or water containing the bacterium. B. pseudomallei enters the body by percutaneous inoculation, inhalation or ingestion. Initial infection following exposure may be asymptomatic and the bacterium may remain dormant for many months, years or even decades before activation in a fulminant form. However, in most cases, the incubation period varies from a few days to weeks.

Are there any risk factors for melioidosis?
B. pseudomallei is an opportunistic pathogen, and though clinical disease is seen in healthy individuals it is more common in persons with chronic co-morbidity, particularly uncontrolled diabetes mellitus. Other risk factors include renal or liver disease, excessive alcohol consumption, chronic lung disease and thalassemia.

What are the common presentations?
Melioidosis may involve any system and is often multifocal. Clinical presentation ranges from severe to mild and from acute to chronic. Typical presentations include fulminant septicemia, severe community acquired pneumonia or lung abscess, single or multiple abscesses of the superficial or deep tissues including skin and soft tissue, salivary gland, liver, splenic or cerebral abscess, musculoskeletal disease such as abscess of the psoas muscle, septic arthritis or osteomyelitis, skin infection, genitourinary infection and lymph node suppuration. Patients often have multifocal involvement, with combinations of the above presentations, with or without blood stream infection. Less common presentations include brain stem encephalitis, myelitis, urinary tract infection and prostatitis. Melioidosis is one of the differential diagnoses of tuberculosis.

Relapses after apparent cure are characteristic and a history of recurring infection may be elicited.

Does it occur in outbreaks?
Melioidosis is usually sporadic, in keeping with the accidental and opportunistic nature of the infection, with a sharp increase in the number of cases during the rainy season. Case clusters may be seen after severe weather events, probably due to aerosolisation of bacteria and increased inhalation risk. Case clusters were reported in many countries following the 2004 Indian Ocean tsunami. Nosocomial outbreaks and point source outbreaks from untreated natural water sources have been described in Australia.

What is the mortality of melioidosis?
Melioidosis is a potentially fatal infection and mortality can be as high as 50%. However early diagnosis and appropriate treatment reduce morbidity and mortality considerably.

Is melioidosis found in Sri Lanka?

Between 1927 and 2006 only three cases of local transmission of melioidosis were reported. However, after 2006 more than 285 culture proven cases and a large number (>100) of cases diagnosed by high antibody titres have been identified. B. pseudomallei appears to be ubiquitous in soil in Sri Lanka and cases of melioidosis have been reported from all provinces. Genotyping of strains show that the bacterium is an established endemic pathogen in Sri Lanka and that the emergence of melioidosis in the recent past is not due to recent introduction.

Profile of patients with melioidosis in Sri Lanka				
Presentation	**Demography**	**Co-morbidity**	**Exposure**	**Geography**
Pneumonia Septicemia Septic arthritis Osteomyelitis Skin and soft tissue abscesses Abscess of spleen, liver, kidney, Cerebral abscess Lymph node abscess Salivary gland abscess	Male 40-50 years Rural	Diabetes Renal disease Liver disease / alcoholism Chronic lung disease Thalassemia	Farmer / cultivator/ home garden Farming family Construction labourer Police or defense forces Drivers and motorcycle riders Flooding Bathing in tanks or rivers	Gampaha District Kurunegala District Puttalum District Battiacaloa District Colombo District Kalutara District Polonnaruwa District

What are the geographic and demographic features of patients with melioidosis in Sri Lanka?

Most cases are seen in rice growing areas and it seems to be less common in rubber and tea growing districts. Infection occurs in all age groups, including children, and in both sexes, though the highest incidence is seen in middle aged males. Persons with occupational exposure to soil, such as farmers and cultivators and house or road construction workers, are at special risk, as are persons in the police and defence forces. Recently we have found an increased incidence in three wheeler drivers and motor cyclists, probably due to exposure to dust and in persons affected by flooding. However, it is clear that the majority of the Sri Lankan population is exposed to risk, as even areas classified as 'urban' are contiguous with cultivated lands and most of the population is directly or indirectly engaged in cultivating food crops for commercial or domestic use. The custom of walking barefoot and of using natural sources of water for drinking and bathing may also contribute to an increased risk of infection. The rapidly increasing incidence of diabetes is likely to result in an increase in clinical disease in the future.

Is there a seasonal trend in incidence?

Melioidosis occurs throughout the year but the incidence appears to increase with the onset of the monsoon rains, especially the north east monsoon. Peak number of cases occurs about 1 month after peak rainfall.

How can we make the diagnosis?

As the clinical presentation and baseline laboratory tests in melioidosis are non-specific and may mimic many other infectious

diseases, the diagnosis of melioidosis can only be established by the microbiology laboratory. This is best achieved by isolation of the pathogen from patient specimens. B. pseudomallei is not fastidious and is easily isolated on all common culture media used in a clinical microbiology laboratory. Specimens for culture include blood, pus from abscesses, sputum, urine and infected tissues. Specimens from sterile sites should be inoculated into an enrichment medium such as brain heart infusion broth and incubated for up to 2 weeks. Although selective media are described for selective isolation of B. pseudomallei they do not increase the yield from sterile sites and may not be cost effective for use on specimens from non-sterile sites.

How do I treat melioidosis?

There are well established antibiotic treatment guidelines for the treatment of melioidosis (see tables below). Treatment comprises an early phase of acute treatment with parenteral antibiotics such as ceftazidime OR imipenem OR meropenem, with additional adjunct therapy with oral cotrimoxazole for deep seated focal involvement. Duration of the intensive phase ranges from 2-8 weeks. This is following by 3-5 months of eradication therapy using a combination of any two of oral cotrimoxazole, doxycycline or amoxicillin / clavulanic acid.

(See tables below for recommended antibiotics and duration of therapy).

Application	Agent	Amount *	Route	Frequency	Duration	Variations	References
Phase 0: post-exposure prophylaxis							
Within 24 hr of high-probability exposure [a]	trimethoprim-sulphamethoxazole	320:1600 mg	p.o.	12 hourly	3 weeks [a]	amoxicillin/ clavulanic acid if allergic to trimethoprim-sulpha-methoxazole	[30,31]
Phase 1: acute & severe infection, induction stage							
Alternative agents for primary therapy	Ceftazidime	2g	i.v. [b]	8 hourly	≥ 14 days	4-8 weeks for deep infection	[1,2,3,5]
	OR Meropenem	1g (2g for C.N.S. infection	i.v.	8 hourly	≥ 14 days		[2,6]
	OR Imipenem	1g	i.v.	8 hourly	≥ 14 days		[5]
Adjunct therapy for deep-seated focal infection	AND trimethoprim-sulphamethoxazole	320:1600 mg	p.o. [c]	12 hourly	≥ 14 days	for neurological, prostatic, bone, joint infections	[1,2,3]
	AND folic acid	5 mg	p.o.	daily			
	AND consider G-CSF [d]	263 µg	s.c.	daily	3 days	Within 72 hrs of admission	[20, 21]
Step-down combination for outpatient or extension clinic use	Ceftazidime	6 g in 240 mL Normal saline	i.v.	24 hour infusion	2-4 weeks	For hospital in the home (HITH)	[9]
	AND trimethoprim-sulphamethoxazole	320:1600 mg	p.o.	12 hourly			
Phase 2: eradication stage							
2 of , in order of preference, after Phase 1 or for primary use in superficial infections	trimethoprim-sulphamethoxazole	320;1600 mg	p.o.	12 hourly	≥ 3 months[e]	Subject to antibiotic susceptibility	[13]
	doxycycline	100 mg	p.o.	12 hourly	≥ 3 months		[13]
	amoxicillin/ clavulanic acid	500/125 mg	p.o.	8 hourly	≥ 3 months		[14,15]
	folic acid	5 mg	p.o.	daily	≥ 3 months	With trimethoprim-sulphamethoxazole	[30]

* doses may require adjustment in renal failure [a] suggested by expert consensus, but lacks trial-based clinical evidence; [b] doses provided as guide only based on 70kg male; [c] i.v. = intravenous, p.o. = oral, [d] G-CSF = granulocyte- colony stimulating factor; [e] some recommend 5 months eradication therapy.

Table 1. Darwin melioidosis guideline.

Antibiotic Duration-Determining Focus		Minimum intensive phase duration (weeks)[a]	Eradication phase duration (days)
Skin abscess		2	90
Bacteremia with no focus		2	90
Pneumonia	without lymphadenopathy[b] or ICU admission	2	90
	with either lymphadenopathy[b] or ICU admission	4	90
Deep-seated collection[c]		4[d]	90
Osteomyelitis		6	180
Central nervous system infection		8	180
Arterial infection[e]		8[d]	180

a. Clinical judgement to guide prolongation of intensive phase if improvement is slow or if blood cultures remain positive at 7 days.
b. Defined as enlargement of any hilar or mediastinal lymph node to greater than 10mm diameter.
c. Defined as abscess anywhere other than skin, lungs, bone, CNS or vasculature; septic arthritis is considered a deep-seated collection.
d. Intensive phase duration is timed from date of most recent drainage or resection where culture of the drainage specimen or resected material grew *B. pseudomallei* or where no specimen was sent for culture; clock is not reset if specimen is culture-negative. On 1st October 2010, the minimum duration for deep-seated collection changed from 2 to 4 weeks from last such drainage/resection.
e. Most commonly presenting as mycotic aneurysm.

Pitman MC, Luck T, Marshall CS, Anstey NM, Ward L, Currie BJ (2015) Intravenous Therapy Duration and Outcomes in Melioidosis: A New Treatment Paradigm. PLoS Negl Trop Dis 9(3):e0003586. doi:10.1371/journal.pntd.0003586

How is B. pseudomallei identified in the laboratory?

Colony appearance on blood and MacConkey agar, Gram stain morphology, oxidase reaction and a typical antibiotic sensitivity profile are sufficient for presumptive identification of B. pseudomallei in the clinical microbiology laboratory.

Growth on most media occurs after overnight incubation in ambient air at 37°C. Colonies at 24 hours are usually pin point in size. After 48 hours colonies are larger (2mm) and circular with an entire edge. On blood agar, the well is usually chalky white with a metallic sheen and shows underlying beta haemolysis. The culture has a characteristic earthy odour. After further incubation the colonies become umbonate and then acquire a concentric appearance. Wrinkling, if it occurs at all, is seen only after 5-7 days of incubation at room temperature. On Mac Conkey agar colonies are pin point in size and have a non-lactose fermenting appearance at 24 hours, changing to dark pink (similar to lactose fermenting bacteria) at 48 hours. Some strains give rise to colonies with varying morphology and may be dismissed as mixed cultures. A minority of strains differ considerably from the typical one described and can appear serous to frankly mucoid. These strains give a non-lactose fermenting appearance on MacConkey agar.

On Gram stain, the bacterium is a Gram negative bacillus showing a characteristic 'safety-pin' appearance due to lack of stain uptake in the centre of the bacterium caused by inclusions of beta hydroxybutyrate. The isolate is oxidase positive, which may be more apparent if the test is done with a sweep from the well than from an isolated colony. It is important to perform the oxidase test even in cultures that appear lactose fermenting on Mac Conkey agar, or B. pseudomallei may be missed.

The typical antibiotic sensitivity pattern of resistance to gentamicin, polymyxin and colistin and sensitivity to coamoxyclav may be demonstrated using the disc susceptibility testing method. A vancomycin disc could be included if it is necessary to exclude the possibility of the isolate being a spore bearer.

Commercial identification systems such as API20NE or Rapid ID NE often misidentify B. pseudomallei and are unreliable. Definitive identification requires PCR amplification and detection of genes specific to B pseudomallei. The detection of two separate genes is recommended. This facility is available in Sri Lanka.

What are the pitfalls and dangers in the laboratory diagnosis of melioidosis?

Unfamiliarity of laboratory personnel with this bacterium may lead to misidentification of B. pseudomallei as a Pseudomonas sp., E.coli or coliform. Isolates may be discarded as insignificant contaminants, especially if found in mixed culture in specimens from non-sterile sites. Pellicle forming isolates in blood culture broths may be dismissed as sporing contaminants.

Laboratory acquired infection has been well documented, usually through exposure to aerosols. Once a culture is suspected to be B. pseudomallei, further handling should be done in a Class II safety cabinet.

Presumptive isolates could be sent to the reference laboratory c/o Dr Enoka Corea, Microbiology Department, Faculty of Medicine, University of Colombo for definitive identification by PCR.

Is there an antibody test for melioidosis?

Antibodies to B. pseudomallei are detected using the indirect haemagglutination assay (IHA). Antigen for the test is made in-house, from local strains. While the IHA titre of infected persons and exposed persons may overlap, antibody tests are useful to support a clinical diagnosis in patients where culture is not possible due to prior antibiotic treatment or inaccessibility of specimens. A single low titre of up to 1:160, may be seen in acute infection but may also represent environmental exposure to B. pseudomallei. Higher titres (≥1:320) are likely to depict acute infection. Antibody tests may be negative early in acute infection so a negative test does not exclude infection. A blood sample of around 2ml of blood taken in a plain bottle may be sent to the reference laboratory c/o Dr Enoka Corea, Microbiology Department, Faculty of Medicine, University of Colombo for antibody testing. If there is any delay it is preferable that serum should be sent after separation of the clot.

Conclusion

Melioidosis is no longer an 'emerging' infection in Sri Lanka but can be considered an endemic disease, with more than 300 culture and antibody proven cases diagnosed since 2006. It is widely distributed throughout the island and affects all age groups. Mortality has dropped from 50% to around 20% during this period. It should be included in the differential diagnosis of febrile illness in Sri Lanka and suspected cases referred to a clinical microbiologist for microbiology laboratory support for definitive diagnosis.

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Dengue Haemorrhagic Fever- Two unusual Cases

Dr. N P S Gunaratne

I am reporting here two unusual presentations of Dengue Haemorrhagic Fever in two patients that I treated at a private hospital in Colombo during the last two years.

The first, an eight year old girl with a history of fever for three days accompanied by nausea. She had vomited once. She also complained of abdominal pain and frequency of micturition. On examination she was afebrile and had no abnormal physical signs. The blood pressure was 90/60mmHg and her weight 31kg. The results of investigations done prior to seeing me were as follows:

Hb 12.9g/dL
PCV 37%
Platelet Count 134,000
WBC 3080, Neutrophils 72%, Lymphocytes 27%
Dengue NS1 Antigen – Positive
Urine FR- N.A.D

Because the Dengue NS1 Antigen test was positive, the Platelet Count of 134,000, the abdominal pain, nausea and poor oral intake, she was admitted to hospital the same night.

On admission, she was started on an IV Drip of Normal Saline at 60ml per hour and given Domperidone three times per day for the nausea, Zellox 11 syrup (antacids) because of the abdominal pain and Paracetamol as necessary for fever.

She was also put on a Dengue chart, Urine Output chart and an Input chart. On the day after admission which was day 4, the patient had fever, abdominal pain and vomiting. On examination, there were no abnormal physical signs. The BP was 100/70 mmHg. The urine was sent for a Full Report and for Culture and ABST. On that day, the patient passed a semisolid stool. On the next day (day 5) the patient had lower abdominal pain with lower abdominal tenderness but no flank dullness. The bowel sounds were normal. The blood pressure was 100/70mmHg and Urine Output 1ml/kg/hr. Because of the poor intake of solids and oral rehydrants, glucose was added to normal saline IV. On the evening of the same day the following results were noted:

	On day 5	Prior to admission
Hb	14.4g/dL	12.9g/dL
PCV	42%	37%
Platelet Count	42,000	134,000
Serum Albumin	3.4g/L	
Liver Enzymes(SGPT, SGOT, γGT)	Slightly elevated	

On day 6, in the morning when the fever had settled, the Platelet Count dropped to 35,000 and the Serum Albumin was low at 3.21g/L. On physical examination, the breath sounds were diminished at the base of the (R) lung posteriorly. I requested an Ultrasound Scan of the Chest and Abdomen by the radiologist. The report was as follows: Small (R) pleural effusion suggestive of Dengue Hemorrhagic Fever-Leakage Phase. Gall bladder wall

thickened. However, the BP, heart rate and respiratory rate were all normal and the patient looked well and there was no evidence of any bleeding.

On the next day (day 7) the Urine Culture report read Pseudomonas aeruginosa 105 CFU/ml isolated. As the organism was sensitive to Ceftazidime she was given this antibiotic intravenously, every eight hours.

On the same day, as there were two loose stools and some tenderness in the hypogastrium, a stool was sent for a Full Report, Culture and Cryptosporidium. The stool full report and culture showed no abnormality but Cryptosporidium oocysts were reported as positive on day 8.

By the evening of day 8, the child was well with no fever, vomiting, abdominal pain, loose stools, and frequency of micturition. The IV fluids had been stopped that morning. All the blood investigations had come back to normal, except the Platelet Count which had improved to 107,000, but was still low, and the Liver Enzymes which were still elevated.

She was discharged from hospital the same night on Vitamin B Complex syrup and Ciprofloxacin. The latter drug was given as the Ceftazidime IV had to be stopped and this was the only oral antibiotic to which the Pseudomonas aeruginosa isolated on Urine Culture, was sensitive.

Seven days after discharge from hospital, Urine Culture was done and showed no growth and Stool Examination for Cryptosporidium was negative. No treatment for Cryptosporidium was given because the mild diarrhoea subsided without treatment.

Conclusion

This eight year old girl from Rajagiriya had Dengue Haemorrhagic Fever together with a Urinary Infection due to Pseudomonas aeruginosa, and a Bowel Infection due to Cryptosporidium, all at the same time. Reference to the world literature shows that Dengue Haemorrhagic Fever with two concurrent infections in children is not common. However, I give a case report from Bangladesh of Dengue Haemorrhagic Fever with two concurrent infections in a child.[1]

My second unusual case of Dengue Haemorrhagic fever was a 14 year old girl who was brought to me on the 5th day of fever. On examination there were no abnormal physical signs. A blood report done elsewhere on the second day of the illness showed the following results.

Hb 12.2g/dL
PCV 36%
Platelet Count 186,000
WBC 8130, Neutrophils 88.7%, Lymphocytes 7.23%, Monocytes 3.29%, Eosinophils 0.54%
Dengue NS1 Antigen- Negative

I repeated the FBC and got the C - Reactive Protein done. The results were as follows:

Hb 14.5g/dL
PCV 40.8%
Platelet Count 110,000
WBC 2340, Neutrophils 40%, Lymphocytes 55%, Monocytes 3%, Eosinophils 2%
C-Reactive Protein 24.2mg/L

The parents were concerned about the results of the investigations and requested that the child be admitted to hospital. This was done on the same evening. On examination there were no abnormal physical signs.

The results of the FBC and Liver Profile done on the following morning and on the next two days are shown in the chart below:

The lowest Platelet Count of 71,000 was on the evening of the 6th day of illness and the highest PCV of 41.5% on the 7th day. Though

FBC

	On day 6 morning	On day 6 evening	On day 7 morning	On day 8 morning
Hb (g/dL)	14.1	14.0	14.1	13.7
PCV (%)	40.0	40.4	41.5	39.5
Platelet Count	92,000	71,000	90,000	118,000
WBC	2700	3320	5440	7370
Neutrophils (%)	20	20	20	21
Lymphocytes (%)	70	75	73	72
Monocytes (%)	6	4	3	3
Eosinophils (%)	4	1	4	4

Liver Profile

	On day 6 morning	On day 7 morning	On day 8 morning
Total Protein (g/dL)	6.49	6.27	6.63
Albumin (g/dL)	3.98	3.68	3.96
Globulin (g/dL)	2.51	2.59	2.67
Alkaline Phosphatase (U/L)	101	90	100
Bilirubin (mg/dL)	0.43	0.41	0.42
SGPT (U/L)	99	75	70
γGT (U/L)	200	162	100
SGOT(U/L)	126	82	90

these were abnormal, the platelet count of 71,000 is not very low and the PCV of 41.5% is not very high. The Liver Profile showed an elevation of the liver enzymes on the 6th day, which decreased on the 7th and 8th days.

The Ultrasound Scan of the chest and abdomen done on the 6th day, was reported by the radiologist as follows: Mild Ascites, Gall Bladder wall Thickened (6mm).No pleural effusion.

The Dengue RT-PCR done on the 6th day of the illness was negative.

Intravenous Normal Saline with 5% Dextrose was given on day 6 and day 7 as oral intake was poor.

On the 9th day after the onset of the illness, one day after discharge of the patient from hospital, Dengue IgM and IgG antibody tests were done and both were positive.

This was therefore an unusual case of Dengue Haemorrhagic Fever with negative NS1 Antigen and Dengue RT-PCR, but confirmed as Dengue by the positive Dengue IgM and IgG antibodies.
Experts on Dengue Haemorrhagic Fever have told me that only 70% of Dengue patients are Dengue NS1 Antigen positive and 90-95%

are positive for the Dengue RT-PCR test. Therefore a negative Dengue NS1 Antigen and a negative Dengue RT-PCR does not exclude Dengue Haemorrhagic Fever and the necessary investigations and management for Dengue Haemorrhagic Fever must be done in a suspected case, as the Dengue IgM and IgG tests are positive only after the acute phase of the illness.

Conclusion

Dengue Haemorrhagic Fever may occur without a positive Dengue NS1 Antigen test or a positive Dengue RT- PCR test. The Dengue IgM and IgG tests being positive on the 9th day of the illness, together with blood and ultrasound scan results done earlier confirmed that this illness was Dengue Haemorrhagic Fever.

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Changing your paper based medical record to an Electronic Medical Record in general practice

Dr. Ananda Perera

This title assumes that you already have a paper based medical record. It also assumes that you want now to adopt an electronic medical record system (EMR) for your practice. If it is so then of course it is extremely simple. The solution is you simply have to look for a person who will do that for you or you yourself will have to do it. Having been primary care physician for several decades and an intimate knowledge about the health information technology, the answer for the majority of the general practitioners in Sri Lanka would be getting software vendor to do it for you. This immediately puts you into the role of a customer and the other person into the role of a software vendor.

Now what you want is a product which will help you to carry on what you have been doing as a general practitioner for last several decades. The first CAVEAT :

CAVEAT 1

Look for vendor who can deliver results : that is a computer based record where you can enter what you have been entering for several decades within the time and resource constraints (money, after all paper is very cheap, ancillary staff where some of them at least may have to interact with the system as following prescription orders and lab orders etc like staff nurses, clerks and MLTs) under which you have been working.

ADVICE 1

Give a sample of your prescriptions, lab requests, patient clinical data, patient identification data etc to the prospective seller.

The second caveat is something to do with what all customers should possess : know what you want and act within your capabilities – financially, socially, culturally and technically. That is a skill which it is well known that most software buyers do not have. This ultimately brings us headlong to the question of technology push or technology pull.

CAVEAT 2

Articulate clearly what you want without assuming anything what the seller might say. Because customer is the king and beware of the seller who will sell nails along with hammers unless of course you want it and become totally dependent on the seller who poses as a professional in system analysis – whatever it means for you.

ADVICE 2

You simply assume you are the master of your professional life and it is very likely any software vendor currently in Sri Lanka has the capability of doing so (with excuses for those who are true system analysts and software vendors)

The third caveat deals with hallmarks of

a vendor who is going to do what so for we have specified. A true system analyst and a software vendor will have to know not only about your work, workflow and personal values and attributes but also all other systems like nurses, MLTs, labourers, and above all your patients. How can that person achieve that knowledge ?

CAVEAT 3

The true software vendor who is going to sell you something what you want so personal will have to sit with you during your consultations for about several weeks to months

ADVICE 3

For a software personnel to have that knowledge at a minimum will have to have a degree in software engineering and majored in knowledge engineering. So go for the authorship of the software you are going to buy.

The fourth caveat is a common problem which all of us have whether we are professionals, physicians, software vendors or system analysts. All of us hardly articulate what we want and need – not only in the filed of software but also in the fields of Medicine, Architecture, Engineering or even Law. In all of these fields the first fundamental in human interactions is the adage "Convert the ill defined problem into a well defined problem in terms of the theoretical foundations of your profession"

CAVEAT 4

Know your exact problem. Why are you changing from a paper based to an electronic medium.

ADVICE 4

No software vendor nor any system analyst nor anybody other than you probably will know this.

This brings us to the turning point of this presentation. Why are you changing from a paper based to paperless ? Is it because somebody told you to do so at a seminar or a workshop ? Or all the other doctors are turning into electronic medical records and you also simply want to roll on with the waves ? Or else you are just plain fed up of the way the things are and you just want a change in your work ? Or else you did an audit and found that your performance is deteriorating ? Or else your neighboring competitor has just bought a system and the software vendor challenges you to board the wagon ? Or just that you want to impress your patients with an aura of high technology ? The possibilities are limitless as all of us are humans and as varied as humans.

CAVEAT 5

Articulate for yourself why do you want to do this – going from paper to hybrid or totally paperless.

ADVICE 5

This cannot be given an answer. Because you are your own boss.

If you have read this far and if you are still with me you probably might also be interested in other ways of changing your paper based practice into the paperless or a hybrid system of implementation. So far we have been discussing about a custom solution or a very personal solution for a personal requirement. But as you know the world is not so very customized and individualized nor is it so very chaotic.

The word processors, spreadsheets, paint programs, email, calculators and Internet was discovered not for individuals. They have gained popularity for what they are worth for. For meeting current requirements of majority of the masses. Take for instance a word processors – not every body wants all the functionality available in a given program. In fact it is said that most of the people use only about 30% of the functionality of a true word processors. So in the real world of software market there is always the tension between individual and mass requirements. So is it in the world of EMR in particular and medical software in general.

To focus on the mass requirements of the electronic medical records there are 2 principles which we have to bear in mind – principle of quality and the principle of meaningful use. For average physician in the world quality of health care delivered is an overarching requirement. Meaningful use refers to a set of functions which an average physician in the world wants to have in an EMR.

While it is not necessary to deal in a detailed manner on these 2 principles it is self explanatory and self evident as well. But to complete a gross summary of the entire presentation it is suffice to say the following : all EMRs are supposed to have an in-built functionality which ensures and enhances the quality of healthcare delivered. It is probably unthinkable to have EMRs which will not deliver quality healthcare. On the other hand whatever said and done our clinical workflow whatever the individual variations and quirks we may have has some common underlying pattern. For instance we collect clinical data, we order investigations, we enter results of lab data, we prescribe, we advise our patients, we provide health education for our patients, we notify certain diseases to the relevant authorities, we issue medical certificates, we recommend letters for employment, insurance etc etc. Meaningful use refers to this core functionality of our routine day to day clinical activities. So a basic EMR system should have functionality to enter identification data, clinical data, lab data, miscellaneous order entries, prescribing and follow up. But there are systems and in fact most of the currently available systems far exceed this basic functionality.

But all these come with certain fundamental prerequisites. First as it is not personalized there is a requirement for change. The degree of change required is directly proportional to the distance you are away from the population norms. Second is the requirement of a learning curve. Here again the slope is proportional to the distance one is away from the population norms. Third the unique skills required to adopt a new environment – typing skills, new ways of patient interviews, new models of patient physician relationships, new ways of prescribing and new ways of professional interactions. Fourth is the adaptation to a new business model of health care delivery. It is well known most physicians will look at the investment in an EMR as something of a no-return. In fact unfortunately this is true. The real beneficiaries of a well functioning EMR system are patients not physicians. The argument that this in turn will cause many more patients for individual practitioners is a no-brainer Fifth is the survival or extinction in the face of a new sociopolitical pressures as recently happened to the American physicians.

On the balance of probabilities the best advice is to go for mass solutions and customizations. In the meantime for your own sake keep in mind :

1) EMRs are specialty specific – if someone is promoting a EMR for all the specialties it is a myth

2) The prices of decent EMRs range from free to close up to several hundred thousands. Choice is yours

3) Focus on mobile products as they are future proof

4) Focus on web based products as they are probably future proof

5) Focus on availability of what of the meaningful use criteria mentioned earlier

6) Focus on usability – that is whether you can use it in your own practice with your personal practice constraints

7) Never feel shy about the ignorance of health information technology – when you buy a vehicle you are not supposed to be engineer in aerodynamics

8) Always ask for a time for evaluation in your practice in usual circumstances so that you can get a feel for the change ahead

9) It is probably wise to focus on web based systems rather than on desktop systems for average Sri Lankan GP

10) Never get shaken up by tech speak – it is not humiliation

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Case Report
A case of Pancreatic Adenocarcinoma

Dr. Prageesha Gamage, Dr. Sanath Hettige

Introduction

Pancreatic adenocarcinoma is a rapidly fatal malignancy, which accounts for 5 year survival of 5% and a median life expectancy of less than one year [1, 2, 3, 5, 6]. This poor outcome is due to many factors. Pancreatic cancer disseminates to distant sites early in its course, therefore at the diagnosis many are at a later stage of the disease [2, 3, 4, 6]. Its response to radiotherapy and chemotherapy are minimal [2, 3, 4]. Due to being diagnosed at the disseminated or locally advanced stage, 80% of patients are not suitable for curative surgical procedures [2, 4, 5]. Even in surgically resectable disease, 5 year survival is only 20% [1, 2, 3, 5]. On the other hand, the surgical procedures are technically difficult and debilitating. Adjuvant and palliative chemotherapy can be offered but lengthen the survival only in a few months [3, 4, 5, 6]. There are no effective screening strategies available to detect pancreatic carcinoma at its early stage as well.

In many incurable diseases, patients tend to use alternative and complementary medicine. Carica papaya has shown many medical benefits including anti-tumour activity and immunomodulatory effects [7, 9, 11]. It is used worldwide for treatment of many cancers namely prostate, breast, colorectal, gallbladder and cervical [11]. It has also shown to be effective in infections of dengue and malaria as well as cardiovascular diseases and wound healing [10, 11]. There is evidence that many parts of Carica papaya (papaw or papaya) plant; fruit, seed, leaf, latex, peel and root extraction have been used [8, 10]. In world literature, there are anecdotes of patients with advanced cancers who have achieved remissions following use of Carica papaya leave extraction but very little scientific data is available [9].There are some cases reported with regard to , increased survival in various malignancies, stomach, pancreas, lung, liver and blood but no reported randomised controlled studies [11]. One in vitro study is available on cytotoxic effects of Carica papaya [9].

Dr Sanath Hettige, consultant family practitioner, has observed the haemopoetic activity, resulting increase of platelet count and white cell counts in patients with various malignancies on chemotherapy when Carica papaya leaf extract is given concurrently [14, 15, 16]. He has noted a longer survival in those patients who were started Carica papaya leaf extract as well. He has conducted studies and published on the use of Carica papaya leaf extract in dengue patients with low platelet and white cell levels [14, 15, 16]. He is now manufacturing syrups, tablets and capsules from Carica papaya leaf and holds patent rights to use in dengue fever and other relevant diseases [16]. With experience of haemopoetic activity and observation of longer survival, he now prescribes Carica papaya leaf powder capsules (Papayacap) as a concurrent treatment in all his patients with malignancies irrespective of the other treatment modalities.

As a MD trainee, of Dr Sanath Hettige during my postgraduate training period I came across a patient with adenocarcinoma of pancreases with disseminated disease who was treated with Carica papaya leaf powder capsules.

Case presentation

Mr RP 62 years factory manager diagnosed with a pancreatic carcinoma one year and 8 months back as an incidental finding at a tertiary care hospital. He was investigated for bladder calculi, during ultrasound scan other than the prostatic urethral calculi, found a splenic hilar cystic and solid mass with calcification, suggestive of Bosniak 4 cyst in left kidney or pancreatic tail neoplasm. Contrast Enhanced Computer Tomography (CECT) revealed a mass with predominant cystic areas with enhancing solid areas and calcification, between the tail of pancreas and spleen. The mass was infiltrating the hilum of the spleen, most probably arising from the tail of pancreas. Non-enhancing focal lesion of the liver and mild ascites were identified too.

He had no features of abdominal pain, anorexia, weight loss, jaundice, acute or chronic pancreatitis or diabetes. He is a known patient with hypertension which is under control with medication. He was a smoker of 20 pack years.

Surgical treatment planned was to proceed with distal pancreatectomy, splenectomy and liver lesion resection. He was assessed for fitness for surgery and general anaesthesia. Surgery was planned in one month duration since the ultra sound finding of the mass. The patient refused surgery at the last moment and preceded with laparoscopic assessment of pancreatic tumour with patient's informed written consent. Peritoneal fluid histopathology expressed inflammatory cell and no malignant cells. Peritoneal and gastro colic omentum deposits' histopathology revealed moderately differentiated adenocarcinoma with possible primary pancreatic tumour or upper gastro intestinal tumour. The patient was referred for oncology opinion from the surgical team. He is looked after now by the oncology team and receives chemotherapy for one year and 8 months.

Initially he was started a combination chemotherapy with Gemcitabine and Oxaliplatin. He had no symptoms related to pancreatic malignancy and CA 19-9 was within the normal range. After one year of starting chemotherapy with rise of CA 19-9 up to 728 IU/ml, his drugs were revised and shifted to Paclitaxel. With third dose of Paclitaxel he developed numbness of legs and hands.

He was started Carica papaya leave extraction since the diagnosis of pancreatic mass as a concurrent treatment while being prepared for surgery as well as with chemotherapy. Initially in the form of homemade aqueous extraction for two months followed by Carica papaya leave capsule which was taken on daily basis (one 360mg dried carica papaya leaf cap three times a day).

He has quit the job at the diagnosis of disease as he was working outstation, which caused practical difficulties in receiving treatment with inpatient care. Now he is employed for last three months in the same field, stays outstation many kilometres away from his family and home, visits every other weekend using public transport services and leads an independent life.

He has undergone CECT scan three times since the diagnosis; during last one and 7 months, the pancreatic mass remain the same time. He has no symptoms of pancreatic malignancy. Though Positron Emission Tomography Scan (PET scan) should be performed to comment on the disease and treatment progression, it was not available due to high cost.

Discussion

Biopsy proven metastatic pancreatic adenocarcinoma has a poor prognosis with life expectancy of few months [2, 5, 6]. Epigastric or left upper quadrant abdominal, back pain, obstructive jaundice are common presentations of the primary disease as well as cachexia and asthaenia predominates with the disease progression [2,5]. It has a dynamic presentation with sudden change

in clinical status by rapidly worsening pain, biliary obstruction, gastric outlet obstruction, thromboembolism, and ascites [2]. Metastasis to distant sites at the initial stage of the disease minimise the chance of surgical resection [2]. Locally advanced disease involving the adjacent vascular structures such as superior mesenteric artery, coeliac axis, and superior mesenteric – portal vein confluence also creates an unresectable disease ending up with palliative treatment [2].

Though it was an incidental finding, his disease was histologically proven as a pancreatic adenocarcinoma with peritoneal dissemination. Radiological evidence suggested liver involvement as well. According to the natural history of pancreatic adenocarcinoma, this is a very debilitating disease with a short life span expectancy of few months [1, 2]. Surprisingly he is free of any symptom related to pancreatic adenocarcinoma. According to the CECT scan evidence, the primary tumour remains in the same size. Ideally a PET scanning should be done in evaluating the disease progression; it is under consideration due to unaffordability.

What is the reason for the constant disease state in a biopsy proven pancreatic adenocarcinoma with distant metastasis? Is there a place for Carica papaya halting the disease progression?

Human pancreatic carcinoma overexpresses many growth factors and their receptors such as epidermal growth factors, vascular endothelial growth factors as well as many cytokines such as transforming growth factor β, tumour necrosis factor α, interleukin 1, 6 and 8 [2, 7, 9]. Studies have revealed that Carica papaya down regulates the expression of pro inflammatory cytokines such as interleukin 2 and 4 as well as anti- tumour cytokines such as interleukin 12p40, interleukin 12p70, tumour necrosis factor α and interferon [9]. It exerts its anti-proliferative and cytotoxic effects by inducing apoptosis [11].

Conclusion

This is a case which has proven the benefit of Carica papaya leave extract on a patient with pancreatic adenocarcinoma with dissemination.

It is recommended to evaluate further the place of Carica papaya in treating patients with malignancies. Double blind placebo control randomized controlled trials could be suggested to evaluate the cytotoxic effects of Carica papaya.

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