Correspondence

Ayurvedic Pharmacoepidemiology: A Proposed New Discipline .................................................... 528

Concomitant Infection with Pulmonary Tuberculosis and Lepromatous Leprosy ....................... 528

Lucio’s Phenomenon ......................................................................................................................... 529

Wilson’s Disease in Late Adulthood presenting with Ascites ......................................................... 529

Sulfonylurea-Induced Prolonged Hypoglycemic Coma: Experience with Dexamethasone ........ 530

Acute Thrombocytopenic Purpura Due to Meloxicam .................................................................. 531

Low Dose Theophylline in Asthma: Let Us Give A Chance ............................................................. 532

Pneumocystis Carini Pneumonia: Role of High Resolution Computed Tomography ............... 533
Ayurvedic Pharmacoepidemiology: A Proposed New Discipline

Pharmacoeconomics is a new field developed by synergy of the fields of clinical pharmacology and epidemiology. In India, both these fields have not flowered as robust disciplines in the medical colleges, industry and government. As a consequence the endeavour in Pharmacoeconomics has been infrequent and at a few centers. But with the growing global demand of Ayurveda and its widespread use in India (70% of people), an emergent discipline of Ayurvedic Pharmacoepidemiology is likely to strike deep roots, with interesting avenues for research, education and services.

The Council of Scientific and Industrial Research (C.S.I.R.) has embarked on a major project - the New Millenium Initiative for Technological Leadership of India (NMITLI). NMITLI has diverse components. But a major component is to develop globally acceptable herbal drugs from the Ayurvedic Therapeutic heritage. Three projects have been already initiated in - diabetes mellitus, osteoarthritis and hepatitis. As a part of this project, it occurred to one of the authors (RAV) to survey the Ayurvedic drug utilization for these diseases. Hence the new discipline was proposed - Ayurvedic Pharmacoepidemiology, after discussion amongst the group.

Ayurvedic Pharmacoepidemiology will be the study of the usage, acceptability, efficacy, safety, complementarity, and cost-effectiveness of Ayurvedic drugs in a large number of people. It will also encompass fields such as Ayurvedic prescription audits, Ayurvedic drugs outlets/utilization, population pharmacodynamics/kinetics and documentation of untoward or unexpected beneficial effects of Ayurvedic drugs. The data collection, wetting, storage and analysis will utilize state-of-the-art software and automation.

The spin-offs and potential contributions of Ayurvedic Pharmacoepidemiology are expected to be sizeable: (1) Usage safety records for Ayurvedic drugs, (2) Data useful for herbal drugs registration, (3) Adverse drug reactions registry for rational therapeutic precautions, (4) Drug dosage adjustments in special age or disease groups of patients, (5) Pharmacoepidemiology of Ayurveda vis-à-vis marketing and rational drug policy, (6) Discovery of novel beneficial effects as leads for further research, (7) ‘Quality of life’ (QOL) studies with Rasayana Dravyas, (8) Patterns of drug usage across the systems of medicine, (9) Drug interactions likely due to concomitant administration of intersystem drugs and (10) Fulfillment of ethical and cultural obligations for the heritage of healing.

We request the readership of the journal to share with our group any experience or ideas they have on the proposed Ayurvedic Pharmacoepidemiology.

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simultaneously. Kumar et al² studied 117 patients of leprosy for evidence of concomitant tuberculosis. Nine patients (7.7%) showed evidence of active tuberculosis, bacteriologically and radiologically.

Lietman et al³ suggest that the infrequent occurrence of both tuberculosis and leprosy is based on the transmission dynamics of both infections. The higher reproductive rate of tubercle bacilli as compared to the lower reproductive rate of lepra bacilli, and the degree of cross-immunity within an individual do not allow both infections to occur simultaneously. This hypothesis was first proposed by Chaussinand who first reported the disappearance of leprosy following exposure to, and treatment of tuberculosis. Whether these transmission dynamics are applicable when leprosy precedes tuberculosis is not clear.

However, Kumar et al² state that tuberculosis can occur throughout the leprosy spectrum. It is important to recognise that these two mycobacterial infections may co-exist in an individual, so that proper therapeutic measures are taken to avoid monotherapy of tuberculosis. Hence it is important to screen all patients of leprosy for presence of co-existent tuberculosis, to prevent resurgence of multidrug resistant tuberculosis.

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Received : 20.8.2002; Revised : 28.1.2003; Accepted : 4.4.2003

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Lucio’s Phenomenon

Lucio’s phenomenon is an acute necrotizing type 2 reaction in untreated case of lepromatous leprosy. This kind of reaction is common in patients from Latin America and Mexico however uncommon in India.

In December 2001, a 54 year old male patient was brought to emergency ward to District Hospital in a precarious condition. He was suffering from fever with rigor for the last one month. During febrile period he developed multiple ulcerations over abdomen and the lower limbs. Before admission he visited various clinics where a battery of tests like FBC, blood smear for malaria parasite, urinalysis, blood sugar, blood urea, ANF, ELISA for tuberculosis and HIV infection, VDRL, HbsAg, antibody for hepatitis C virus and CXR etc were done. These tests were either normal or negative. His fever was diagnosed as pyrexia of unknown origin (PUO). He received empirical treatment with several antibiotics and antimalarial drugs but his condition deteriorated further. At the time of admission clinical examination revealed multiple superficial non-healing ulcers with hyposthetic margin over left flank of abdomen and both thighs. These ulcers coalesced to form a large raw area over anterior aspect of left thigh. In addition to these ulcers he had large thick nose and ears giving appearance of somewhat Leonine face. Skin snip around ulcer margins under oil immersion showed macrophages loaded with lepra bacilli and necrotic tissues. Culture of the swab taken from some of the oozing ulcers showed \textit{Streptococcus pyogenes} sensitive to cloxacillin. He was treated with the WHO’s multidrug therapy for multibacillary leprosy along with ofloxacin-a quinolone derivative. In order to treat secondary infection cloxacillin was added to the antileprosy drugs. Ulcers healed slowly and fever subsided in a month time. Cloxacillin was stopped after healing of the ulcers while antileprosy drugs were continued as per WHO guideline.

These ulcers are the result of necrotizing type 2 reaction in lepromatous leprosy. This kind of phenomenon known as Lucio’s phenomenon happens as a result of obstruction of arterioles from immune complex deposition in lepromatous leprosy.¹ Fever resulted from secondary infection to the ulcers present over the body. Diffuse patchy thickening of skin instead of ulcerations has been reported in a 42-year old male patient suffering from Lucio’s phenomenon in Mexico.² Reports regarding Lucio’s phenomenon are scarce in Indian patients however a case has been reported recently.³ Rarity of the condition in Indian patients leads diagnostic wilderness as happened in this reported case.

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Received : 10.6.2002; Revised : 19.10.2002; Accepted : 3.4.2003

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Wilson’s Disease in Late Adulthood presenting with Ascites

Sir,

Wilson’s disease usually presents first between 5 and 40 years. Presentation after the age of 40 years is uncommon. We report a 58 year old man with newly diagnosed Wilson’s disease who was previously asymptomatic.

A 58 year old man presented with abdominal distension of 8 months duration. There was no history of jaundice, dyspnoea, oliguria, alteration of bowel habits, hematemesis or malena. On physical examination there were no pallor,
icterus, spider naevi, clubbing, caput medusae or lymphadenopathy. KF rings were present. There was bilateral pitting pedal edema. Pulse rate was 76/minute and blood pressure 120/70 mm Hg. Jugular venous pressure was not raised. There was hepatosplenomegaly. Free fluid was present. Rest of the examination was normal.

Investigations revealed a hemoglobin of 12 gm%, total count 8000 differential P68 L30 E 2, normal urine examination with nil 24 hour urine protein, blood urea 30 mg%, serum creatinine 0.8 mg%, serum bilirubin 1.8 mg%, SGOT/SGPT 38/64 IU/L, serum alkaline phosphatase 112 IU/L, serum total protein 6.0 gm%, serum albumin 3.0 gm%, serum sodium 134 meq/L and serum potassium 4.2 meq/L. Electrocardiogram, chest x-ray and echocardiogram were normal. Sonogram abdomen showed hepatosplenomegaly with dilated portal vein and free fluid. Ascitic fluid was straw coloured with sugar of 30 gm%, albumin of 1.8 gm% and a SAAG of 1.2. There were 100 cells/mm³ with 80% lymphocytes. Endoscopy showed grade 3 esophageal varices. Slit lamp confirmed the presence of KF ring. Serum ceruloplasmin was low - 10 mg% (normal 20-45 mg%). The combination of a low serum ceruloplasmin and positive KF rings established the diagnosis of Wilson’s disease. The patient was initiated on penicillamine therapy and followed up.

Wilson’s disease is an inborn error of metabolism characterised by copper accumulation in liver, brain and other organs. Mutation of ATP7B genes coding for copper transporting ATPase is the genetic abnormality. In Wilson’s disease ceruloplasmin synthesised in the presence of ATP7B mutant is catabolised in the liver leading to hepatic retention of copper ions.

Clinical manifestations are rare before 6 years and occur most frequently between 5-40 years. Unrecognized Wilson’s disease is thought to occur rarely in patients over the age of 40. In about half the patients acute hepatitis, chronic hepatitis, cirrhosis or fulminant hepatitis may be the presenting features. In others neurologic or psychiatric disturbances predominate and are always accompanied by Kayser Fleischer rings. KF rings do not interfere with vision and their size correlates well with Wilson’s disease severity.

Treatment consists of copper chelating agents and must be started at the time of diagnosis. Penicillamine is the drug of choice. Monitoring of blood counts and urine analysis should be performed during treatment. Other treatments available are trientine, zinc salts and dimercaprol. Treatment is for life.

Although Wilson’s disease most commonly presents between 5-40 years the diagnosis should still be considered in individuals above that age group who present with unexplained liver or neurological disease.

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Received : 7.8.2002; Revised : 12.2.2003; Accepted : 29.3.2003

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Sulfonylurea-Induced Prolonged Hypoglycemic Coma: Experience with Dexamethasone

Sir,

Hypoglycemia is an important complication of intensive glucose lowering therapy. We report our experience with the use of dexamethasone in subjects with prolonged hypoglycemic coma following sulfonylurea therapy.

Fig. 1: Kayser Fleisher rings present in the cornea of the patient
We studied twenty-nine consecutive subjects who were referred to our tertiary hospital with prolonged (> 4 hours) hypoglycemic coma due to sulfonylurea therapy. Coma was graded by Glasgow Coma Scale (GCS). Dexamethasone was given intravenously (4 mg eighth hourly for first two days, then tapered and stopped on the fourth day) if there was no GCS improvement to glucose normalization within hours. CT scanning was done to rule out stroke in all cases. The dexamethasone-treated group was compared to the rest with respect to the following clinical outcomes at discharge: death, normalization of GCS (score of 15) or irreversible encephalopathy (subnormal GCS).

Baseline characteristics are summarized in Table 1 and values are in mean ± standard error of mean (SEM). Glibenclamide was the sulfonylurea used by 19 of 29 (66%) patients. Other drugs were glipizide (10%) and glimepiride (7%). After admission, hypoglycemia did not recur. Dexamethasone therapy (DT) was given in 16 out of the 29 subjects. Baseline GCS score was 5.7 ± 0.4 in the DT group as compared to 6.1 ± 0.4 in the non-dexamethasone treated (NDT) group (p = NS). Other baseline features of the DT (n = 16) and NDT (n = 13) groups were similar (Table 1), except for higher prevalence of retinopathy in the NDT group. During hospital stay, near-normal glycemia was maintained; glucose values during hospitalization were 168 ± 9 mg/dl in the DT group as compared to 170 ± 10 mg/dl in the NDT group. Five (17%) subjects died: three due to myocardial infarction and two due to aspiration pneumonia. Mortality was similar in the two groups (3/16 in DT vs. 2/13 in NDT; p = NS). Overall, 69% of subjects receiving dexamethasone (11/16) normalized their GCS score as compared to only 23% (3/13) in the non-DT group (p <0.05). When the deaths were excluded, among survivors, 11 out of the 13 survivors (85%) of the DT group achieved a normal GCS (Glasgow Coma Scale) score at discharge, significantly higher than three out of 11 (27%) survivors in the non-DT group (p <0.05). Duration of hospitalization was higher in the NDT group (15 ± 0.8 days in NDT vs. 13.9 (0.8 days in DT), but this did not reach statistical significance.

### Table 1 : Baseline characteristics of study subjects

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Dexamethasone treated (n=16)</th>
<th>Non-dexamethasone-treated (n=13)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>58.1 ± 3.2</td>
<td>55.7 ± 5.2</td>
</tr>
<tr>
<td>Duration of DM (yr)</td>
<td>5.8 ± 1.7</td>
<td>5.9 ± 1.2</td>
</tr>
<tr>
<td>Admission Blood glucose (mg/dl)</td>
<td>37.2 ± 1.8</td>
<td>34.9 ± 2.3</td>
</tr>
<tr>
<td>Neuropathy (%)</td>
<td>15.4</td>
<td>12.5</td>
</tr>
<tr>
<td>Clinical nephropathy (%)</td>
<td>15.4</td>
<td>18.8</td>
</tr>
<tr>
<td>Retinopathy (%)</td>
<td>30.8</td>
<td>12.5</td>
</tr>
<tr>
<td>Macroangiopathy**</td>
<td>23.1</td>
<td>31.3</td>
</tr>
</tbody>
</table>

*Plus minus values are mean ± SEM. **Except stroke, which was excluded in all cases by CT scan

Hypoglycemia is the commonest treatment related complication of diabetes. Glibenclamide was the commonest sulfonylurea used by our study patients. Newer sulfonylureas should be preferred over long-acting molecules, as the latter can induce dangerous hypoglycemia. Use of steroids in sulfonylurea-induced coma is controversial. Some recommend steroids only in proven cases of cerebral edema. Our study shows the favorable effect of dexamethasone in improving neurological outcome, as evidenced by the significant benefits on GCS normalization in our patients. This suggests that the early institution of dexamethasone therapy in severe cases could limit the neurological damage due to the sulfonylurea-induced coma. Benefits of dexamethasone may have accrued from an amelioration of cerebral edema. Though further trials are required for confirmation, the early use of dexamethasone was beneficial and should be considered for all patients who do not recover from drug-induced hypoglycemic coma despite glucose normalization.

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### Acute Thrombocytopenic Purpura Due to Meloxicam

Sir,

Current reports suggest that selective Cox-2 inhibitors are effective non-steroidal anti-inflammatory agents (NSAIDs). Meloxicam is a novel Cox-2 inhibitor. It is associated with fewer adverse effects.

A 57 year old female with a history of arthralgia presented with ‘bleeding from the mouth’, otherwise she was asymptomatic. Her history was notable that approximately one day prior to admission, she had taken two tablets of 7.5 mg of Meloxicam for arthralgia prescribed by a private practitioner. She gave the history of drug sensitivity to sulpha group of drugs and was suffering from bronchial asthma and hypertension, for which she was taking no medicines at the time of presentation. She had undergone cholecystectomy in 1974 and hysterectomy in 1976 with an uneventful postoperative period. She gave no prior history of taking meloxicam. Physical examination revealed numerous purpuric
spots and petichae over leg, face, trunk and neck. There was active bleeding from the lips, soft palate, and anterior pillar of right tonsil. Hemorrhage in the right palpebral conjunctiva was also seen. Patient had no hepatosplenomegaly, lymphadenopathy, and remainder of physical examination was normal.

Laboratory test results on admission disclosed the following data:

- Hemoglobin - 10.8 mg/dl, total leukocyte count - 11.7 x 10⁹/l (normal differential count), platelet count 7.0 x 10⁹/l and a normal peripheral smear. LE cell and ANA was negative. Bone marrow examination revealed peripheral destruction of platelets.

- All other laboratory tests were normal including blood culture, clotting time and coagulation tests were normal.

The drug was discontinued and the patient was started on prednisolone in doses of 1 mg/kg of body weight but following morning she developed gastrointestinal hemorrhage. She was given one unit of blood transfusion. However, she continue to have gastrointestinal hemorrhage, her haemoglobin fell to 8.1 gm/dl and on day three of steroids her platelet count was only 10.0 x 10⁹/l. The patient was put on intravenous immunoglobulin in the doses of 1 gm/kg of body weight for two days and steroids were continued. On day four her platelet count improved to 16.0 x 10⁹/l and she stopped bleeding from gastrointestinal tract and oral cavity. The endoscopic evaluation of gastrointestinal tract was normal. On day six, the platelet count showed improvement and it was 20 x 10⁹/l. Patient was receiving decreasing doses of prednisolone, which was tapered over a period of six weeks. After one year follow up she is on no medication and asymptomatic with a normal platelet count. Platelet studies were not done nor the patient challenged a second time with the drug.

Thrombocytopenia is a well-documented adverse reaction to variety of commonly used NSAIDs such as Diclofenac, Piroxicam, Nimuleside and other NSAIDs. No dose dependent or age preference factor is noted. From September 1996 when meloxicam was first marketed in U.K. till mid June 1998, the U.K. committee on safety of medicines had received a total 773 reports of 1339 suspected adverse reaction with meloxicam.1 773 reports of 1339 suspected adverse reaction with meloxicam.1 773 reports of 1339 suspected adverse reaction with meloxicam.1 The most frequent adverse effects related to meloxicam are gastrointestinal, cutaneous, cardiovascular and neurological. Of all the reactions 41% are gastrointestinal such as perforation, ulceration and/or bleeding. Pruritis, rash, and urticaria were the most common cutaneous manifestation. Other frequently reported reactions were neurological mostly headache and dizziness. Thrombocytopenia was not reported at that time. Thrombocytopenia has been reported in a recent study conducted on the incidence and risk factor associated with meloxicam use amongst 19,087 patients in general practice in England.2 They have reported thrombocytopenia only in two patients but no case of thrombocytopenic purpura was reported. Most of the drugs are said to cause thrombocytopenia by either suppressing platelet production or causing immunological destruction of platelets. In the most cases it is usually immune complex mediated. Current laboratory tests can identify the causative agent in 10% of patients with clinical evidence of drug induced thrombocytopenia.3 The best proof of a drug induced etiology is the clinical course and the prompt rise in the platelet count as it occurred in our patient. Although most patients recover on stopping offending drug, occasional patients with platelet count below 10 x 10⁹/L to 20 x 10⁹/L may require temporary support with corticosteroids while waiting for the platelet count to rise.3 High dose intravenous gamma globulin (1 gm/kg given over 6-8 hrs for two consecutive days) is generally effective treatment for drug induced immune thrombocytopenia. Severe thrombocytopenia usually develops within 12 hours in a previously sensitized individual. In our patient it developed after 24 hours of drug ingestion but patient gave no history of prior sensitization.

Though the safety profile of meloxicam have been produced by various clinical trials, further rigorous trials and long term experience is needed to assess the various adverse reactions, especially hematological.

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**Low Dose Theophylline in Asthma: Let Us Give A Chance**

Corticosteroids form the cornerstone of treatment in bronchial asthma. Topical therapy is having very few systemic side effects. The prevention of local inflammation by topical therapy however, cannot check the systemic inflammatory process of ‘T’ cells and mediators. That is why inflammatory process resets as and when topical corticosteroids are taken off. A potent, safe, systemic anti-inflammatory drug devoid of side effects is being researched to be used in chronic bronchial asthma.1

A widely prescribed drug, theophylline that has been used in bronchial asthma the world over since more than five decades, has been pushed to a position of an adjunct therapy, keeping in view of weak bronchodilation, more of side effects and drug interaction and a narrow range of safety profile. Yet a new interest has rears to use this systemic

532 JAPI • VOL 51 • MAY 2003
immunomodulators as a primary drug, utilizing it’s potential benefit at the low dose. This is currently being considered important in view of shifting bronchodilators-oriented approach to an anti-inflammatory management approach.

Anti inflammatory effect of theophylline is through inhibition of prostaglandins, suppression of response to mast cell mediators and decreased both microvascular leakage of plasma into the airways. Low dose theophylline reduces the number of activated CD4 and CD5 lymphocytic subsets as well as allergen induced eosinophilia of the airway as revealed by BAL analysis, exhibiting lymphocytosis following allergen challenge. Lower plasma theophylline levels show improvement in asthma by modulating T-lymphocyte function. It also reduces both IL-4 and IL-5 expression on cells as well as their respective circulating levels. Though the exact mechanism is not clear, the current evidence shows the antiinflammatory action of theophylline could be due to inhibition of phosphodiesterase and recently type III and IV isoenzymes have been characterized in a number of inflammatory cells. Even late asthmatic reaction following allergen challenge can be inhibited by low dose theophylline. Hence a low dose theophylline with plasma concentration of 5-10 mg/l can be used as an alternative to other antiinflammatory drugs in mild to moderate asthma with a low risk of side effect. Compare to topical steroids, it is a less potent antiinflammatory drug but the only potential immunomodulators with highly benefiting systemic effect that needs to used in the clinical management of asthma, in a low dose, having enhanced safety profile. Increasing the dose to achieve bronchodilators effect to plasma levels of 10-20 mg/l narrows the safety profile that had put this potentially useful drug into the back seat. The arousal of newer interest at low dosage shall bring forth this useful drug as a front runner in treating mild to moderate chronic bronchial asthma. Dysphonia and oral candidiasis are not uncommon even with lower doses of inhaled corticosteroids, side effects such as throat irritation and coughing are also documented. Similarly, over treatment with inhaled steroids can produce systemic effects such as effect on bone metabolism and adrenal gland in adults and changes in growth velocity in children. Rarely, posterior sub capsular cataract, occasional behavioural disturbance, disseminated infection with varicella and altered lipid and carbohydrate metabolisms have also been reported.

In such a situation, resurface of case of low dose of theophylline in asthma may be prudent. In acute bronchial asthma, when potent anti inflammatory drug - corticosteroids and potent bronchodilators - salbutamol are being given, parenteral theophylline is not going to provide any additional benefit to both antiinflammatory and bronchodilation. So it’s use is unlikely to provide any relief, adding toxicity rather than benefiting the patient.

Possible mechanism of action of theophylline includes phosphodiesterase inhibition, adenosine receptor antagonism, elevation of circulating adrenaline and mediating antagonism & inhibition of calcium reflux. In addition, theophylline perhaps inhibits of F-Kappa β (-a transcriptor in human purified mast cells - responsible for transcription of TMNF alpha, GM-CSF and IL-8 with in cells) which is responsible for its potential antiinflammatory activity, in addition to bronchodilation.

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Received : 5.3.2003; Accepted: 17.4.2003

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Pneumocystis Carini Pneumonia : Role of High Resolution Computed Tomography

Sir,

I read the above article1 with interest and although the changes on HRCT scanning are usually classical in most cases of Pneumocystis, HRCT scans can be non-specific or normal in some cases. I would like to mention our unique experience of a patient with Pneumocystis carinii pneumonia. This was a 64 year old man who had undergone a single lung transplantation 10 years back for chronic severe obstructive airways disease. After the transplant he continued to work as a hospital porter till 6 months before presenting with progressive dyspnoea on exertion associated with a productive cough. His sputum remained sterile, mucoid and frothy. His lung function parameters remained normal, HRCT scan and DTPA aerosol scan excluded bronchiolitis obliterans and bronchoscopy did not show airway obstruction and lave and biopsy were normal.

He was treated with cyclosporine, azathioprine and prednisolone along with broad spectrum antibiotics with no improvement. Although his biopsy, and DTPA scan did not show convincing evidence of chronic graft rejection we upgrad his immunosupression to tacrolimus and mycophenolate, replacing the cyclosporine and azathioprine. Four weeks later he became progressively more breathless now even at rest and his lung function showed a restrictive defect. His CXR showed a reduction in the size of his transplanted lung and HRCT scan failed to demonstrate any abnormalities. A further bronchoscopic lavage was undertaken which confirmed Pneumocystis infestation. He became extremely breathless with a type I respiratory failure and required ICU admission for mechanical ventilation. We discovered that he was transplanted at a time when septrn prophylaxis for PCP was only continued during the early (3-6 months) after the organ transplantation and he was subsequently without PCP prophylaxis for over 9 years. all
recent transplant patients are kept on PCP prophylaxis with a
daily dose of septrin keeping PCP infestation extremely
negligible in our unit.

Unfortunately this patient succumbed to his illness inspite
of high dosage treatment with IV septrin and a postmortem
examination showed PCP in his transplanted lung with
interstitial thickening. Hence although he was investigated
extensively for chronic rejection given his worsening lung
function, his HRCT scan remained normal and his initial
lavage was unhelpful. Profound dyspnoea and hypoxia in an
immunocompromised host specially when not an PCP
prophylaxis should raise the suspicion of PCP and even
repeated lavages need to be undertaken with empirical therapy
to confirm the diagnosis.

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**Book Review**

**Primer on Insulin Resistance**

*by Dr Shashank R Joshi*

*Published by : Asian Health Care*

The present day hot topic in Clinical Medicine - "Insulin Resistance", has been reviewed in a lucid
manner by the author in less than 100 pages. This shows the simplest way of presenting a complex
metabolic problem, which is rapidly advancing. The book gives a clear definition of the problem, gives a
detailed account of the molecular components and the way they interact to produce the clinical syndromes.
Author also gives clear guidelines for making the diagnosis and details of pharmacological and
nonpharmacological treatment modalities. Being an Endocrinologist, he has obviously given a little more
importance in managing the diabetes part of the Insulin Resistance. Even though the author has given
references in the form of suggested readings, one may find, difficulty in locating the reference for the
Indian criteria for the metabolic syndrome. The printing, lay out and the design of the book are excellent,
which will make this compact book a "collectors item" for the doctors.

**Prof. R. V. Jaykumar**
Prof. of Endocrinology,
Amrita Institute of Medical Sciences, Kochi.

**Indian Price: Rs. 250/-**

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