Pulmonary Strongyloidiasis: Time to Wake Up

Sir,

I read with interest the report on a case of “Pulmonary Strongyloidiasis” by Jayaprakash et al.1 Pulmonary strongyloidiasis is a difficult disease to diagnose and unless suspected early in the course of the disease the patient usually succumbs to the disease;2,3 inspite of treatment. Fortunately in this case the patient received right management in time and recovered.

Usually symptoms of pulmonary strongyloidiasis include dyspnoea, wheezing, cough and haemoptysis.2 These symptoms can be easily confused with bronchial asthma with infection or some other serious disorder like tuberculosis. The patient described in the paper had two months history of irregular fever and received several times corticosteroids and bronchodilator etc. Chest X-ray showing an opacity in the left upper zone with two months history of irregular fever and haemoptysis could have easily been confused with the diagnosis of tuberculosis, probably this was prevented by accidental discovery of the parasite in the sputum. Eosinophilia is not an uncommon manifestation of strongyloidiasis but was absent in this patient most likely because of intermittent corticosteroid therapy. Eosinophilia was also absent in another reported patient who was immunosuppressed because of his disease.3 As the patient with pulmonary strongyloidiasis is often very sick and presents with ARDS, tracheal aspirate, in addition to sputum has also been used to detect the parasite.2,3 Generally pulmonary or systemic strongyloidiasis has been described in association with immunosuppressed patients4 and specially with HTLV-1 infection. However, several reports including ours1,2 showed even a few days of steroid therapy in immunocompetent patient can also bring out this disastrous complications in patients who are infected with the parasite. Once a patient is infected with the parasite the infection could be lifelong without treatment and may be largely asymptomatic.

India is not recognized as a country where strongyloidiasis may not be uncommon and use of steroid for bronchial wheezing is rampant, this will predispose a few patients to life threatening pulmonary strongyloidiasis leading to ARDS. In the case report under discussion unfortunately only one Indian reference was quoted while a search in Pubmed immediately brings out 43 articles on strongyloidiasis from India of which 12 articles were quoted and specially with HTLV-1 infection. However, several reports including ours1,2 showed even a few days of steroid therapy in immunocompetent patient can also bring out this disastrous complications in patients who are infected with the parasite. Once a patient is infected with the parasite the infection could be lifelong without treatment and may be largely asymptomatic.

More over even if corticosteroid have been given to the patient, should any deterioration occurs following steroid therapy prompt search for the parasite should be initiated. Moreover we need epidemiological studies in this country to understand prevalence of this condition.

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Inducible Ischemia with Patent Stents

Sir,

Coronary artery stents are metallic devices placed in areas of stenosis and occlusion to facilitate coronary flow to prevent myocardial damage and restenosis. Stents revolutionized the treatment of acute coronary syndrome by saving “myocardium at risk” and then maintaining the coronary patency. But, it is also a fact that stents including drug eluting ones can have both symptomatic as well as angiographic restenosis in spite of adequate antiplatelet medication. We must also give a thought to whether metallic stents can match the special capability of normal coronaries to dilate in times of demand to increase the coronary flow four to five times that of the baseline flow. Cardiomyocyte is very efficient in extracting the oxygen and other nutrients from the blood flowing through coronaries. It extracts 70 to 80 % of the nutrients from the blood flowing through coronaries.1 Any additional demand has to be met by extra flow through coronaries which is achieved by coronary dilatation. Ability of the coronary circulation to increase flow up to five times the basal resting level to meet demands during exercise is called “coronary reserve”. Coronary reserve is compromised in stenosed vessels causing inadequate flow and consequent exercise induced ischemia.

A patent stent will permit adequate coronary flow at rest as well as at all normal activities. But metallic stents may not have the capability to dilate (like normal coronary artery) to
meet demands at high levels of exercise. The threshold stress level up to which stent related artery will provide adequate flow is not clearly known. I found inducible ischemia in stent related area in stress myocardial perfusion study in two patients who had undergone PTCA with stenting six months ago. Both patients exercised up to a workload of 13.5 METS and had no symptoms. Check angiography showed patent stent with TIMI 3 flow. Probably, any exercise up to 10 METS may not create significant problem but at higher levels of exercise, the stent related area may not get blood supply equal to that in a normal coronary artery due to inadequate coronary reserve in coronary artery with stent. Multiple stents in the same coronary artery may cause more problem. Relatively rigid metallic stents placed in coronary artery which has dynamic and need based diameter may lead to a local restrictive lesion at peak flow requirements.

So, stents should be put with discretion. A stent should be placed to tackle a local crisis in coronary endothelium as part of a diffuse or systemic endothelial disease to save “myocardium at risk”. The unstable endothelium will recover in a matter of time. Stent is needed only during the period of endothelial instability. The emphasis after that should be on management of the endothelial disease causing atheroma. Presence of a metallic stent makes the person a life long patient who has to take antiplatelet drugs to prevent stent occlusion. This may be justifiable in acute coronary syndromes, but many a times stents are placed based on angiography findings in persons with atypical chest discomfort with doubtful ECG and TMT abnormality. Majority of times, the physiological significance of the angiographic findings are not confirmed by a study like stress myocardial perfusion study. If myocardial perfusion study shows inducible ischemia, then it has to be tackled to save the myocardium at risk. If there is no perfusion defect at adequate level of exercise in a well conducted myocardial perfusion study in the myocardium related to the angiographic stenotic area that means that already natural bypass has happened and no intervention is needed. Placing a metallic stent in such cases makes a healthy person cardiac patient for life forcing him to take antiplatelet drugs to prevent stent occlusion. Research is already on to make dissolving stents. After doing its job at the time of need, the stent should disappear. Anything which persists after its actual requirement is likely to become a liability. Till the time dissolving stents becomes available, stenting should be restricted to actual myocardial salvage situations.

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References