Abstract
A 61 year old male, with a bilateral persistent and recurrent pleural effusion, had undergone frequent tapping over a period of eight months, prior to the referral. The patient was treated earlier to the referral empirically for pulmonary tuberculosis with no response to the treatment. Malignancy was suspected and ruled out. A detailed examination showed that he also had atrophic nails with yellow discoloration and lymphedema of feet. Yellow nail syndrome was diagnosed on the basis of the clinical findings of the triad viz. yellow atrophic nails, lymphedema and bilateral pleural effusions. Pathogenesis still remains elusive for the syndrome. Pleurodesis provided both symptomatic relief and a respite from repeated tapping. The case is presented as a rare condition, which was missed for diagnosis, for quite some time by several specialists.

Introduction
Yellow nail syndrome (YNS) is a rare condition that is characterized by the clinical triad of yellow nails, lymphedema, and respiratory tract involvement, mainly pleural effusion. Two of these three features have to be present for the diagnosis, as the complete triad is seen in only one-third of the
patients. Onset of YNS is usually after puberty, but can occur at a later age also. It often goes undiagnosed because of its rarity and lack of physician’s familiarity with the condition. It may not also get recognized early because all the features of the triad may not develop simultaneously but evolve over a period of time.

Respiratory manifestations are diverse; pleural effusion that is often bilateral like in the present case, bronchiectasis, sinusitis, and chronic cough. Though there are reports of familial cases of YNS, most often the time it occurs sporadically. Phenotypic classification of dominantly inherited lymphedema syndromes comprise of several disorders including YNS. Truncating mutations in FOXC2 are reported in 11 families with lymphedema syndromes of which only one had yellow nails.

We report a case of YNS whose diagnosis was missed. He had undergone repeated pleural tapping and had received treatment for tuberculosis because of pleural effusion prior to the referral to the present authors.

**Case Report**

A male aged 61 years, was referred in July 2009 to one of the authors (ADBV) for a second opinion for the bilateral pleural effusions and persistent chronic dry cough. The patient complained of breathlessness, persistent dry cough, edema of feet, and marked weakness. There was a history of dry cough for the past one year.

Patient owned a unit of watch manufacturing and repairing/electroplating. He was a heavy smoker of bidis from a young age. He had developed dry cough without any expectoration, since August 2008. His plain x-ray of chest (PA view) was normal. His complete haemogram was normal without any leucocytosis, ESR was mildly elevated. He had received several courses of broad spectrum antibiotics without much relief from his cough. He was also given corticosteroids for two months. Meanwhile he had developed bilateral pleural effusions confirmed on radiography. After tapping, the fluid was sent for cytology. It was negative for malignancy and had shown the presence of plenty of lymphocytes. Mesothelioma was ruled out by a pleural biopsy interpreted by a leading onco-pathologist. The CAT-SCAN of the chest had also ruled out any other lung and cardiac lesions. Colour Doppler echocardiography was normal except for a mild pericardial effusion.

The patient needed repeated tapping (Fig. 1a) as pleural effusion used to collect rapidly after tapping. Pleural effusions used to cause him breathlessness. Patient received standard treatment for tuberculosis for a period of three months. However, there was no benefit what-so-ever observed in the respiratory symptoms or in pleural effusion.

On physical examination, the patient looked breathless, pale and in pain. He had been experiencing pain and tenderness at the site of pleural tapping. His pulse rate was 84/minute, BP was 140/90mm.hg. On reviewing his X-rays (Fig. 1b) of the chest, a lymphatic disorder was suspected due to widening of the thoracic duct. One of us (ADBV) had noticed yellow nails on examination (Figs. 1c, 1d). Several of the nails were atrophic and broken besides being yellow. Both finger nails and toe nails were looking dystrophic. Patient also had bilateral lymphedema of the lower limbs (Fig. 1d). A vivid mental recall of the description and pictures of YNS in a book on “Nails in Health and Disease” had helped in the diagnosis of YNS. The patient and his relatives had noticed yellow discoloration and easy brittleness of his nails. Hypothyroidism was ruled out by laboratory investigations. His blood biochemistry (Table 1) was normal ruling out diabetes mellitus. Renal and liver function tests were normal. Complete hemogram had also remained normal.

Patient was advised pleurodesis for prevention of recurrence.
of pleural effusion by obliterating the pleural space. Sterile
talc powder (30 gms) was used in form of a paste for bilateral
pleurodesis. The procedure was done for one side at a time
with an interval of 15 days. The procedure was without
any complication and was well tolerated. Patient however,
developed severe edema of the entire body within a fortnight
that gradually has now disappeared almost completely. His
respiratory symptoms have improved significantly. There is an
improvement in his appetite as well as in the sense of well-being.
But the peripheral lymphoedema persists.

### Discussion

Yellow Nail Syndrome is a rare syndrome characterized
by the clinical triad of lymphedema, yellow nails, and diverse
respiratory problems. More than 100 cases have been reported
in the literature. Diagnosis has been usually made on the basis
of clinical features. Only a few cases are reported from our
country. However, presence of pleural effusion in YNS leads
often to a suspicion of pulmonary tuberculosis or malignancy.
Our patient had bilateral pleural effusions which do not
commonly occur in tuberculosis. Yellow nails can also be a
paraneoplastic manifestation of malignancy.

Several cases of YNS have been reported to show disappearance
of yellow color of the nails after a successful treatment of the
associated malignancy. Presence of YNS should suggest a need
to rule out any associated malignancy.

The underlying pathophysiological mechanisms of YNS
remain elusive ever since the first recognition of the association
of yellow nails with primary lymphedema in 1964 by Samman.
Attempts have been made to understand diverse associated
clinical manifestations and diseases. Lymphatic block, protein
leakage due to microangiopathy and increased microvascular
filtration at different sites have been proposed as possible mechanisms. Some investigators had shown microangiopathy by employing interesting techniques of finger nail capillarscopy and photoplethysmography. Underdeveloped hypoplastic lymphatics and lymphatic ectasia have been proposed as a common mechanism for the triad of YNS. However, YNS is a slowly developing syndrome and lymphedema and pleural effusion may occur years after the nails become yellow. Treatment in patients of YNS remains and will remain symptomatic until the underlying mechanisms are better understood.

### References

1. Hoque SR, Mansour S, Mortimer, PS: Yellow nail syndrome: not a