The synchronous existence of lung cancer along with parenchymal pulmonary tuberculosis – active and quiescent has been often reported. The coexistence of lung cancer with a malignant and tuberculous pleural effusion on the other hand is rare with on such previously reported instance. We report one such interesting case.

A 61 year old non smoking male came to us with a history of cough, mucoid sputum, loss of appetite and a significant weight loss since 2 months. He had no fever, night sweats, dyspnoea or chest pains. Physical examination revealed decreased thoracic movement, a stony dull note, absent breath sounds and decreased tactile vocal fremitus and resonance on the right side consistent with a pleural effusion. Chest radiograph (PA view) showed a massive right pleural effusion with multiple poorly defined circular shadows in the opposite lung. Aspirated pleural fluid was hemorrhagic, an exude (Protein: 4.7 gm/dL) and having a lymphocyte differential of 54 %. Pleural fluid Gram stain and ZN stain were negative. Cytological examination revealed cell clusters with atypical nuclei. Pleural fluid carcino-embryonic antigen was also elevated (103.7 IU/ml, normal < 3.4IU/ml). Sputum smear examination for AFB was negative for 3 consecutive samples. Sputum examination for malignancy was repeatedly positive for poorly differentiated carcinoma. Bronchoscopy revealed external compression of RUL bronchus and bronchus intermedius and bronchial lavage smears were positive for malignant cell and negative for AFB. USG abdomen showed grade II prostatomegaly but serum PSA was normal. A diagnosis of bronchogenic carcinoma with malignant pleural effusion was kept. The patient refused any further palliative treatment for the same.

At 6 weeks pleural fluid culture for AFB (on LJ media) was found to grow colonies of mycobacteria. Patient was readmitted for further evaluation. Sputum smears were sent for Z.N stain which was positive for AFB. Subsequent sputum culture also grew mycobacteria species.

Our diagnosis was revised to sputum positive pulmonary tuberculosis with bronchogenic carcinoma (Stage IV) with malignant and tuberculous pleural effusion. Patient was started on 4 drug anti-tuberculous treatment (HERZ). After clinical stabilization he was discharged from the hospital.

The coexistence of bronchogenic carcinoma with pulmonary tuberculosis has been explained by the following mechanisms: 1. The chance coincidence without any relation – more relevant in countries like India with a high prevalence of tuberculosis, 2. “Scar carcinomas” i.e. tuberculosis giving rise to epithelial metaplasia with subsequent progression to carcinoma,1 3. Re-activation of tuberculosis due to release of local tumor peptides and antigens from the tumor cells upsetting the milieu of the silent granuloma and allowing the mycobacteria to proliferate4 and, 4. Increased susceptibility to exogenous infection by mycobacteria due to malnutrition and weight loss related to an advanced neoplastic disease.5

Immunosenescence and a decline in cell mediated immunity associated with ageing, poor nutrition, chronic alcoholism, lung neoplasm may all have contributed to

Fig. 3: Life cycle of Echinococcus: Life cycle exit between carnivores and herbivores like dog and sheep; man is accidental intermediate host and end point in parasite’s life cycle. The mature adult worm inhabits the intestine of carnivores specially dogs. Herbivores like sheep and cows become infested by eating grass contaminated with dog’s feces containing eggs. Contaminated vegetables are culprits for human infestation.

Percutaneous drainage of hepatic cysts, known as PAIR (Puncture, Aspiration, Installation of scolicidal agents and Reaspiration) technique has gained acceptance. This procedure is minimally invasive, cost effective, involves reduced hospital stay and has less morbidity and mortality than surgery. It is treatment of choice in patients with hepatic hydatid cysts who either refuses surgery or have significant co-morbid diseases. Cysts relapsing after surgery or failed to regress following chemotherapy are also amenable to PAIR. The procedure is associated with possible complications of liver puncture, bleeding, bile peritonitis, anaphylaxis, allergic reaction and biliary communications.

The purpose of reporting this case is to highlight the uncommon presentation of the disease and the need for improved sanitation in our country.

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Pleural Effusion of a Dual Etiology
Sir,

The synchronous existence of lung cancer along with parenchymal pulmonary tuberculosis – active and quiescent
either endogenous reactivation of TB or predisposed our patient to exogenous reinfection. The extension of the malignant neoplasm from the lung parenchyma to the visceral pleura may have eroded a sub pleural focus of mycobacteria into the pleural space thus setting into motion the chain of events leading to the development of a tuberculous pleural effusion.

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Infectious Causes of Macrophage Activation Syndrome

Sir,

I have read with interest the article “Macrophage Activation Syndrome – Experience from a tertiary referral centre.” by Pinto et al. Among the thirteen cases that the authors had described, the majority of patients had an underlying connective tissue disorder. I would like to add to that list by presenting two cases of infective etiology that can cause Macrophage Activation Syndrome.

A 17 year old boy presented with high grade fever with chills of 2 weeks duration. Ten days into the illness he developed an episode of bleeding gums for which he was brought to the hospital. On examination he was febrile, pulse rate was 120/min, and blood pressure was 110/70 mm of Hg. He appeared toxic, tongue was coated and he had pallor. The rest of the general and systemic examination was unremarkable. Investigations revealed that Hb 6.5g/dl, TC 13,600 /cumm, platelets 6000 /cumm, S. Ferritin 2690, S.LDH 2903 U/L, S. Triglycerides 311 mg%, SGOT 368 U/L, SGPT 166 U/L. Rest of the liver function test, renal functions and electrolyte profile were normal. Bone marrow biopsy showed mild diffuse plasmacytosis and hemophagocytosis. Work up for the cause of fever including connective tissue and vasculitic workup, CT of the thorax and abdomen were all non-contributory. During the course of her hospitalization she had a steady fall in her hemoglobin levels with no evidence of overt or occult blood loss, requiring transfusions. She also developed thrombocytopenia – platelet count fell from 1,27,000 – 72,000 with no evidence of bleeding.

In the setting of prolonged fever with extensive evaluation failing to identify a definite cause, she was started on empirical anti-tuberculous therapy. Fever subsided about 10 days after starting anti-tuberculous drugs. At the same time there was also improvement in the hemoglobin levels and platelet count.

| Table 1 |
|----------|----------|----------|----------|
| Hb (g/dl) | Day 1 6.5 | Day 3 7.0 | Day 7 8.9 | Day 14 10.5 |
| TC (/cu mm) | 13,600 | 9200 | 4400 | 6400 |

Infection has been found to be associated with hemophagocytosis in half of all reported cases. Any of a number of microorganisms can be involved, and the pattern of infection varies according to geographical origin.

About 30 cases of infection associated hemophagocytosis have been related to tuberculosis, diagnosed by culture, or from post-mortem tissue. This may be associated with focal or disseminated tuberculosis.

Thirty cases with documented prominent hemophagocytosis on bone marrow aspiration smears were reviewed. Twenty-one (69%) of the marrows were from patients who had common tropical infections such as malaria, typhoid and visceral leishmaniasis.

The aim of this correspondence is to make aware that common infectious diseases that we see in our country like typhoid and tuberculosis can also cause hemophagocytosis; and that favourable outcomes are seen if prompt therapy is directed at the underlying etiological agent.