

## LETTER TO THE EDITOR

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### The path to subcutaneous emphysema

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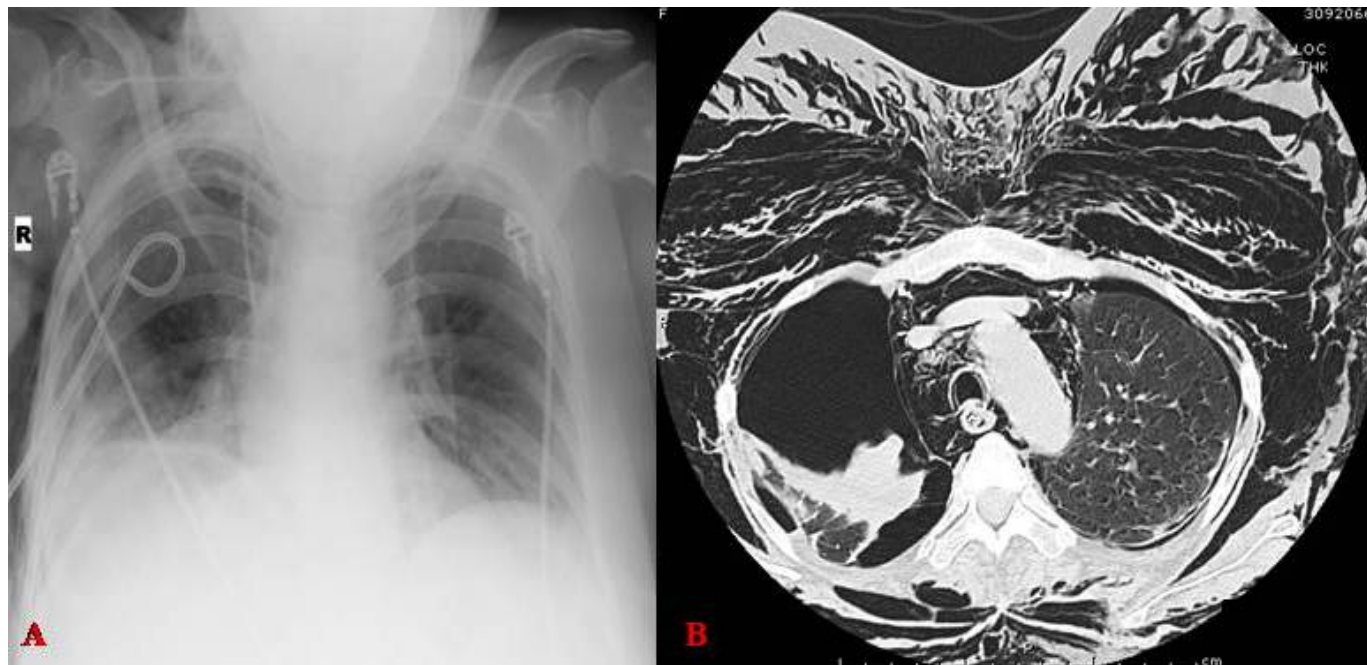
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A middle aged woman, diagnosed case of decompensated cirrhosis of liver (Child Pugh class C, Model for end stage liver disease score 24), secondary to chronic hepatitis C infection presented to our emergency department with severe breathlessness and associated bloating and swelling of face, neck, upper chest and upper arms since one day. Five days prior, elsewhere, she had undergone evaluation for cough, pleuritic chest pain and right sided heaviness over the thorax, following which, she was diagnosed as having localized right sided pleural effusion for which she underwent pig tail intercostals chest drain placement (**Figure 1A**). Subsequently, pleural fluid reports were

suggestive of low protein and high serum to pleural fluid albumin gradient (SPAG) with leukocyte count of 500 cells, which was lymphocytic predominant. During further hospital stay, new onset dull aching chest pain, prompted the treating physician to manipulate the chest drain as serial chest X rays were suggestive of drain migration. Thirty six hours afterwards the patient developed severe progressive breathlessness with rigid chest wall leading to respiratory embarrassment. A computed tomography revealed extensive subcutaneous emphysema (SCE) with pneumo-mediastinum and pneumothorax. (**Figure 1B**)



**Figure 1A:** Chest X ray (AP) view showing loculated pleural effusion with pig tail catheter drain in situ that has migrated to the upper zone; **Figure 1B:** High resolution computed tomography scan of the thorax showing bilateral pneumothorax with pneumomediastinum and extensive diffuse surgical emphysema within soft tissues of thorax, neck and abdomen.

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Subcutaneous large bore needle drain placements dramatically reduced the emphysema, but the patient developed multiple skin and soft tissue infections, nosocomial pneumonia and secondary bacterial pleuritis in hospital eventually leading to death. Pleural effusion (PE)

in cirrhotics commonly occurs on the right side and when > 500mL in the absence of cardiopulmonary, parenchymal disease or malignancy, is known as hepatic hydrothorax (HH), which is seen in 4 to 12% of cirrhotics. [1] It occurs due to passage of peritoneal fluid into the pleural cavity through varied sized diaphragmatic defects in the presence of negative intra-thoracic pressure during inspiration. Right sided effusions are common because of greater chances of development of these defects in the presence of weak diaphragmatic musculature on the right side, in comparison to the left. Other theories include decreased colloid osmotic pressure due to hypoalbuminemia and azygous venous system hypertension leading to plasma leakage into pleural space [2].

Presentations include cough, shortness of breath or pleuritic chest pain. It is very uncommon for HH to present as localized PE. The condition is classically managed with salt restriction, diuretics and pleurocentesis in the presence of overt symptoms. Chest drain placement for HH carries significant morbidity and mortality [3]. Placement of large bore subcutaneous drains and increasing catheter suction has been shown to improve SCE drastically, [4] even though former modality has not been studied in cirrhotics. An approach to pleural effusion in cirrhosis starts with an astute history taking and clinical examination. Historical importance lies in acute development of symptoms related to pleural effusion, associated symptoms like long standing fever, weight loss and chest pain—suggestive of an infectious process and clinical examination revealed significant lymphadenopathy and/or pleural rub. In the event that an infectious process is contemplated, then pleural fluid aspiration and a sensible panel of testing is warranted, which includes total cell count, differential count, protein content, serum to pleural fluid albumin

gradient (SPAG), cultures and adenosine deaminase levels (if strong suspicion of tuberculosis). If spontaneous bacterial pleuritis is diagnosed, antibiotics, albumin infusions and nutritional management are generally followed. There is no role of catheter drainage of infected pleural fluid (low protein, high SPAG) in cirrhotics. In the event that there is mixed picture on pleural fluid analysis (high protein, high SPAG) or an exudative picture (high protein, low SPAG), then therapy for tuberculosis can be initiated after ancillary test confirmation. In such cases also, there is no role of intra-pleural catheter drainage. Patients with rapidly refilling hydrothorax need to be managed with repeated thoracocentesis under albumin cover to prevent circulatory dysfunction or if candidacy is adequate, for TIPS or liver transplantation [5].

In conclusion, cirrhotic patients with pleural effusion should initially undergo pleurocentesis and fluid analysis followed by directed management before invasive measures are practiced.

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