Case report

Chylothorax in a case of systemic lupus erythematosus : a case report

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Abstract

A 19 year female, known case of SLE on immunosuppressant presented with chest pain, shortness of breath and cough for last 5 months. On examination she was found to have bilateral pleural effusion with right side more than left side. The physical and chemical examination of pleural fluid from the right side shows chylothorax. Left sided pleural fluid was exudative and lymphocytic in nature. She was managed with intercostal drainage (ICD) tube drainage followed by pleurodesis. Chylothorax is one of rarest manifestation of SLE and this case report highlights the presentation and management of chylothorax.

Key Words: Chylothorax,systemic lupus erythematosus, Polyserositis, pleurodesis

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INTRODUCTION

Chylothorax is accumulation of milky appearing fluid containing high level of lipid content in pleural space [1]. The cause of chylothorax can be categarized as traumatic and non-traumatic. Traumatic cause is mostly due to surgery or trauma. Malignancy is the most common cause of non-traumatic cause of chylothorax and malignant lymphoma has been considered the most frequent cause followed by metastatic carcinoma [2]. Rarely SLE has been found to be associated with chylothorax. Systemic lupus erythematosus (SLE) is a multisystem chronic inflammatory disease of autoimmune etiology, which affects the skin, joint, kidney, serous membrane, lungs, nervous system and other organ of the body. Polyserositisis is common in SLE patients to result in exudative pleural effusion, but rare case reports of chylothorax have been recorded in the literature[3]. With this background we have reporting a case of SLE with chylothorax.

CASE REPORT

A 19 year old female suffering from Systemic Lupus Erythematosus (SLE) was referred to department of pulmonary medicine, GMCH, Guwahati, Assam as recurrent pleural effusion for further evaluation. She hadexertional dyspnoea and dry cough for 5 months along with intermittent chest pain at presentation. During the preceding five months, she underwent repeated pleural tapping on several occasions and the fluid from the right side appeared consistently milky in appearance on all the occasions. She has received oral predinisolone, azathioprine and other medication for SLE.

On examination, she had stable vital signs with pulse rate as 74/min, BP-116/70 mm of Hg, respiratory rate of 16/min. Her systemic examination was unremarkable except for the respiratory system showing decrease breath sound bilaterally on infrascapular region with diminished percussion note; the feature being more overt on the right side. Laboratory examination revealed mild anaemia (haemoglobin of 10.6 gm%, total leucocyte count of 8,900, and ESR of -6mm/hour. Her routine biochemistry (including blood sugar , creatinine, bilirubin with liver enzymes, serum proteins, uric acids, and lipids as triglyceride, cholesterol and lipoproteins) was essentially normal. Urine routine examination was also normal. Serology for hepatitis B, C, and HIV were negative. Ultrasonography of abdomen and 2D echocardiography showed no abnormalities. Antinuclear antibody was positive with homogenous pattern and titre of 1:40, Anti-

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Sm Antibody was also positive and other antibody like anti- dsDNA, anti-histone, anti-RNP, anti-Ro, anti-La, anti-Scl-70, and antiphospholipid antibodies including lupus anticoagulant C and anticardiolipin were negative. Chest X-ray showed bilateral pleural effusion, right side more than left side. (Fig.1)

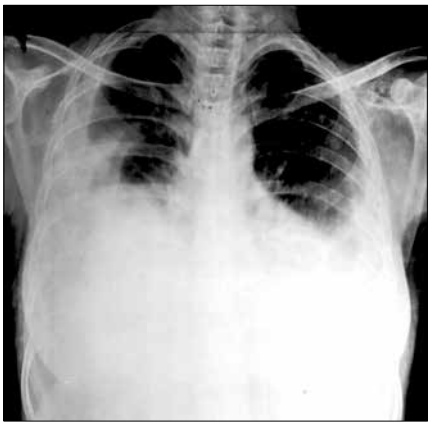

Fig 1. Chest X-ray showing bilateral pleural effusion (Right side more than left side)

Pleural tapping done on right side, appearance of the fluid was milky colour. The pleural fluid analysis shows total protein-4.0 g/dl, total count-200 cells/mm3 of which lymphocytes 80% and polymorph 20%, sugar-96 mg/dl, ADA-19 U/L, LDH-274 U/L, malignant cytology-negative, Gram stain and culture sensitivity of pleural fluid did not show any pathogenic organism and pleural fluid for acid fast bacilli was negative. Pleural fluid triglycerides level of 117 mg/dl, cholesterol level of 50 mg/dl. To differentiate it from pseudochylothorax, this milky fluid was put in test tube and centrifuged. It did not show any sedimentation (Fig.2).

Fig 2. Milky white colour pleural fluid suggesting chylous pleural effusion and on centrifuging the fluid there was no sediment.

The tentative diagnosis of chylothorax was confirmed with the estimation of the ratio of pleural fluid to serum cholesterol level which was less than 1. An ICD was put on right side and around 500 ml chylous fluid a day for 7 days following which the drainagereduced. The repeat chest X-ray done showed fully expanded lungs. Pleurodesis with 10% povodine iodine was done and ICD tube was removed after 24 hours. (Fig.3).

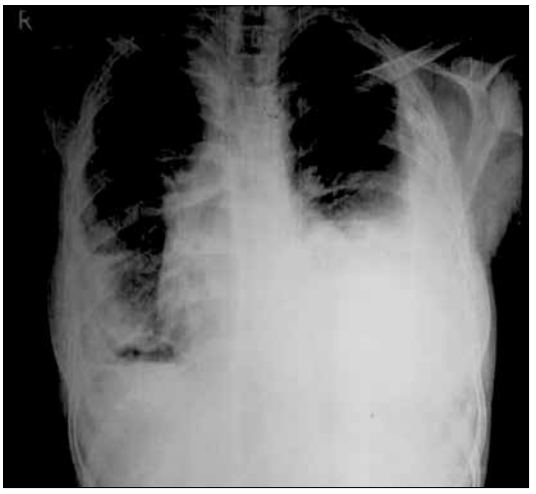

Fig 3. Chest X ray following pleurodesis showing right side fully expanded lung and left side pleural effusion.

Left sided tapping and analysis was done, which show exudative picture of pleural effusion for which patient was put on observation.

DISCUSSION

Chylothorax is the accumulation of chyle in pleural cavity1. Chyle appears visually as a milky fluid due to high triglycerides concentration. Chyle is bacteriostatic and pathogenic organisms were unable to grow in 100% chyle. Chyle is formed from the gut, around 1.5 to 2.5 litres of chyle normally empties into the venous system. The protein content of chyle is usually above 3 g/dl and electrolytes composition is similar to that of serum [1].

A chylothorax results from disruption or obstruction or interruption of the thoracic duct. The incidence of chylothorax after most thoracic surgeries is less than 1.0%. The causes of chylothorax can be categarized as traumatic and nontraumatic. Traumatic cause is mostly due to surgery or trauma. The estimated incidence is

0.5% to 1.0% after cardiovascular surgery [4]. It has been reported after oesophageal, mediastinal, diaphragmatic, and pleuopulmonary surgery. The most common cause of non-traumatic chylothorax is malignancy in which lymphoma account for 70% of cases [2]. The best way to establish the diagnosis of chylothorax is by measuring the triglyceride and cholesterol levels in the pleural fluid. If the pleural fluid triglyceride level is above 110 mg/dL and the ratio of the pleural fluid to serum cholesterol is less than 1.0, the diagnosis of chylothorax is established.

In SLE, the incidence of pleural effusion is 16 to 37 % [5.6]. Most cases are exudative pleural effusion. Rarely in SLE, chylothorax has been reported but the exact incidence is not known. Our case was diagnosed as a case of SLE using revised 1997 American college of Rheumatology criteria.

Management of chylothorax depends on amount of pleural effusion and patients symptoms. It can be managed conservatively by repeated thoracocentesis, dietary modifications and by ICD insertion with pleurodesis. Surgical modality of treatment includes Pleuroperitoneal pump, Fibrin glue to close the leak in the duct, ligation of the thoracic duct by thoracoscopy, and pleurectomy

CONCLUSION

In SLE, Chylothorax is rare cause of pleural effusion and our case highlights the importance of chylothorax in SLE.

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