

The Strange Case of Sternoclavicular Joint Pain in Cirrhosis

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We present the case of a patient of cirrhosis, who has been on multiple evaluation for a left shoulder joint and anterior chest wall pain in whom the diagnosis of tuberculosis of the sternoclavicular joint was made on the basis of clinical suspicion and striking imaging characteristics supported by joint fluid analysis.

A 57 year old, morbidly obese, compensated cirrhotic male, was referred for the complaints of nagging pain in the left upper chest and progressive difficulty in elevation of left arm of one month duration. His X-rays of the shoulder joint, done elsewhere, were normal and symptoms were worsening and prior evaluation for neurological or myopathic diseases was non-contributory. Clinical evaluation revealed mild erythema and non-tender edema (**Fig 1A**) at the sternoclavicular joint (SCJ) on the left side.

Ultrasonography revealed sub pectoral fluid collections and synovial thickening. Aspirated fluid showed 'coagulum' formation (**Fig 1B**), lymphocytic predominance, protein of 4.8 g/dL and adenosine deaminase (ADA) levels 106 U/L (cut off 40 U/L) suggestive of tubercular (TB) effusion. Magnetic resonance imaging (MRI) revealed marked joint effusion, synovial thickening, articular erosions in the contiguous aspects of the left SCJ extending to the manubrium sterni (**Fig 1C**) and medial end of clavicle with large septated para-articular fluid collection under left pectoralis major and minor muscles (**Fig 1D**) with adjacent myofascial edema suggestive of TB arthropathy and cold abscess.

Prevalence rate of TB among cirrhotic patients is 15 times higher than in the general population. Cirrhosis-associated immune dysfunction is a state of systemic immune dysfunction in which dysregulated cytokines, bacteria, and endotoxins predispose to chronic or acute infections [1]. Extra-pulmonary TB involving joints occur in <8% of all TB cases in endemic regions [2]. Cold abscess of pectoral areas are uncommon. Aspirated serosal fluid detects bacilli in <10% cases (bacterial density >10,000/mL). Culture methods are expensive, showing positivity in <25% of cases [3]. PCR assays have little resourcefulness in developing countries. ADA is inexpensive and sensitive collateral test for diagnosis of TB.

In compensated cirrhotics, no studies have been done, comparing the full antitubercular therapy course with regimens containing only two potentially hepatotoxic drugs. Pyrazinamide substitution with fluoroquinolone or aminoglycoside is usually preferred. Prudence calls for use of 2 hepatotoxic drugs in treating TB in compensated cirrhosis with proposed regimens A - rifampicin, isoniazid, pyrazinamide and ethambutol for 2 months followed by 4 months rifampicin and isoniazid; B - rifampicin, isoniazid, fluoroquinolone/aminoglycoside and ethambutol for 2 months followed by 4 months rifampicin and isoniazid; C - rifampicin, isoniazid, and ethambutol for 2 months followed by 7 months rifampicin and isoniazid [4].

In our patient, we used combination C and he is currently doing well at 2 months follow up with improvement in joint movements.

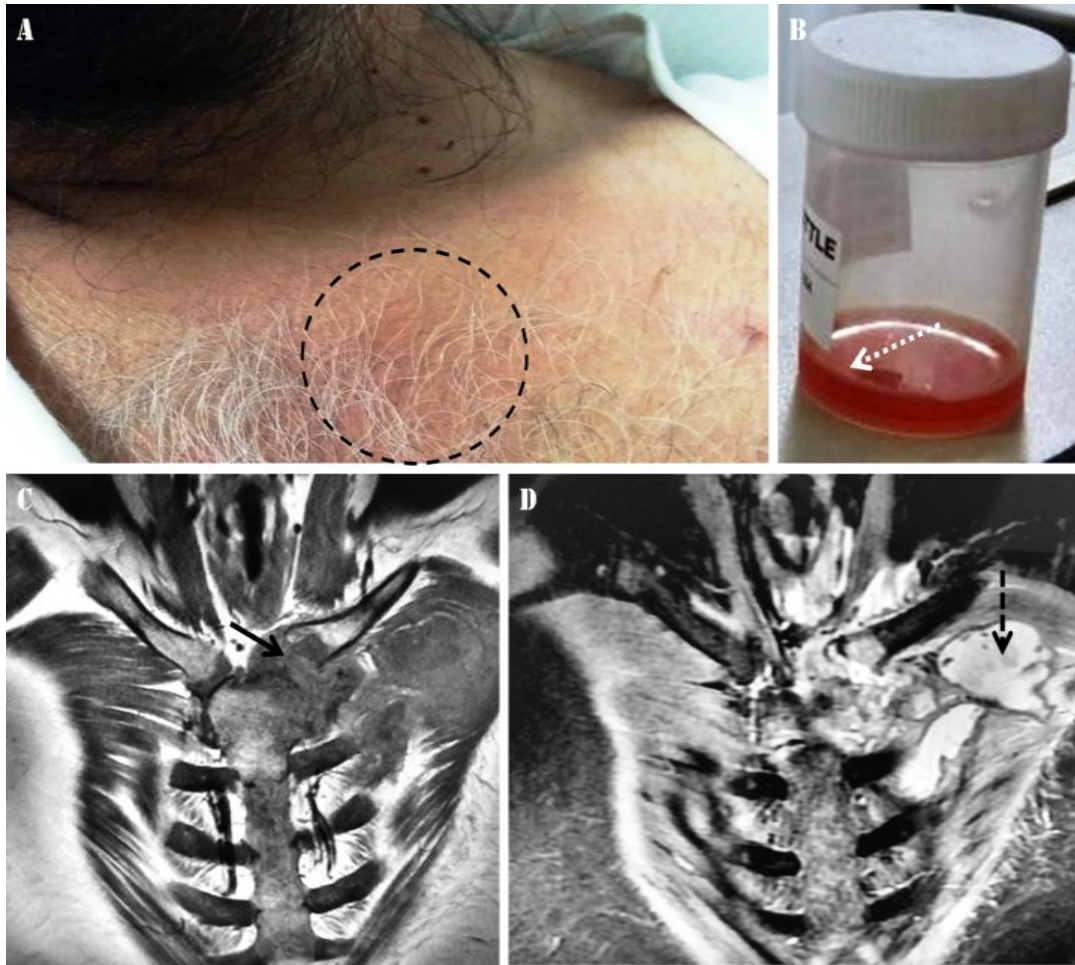


Figure: **Fig 1A** - Mild erythema and non-tender edema at sternoclavicular joint on left side (dashed circle). **Fig 1B** - Fluid aspirated from sub-pectoral collection showed 'coagulum' formation (dashed white arrow). **Fig 1C** - Magnetic resonance imaging showing marked joint effusion, synovial thickening, articular erosions in the contiguous aspects of left SCJ extending to the manubrium sterni (black arrow). **Fig 1D** - MRI showing erosions involving medial end of clavicle with large para-articular fluid collection under left pectoralis major and minor muscles (dashed black arrow).

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