Tuberculous Tenosynovitis

To the Editor:

Osteoarticular tuberculosis is uncommon in our environment especially if only the synovium is involved and even more so if such pathology coexists with active pulmonary tuberculosis. A 69-year-old professional navy man had a history of acute myocardial infarction (14 years ago) leading to dicoumarine treatment, prostatectomy (7 years ago) and smoked 60 cigarettes per day.

The patient came to us complaining of an inflammatory process on the palmar side of the right hand, which had appeared approximately one month previously not presenting much pain but restraining his normal movements. This patient also presented some cough, usually of the irritative type and occasionally with some mucous expectoration, attributed to his smoking habit. He had lost 6 kg in six months.

Exploration revealed scarce dry rales on both pulmonary upper lobes and dactylyogirosis.

Chest radiograph showed nonhomogeneous condensation image, not segmented, on upper halves of both lungs and a small cavity in the left third segment.

Hand radiograph indicated diffuse osteoporosis without signs of focality.

Bacteriologic sputum study results showed the presence of BAAR on visual examination and the culture was positive for Mycobacterium tuberculosis.

During operation, synovitis with abundant serofibrinous fluid was seen, as well as shite, fine, free, ovoid raceminae. The highly-affected superficial tendon had an apparent entry door at the level of the superior flexor tendon, third metacarpal bone.

Anatomopathologic report disclosed synovial tissue consisting of different fragments of tissue with a soft, fleshy consistency.

Microscopically, synovial and pre-tendinous tissue with subacute inflammatory infiltrates, lymphocytes and PMNs were observed; granulomatous areas formed by epithelioid and Langhan’s giant cells with areas of caseous necrosis were also seen.

A diagnosis of caseous granulomatous synovitis was made.

The patient underwent radical (total) synovectomy and closure in two planes. Treatment with rifampin, isoniazide and ethambutol was given for two months, then rifampin and isoniazide for another seven months.

The patient evolved rather well, with full recuperation.

In relation to the pathogenicity of this picture, it is worth mentioning the existence of a quasisymptomatic pulmonary tuberculosis masked by excessive consumption of tobacco. The inoculation chancre on the palm of the hand was caused by the habit among manual workers of spitting on their hands before performing any manual labor such as handling ropes, or the use of picks, shovels, etc. which produces microtrauma and creates an entry door to infections.

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Auscultation of S3

To the Editor:

An interesting contribution to the understanding of cardiac examination has been made by Ishmail et al., who have reported considerable disagreement in the auscultation of S3 under working conditions.

I suggest that a standard for the presence of an S3, such as phonocardiography or perhaps majority opinion of the examiners, would have strengthened this contribution in two ways. First, estimates of the overall S3 prevalence and of the sensitivity and specificity of auscultation for S3 could have been made. Since reliability varies with these parameters, these data would be useful for gauging reliability in screening for (low prevalence of S3), or managing (high prevalence) disease. Secondly, using a standard would have made the reasons for disagreement more clear. For instance, showing similar sex-specific prevalences of audible and “standard”-detected third heart sounds would have been stronger proof against sex as a cause of variability. Furthermore, documentation of changes in intensities of S3 noise between examinations or with provocative measures would have addressed these as sources of variation rather than interexamination time intervals. Reporting of the heart rate during examination may have been a meaningful disclosure.

Definition of the sources of error is important if measures to minimize variation are to be found and ultimately realize the hope for improved skills expressed in the article’s accompanying editorial.

I hope that Dr. Ishmail and colleagues continue their important work towards fulfilling this goal.

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REFERENCES


Almitrine Decreases Compliance of Pulmonary Arteries in COPD

To the Editor:

Almitrine therapy is known to improve blood gases in COPD patients, but the mechanism is not entirely understood. Because the stimulation of ventilation by the drug is inadequate to explain the improved blood gases, a pulmonary vasoconstrictor effect has been suspected. One recently proposed possibility is a change in vascular compliance which—if present—would cause an increase in pulmonary arterial systolic pressure without a change in diastolic pressure, a combination that could be beneficial to gas exchange by affecting the distribution of blood flow within the lung. We have analyzed additional data from the study by Simonneau et al to determine the effect of almitrine therapy on pulmonary arterial systolic and diastolic pressures measured in patients with COPD. Patients (n = 8) were given almitrine orally (3 mg/kg). This dose did not significantly change cardiac output (by thermal dilution), stroke volume (49.5±5 ml vs 50±6 ml) or pulmonary vascular resistance, but did improve blood gases: arterial oxygen tension rose from 53±3 to 70±4 mm Hg (p<0.001) and arterial carbon dioxide tension from 47±3 to 41±3 (p<0.05). Mean pulmonary arterial pressure rose slightly from 32±4 to 36±4 mm Hg, too small a change to account for the large improvement in blood gases.

Our analysis of pulmonary arterial pulse pressure shows more remarkable changes. Systolic pressure rose from 48±5 to 64±5 mm Hg (p<0.001). Diastolic pressure was unchanged. Systolic pressure increased in every patient (Fig 1). The impressive consistency and magnitude of the systolic pressure change associated with no change in stroke volume and no change in pulmonary vascular resistance suggest that almitrine may be acting by decreasing the

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