Case report

Haemophilus influenzae tenosynovitis

SUDHIR BANSAL, C. RICHARD MAGNUSSEN, AND RUDOLPH J. NAPODANO

From St Mary's Hospital, Rochester, New York

SUMMARY  A case is reported of polytenosynovitis in a 31-year-old male during the course of a severe bacteraemic illness caused by Haemophilus influenzae type b. The clinical presentation was similar to tenosynovitis caused by other bacterial or viral agents. As the management of the H. influenzae tenosynovitis would differ from that due to other causes, the addition of H. influenzae type b to a differential of tenosynovitis should be considered. Recognition and prompt treatment by appropriate antibiotics may be important to avoid supplicative complications affecting the tendons. As the pathophysiology of the tenosynovitis is not clear, careful bacteriological and immunological assessment must be obtained.

Haemophilus influenzae has a predilection for infecting serosal surfaces. To our knowledge there has been only one reported case of tenosynovitis associated with arthritis caused by H. influenzae type b (Raff and Dannaher, 1974). In this report we describe a patient with tenosynovitis associated with a H. influenzae type b respiratory infection and sepsis.

Case report

A 31-year-old male was admitted to hospital on 2 September 1977 with a 4-day history of fever, chills, sweats, and pleuritic chest pain. He had also noted painful swelling, erythema, and warmth on the back of the left hand and foot. This pain was exacerbated by dorsiflexion of the fingers and toes. He was 'allergic' to penicillin. The patient appeared acutely ill with blood pressure 170/60 mmHg, temperature 39·0°C, pulse 140/minute, and regular respirations 26/minute. In addition to an injected pharynx, tender bilateral anterior cervical adenopathy, clear lung fields, and sinus tachycardia with no audible rubs, murmurs, or extra sounds, the dorsal aspect of the left wrist and left foot were swollen, warm, and erythematous. Active movements of the left wrist and fingers produced more pain than did passive movements. Radiographs of the affected parts were normal. Four blood cultures obtained in an hourly sequence were positive for H. influenzae type b. The organisms were negative for B-lactamase activity, and the patient was placed on parenteral ampicillin (12 g/day intravenously) after skin tests which were negative for penicillin allergy. The patient's illness resolved over 2 weeks.

Comment

A diagnosis of acute tenosynovitis is based on the presence of swelling accompanied by some erythema and induration (Clain, 1967; Pinals, 1972). Such patients tend to keep the hand and fingers supported and slightly flexed to prevent any movement (Bell, 1968). There is a greater difficulty in active than passive movements (Mills et al., 1976). Stretching the tendons passively produces pain in the area of the inflammation. Additional support for the diagnosis of tenosynovitis is provided by negative x-rays (Mills et al., 1976).

In adults a variety of illnesses have been described as caused by H. influenzae type b, namely pneumonia (Quintiliani and Hymans, 1971), meningitis (Eykin et al., 1975), purulent pericarditis (Crossley et al., 1973), septic arthritis (Raff and Dannaher, 1974), endocarditis (Lynn et al., 1977), epiglottitis (Hawkins et al., 1973), and recently cellulitis (Drapkin et al., 1977). One of the patients with arthritis developed tenosynovitis (Raff and Dannaher, 1974).
Tendon sheath inflammation may be caused by a variety of illnesses, for example, trauma, rheumatoid arthritis, tuberculosis, gout, gonococcal infections, secondary and tertiary syphilis, and coccidioidomycoses.

The pathogenesis of tenosynovitis in each disease varies. Tuberculous tenosynovitis is caused by invasion of the tendon sheaths by mycobacteria. This is validated by demonstrating caseation necrosis in the tendon sheaths and/or isolation of the bacillus via aspiration and culture or guinea-pig inoculation (Bell, 1968). On the other hand rheumatoid arthritis is an immune complex disease. A diagnosis of rheumatoid tenosynovitis is based on associated features of the disease and seropositivity. Involvement of the tendon sheath in rheumatoid arthritis results in a proliferative process (Sones, 1968). Though the tendon sheaths in rheumatoid arthritis have been surgically excised, detailed histopathology and immune-fluorescent studies have not been done. However, the fluid and synovium obtained from joint spaces have been studied and show typical features of an immune complex disease, namely, low complement levels in joint fluid and deposits of antigen-antibody complex on the synovium (Zvaifler, 1974). From these data one may assume that the tendon sheaths undergo similar changes in rheumatoid arthritis. Gonococcal tenosynovitis develops in the acute bacteraemic phase of the illness (Holmes et al., 1971). Studies have been completed on the associated acute synovitis of the joint spaces. In this early phase of the illness either fluid is not obtained or it is sterile, so that some authors have suspected a 'hypersensitivity' phenomenon (Wright, 1963) or cross-reactivity of the gonococcal and synovial antigens. So far these hypotheses have not been confirmed by laboratory testing.

From the above evidence we feel secure in suggesting the possibility of polytenosynovitis occurring in a bacteraemic illness caused by H. influenzae type b. However, we cannot state with certainty whether this is due to direct invasion of the H. influenzae bacteria or an immune reaction involving the tendon sheaths. We would suggest that in similar patients immunological and bacteriological studies may be undertaken to confirm the exact pathogeness. H. influenzae type b. infection should be added to the list of differential possibilities for an acute tenosynovitis.

References


