SHORT REPORTS

Atypical infectious mononucleosis in the elderly

The relationship between Epstein-Barr virus (EBV) and infectious mononucleosis is well established.[1] Since the antibody to EB viral capsid lasts for life,[2] the age at which infection is acquired in any community may be studied. This varies according to country and social group. In rural Uganda over 80% of children have antibodies by the age of 1 year, whereas in the United Kingdom only 40% of children are positive at 5 years and 90% at 25 years. In a prospective study in the age group 18 to 25 years, infection was accompanied by classical infectious mononucleosis in about 45% of people and the heterophil antibody was shown to be present in almost all cases. The other 55% found to have seroconverted during the previous year either had no symptoms or had symptoms indistinguishable from those of a similar uninfected group of the same age.[3] Over the age of 50 infection is rare, partly because there are very few remaining susceptibles. The diagnosis may therefore not be considered. Also the illness may be atypical. We report here two patients aged 55 and 74 with serologically confirmed infectious mononucleosis. In neither case was the diagnosis suspected from the clinical presentation.

Case reports

Both patients were acutely ill on admission with an unremitting fever of eight days' duration. They were thought to be septicaemic, related in case 1 to intra-abdominal sepsis and in case 2 to endocarditis. Clinical details are outlined in the table. A lymphocytosis first developed on day 16 in case 1 and on day 28 in case 2, and was associated with conversion of the monospot test. The EBV IgG antibody titre rose in both patients (1:8→1:256, 1:8→1:128) with a positive IgM. Both completely recovered.

Comment

In young people anginose, glandular, and febrile forms of infectious mononucleosis are well recognised, 80% of cases having cervical lymphadenopathy and 85% sore throat. Reports of elderly patients with heterophil antibody-positive infectious mononucleosis are few. Horwitz *et al* collected seven patients aged between 40 and 78, and concluded that the presentation in this group differed little from that in younger patients except that the fever tended to be more prolonged.[4] Our observations endorse the persistence of fever, but in neither of our patients was the diagnosis suspected. It was not made until a repeat blood film showed atypical mononuclear cells. It was confirmed in both patients by the discovery of heterophil antibodies with a rising titre of specific IgG and IgM antibodies to EB viral capsid antigen. EBV-specific IgM usually disappears within weeks of an acute infection. A particularly fulminant reaction to EBV infection has been reported in a family with a humoral and T lymphocyte defect,[5] but we found no abnormality of lymphocyte function in our case 1. Proliferation of killer T cells in response to circulating infected B cells is responsible for the acute fever, lymphadenopathy, and hepatosplenomegaly characteristic of infectious mononucleosis.[2] The mechanism of persistent fever in cases such as ours is not known, but is probably related to failure of an adequate immunological response. There may be a suboptimal T cell proliferation in response to the virus-determined surface antigen resulting in a prolonged antigenic stimulus. A diagnosis of infectious mononucleosis in patients presenting with persistent fever should not be discounted merely on grounds of age and an initially normal blood film. Storage of acute phase serum is often valuable for confirmation of diagnosis.

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[2] Epstein, M A, and Achong, B G, *Lancet*, 1977, **2**, 1270.
[3] University Health Physicians and PHLS Laboratory Investigation, *British Medical Journal*, 1971, **4**, 643.
[4] Horwitz, C A, *et al*, *American Journal of Medicine*, 1976, **61**, 333.
[5] Bar, R S, *et al*, *New England Journal of Medicine*, 1974, **290**, 363.

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Late complication of endoscopic oesophageal tube insertion

Endo-oesophageal tubes have been used for 20 years to relieve dysphagia due to oesophageal or high gastric malignancies.[1 2] With the advent of fibreoptic endoscopy the technique has been used much more often, and recent refinements have included the development of an introducer for the placement of a self-retaining Celestin tube.[3] We report a late complication of the use of such an introducer.

Case report

A 57-year-old man presented with a six-month history of progressive dysphagia, which was found to be due to a malignant stricture from a carcinoma of the gastric fundus. He was considered a poor surgical candidate because of two previous cerebrovascular accidents resulting in a mild left hemiplegia. He was referred for peroral Celestin tube insertion before radiotherapy. The stricture was initially dilated with Eder-Puestow dilators, and two days later a Celestin tube was inserted using a Nottingham tube introducer. The dysphagia greatly improved. After radiotherapy to a total dose of 5656 rads to the left chest over the next month the patient remained well and could eat small quantities of solid food.

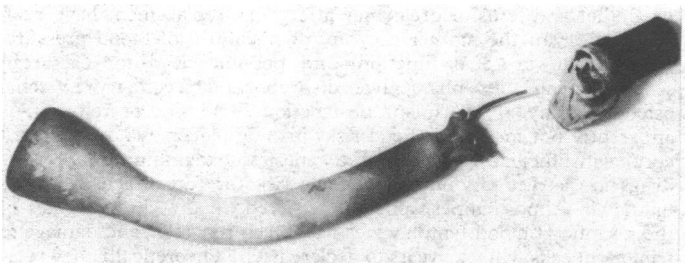
Six months later he was readmitted with a 10-day history of central colicky abdominal pain and vomiting. Plain and barium-meal radiographs showed the Celestin tube apparently in the duodenum. At gastroscopy the funnel of the tube was lodged in the pylorus. It could be pulled into the body of the stomach, but repeated attemps to extract the tube completely were unsuccessful. At laparotomy the proximal funnel of the Celestin tube was

Clinical details of two patients aged 55 and 74 years with infectious mononucleosis

	Presentation		Investigations	
	Symptoms	Signs	Abnormal	Normal
Case 1	Sweating, rigors, confusion, dry cough	Fever 39·2°C	ESR 55 mm/h Serum AST 137 IU/l Serum ALT 93 IU/l Serum bilirubin 31 μmol/l EEG low frequency	Full blood count, urea and electrolyte concentrations Toxoplasma and CMV serology T cell function CSF Blood/urine/sputum cultures Gallium isotope scan
Case 2	Fatigue, malaise	Fever 38·1°C, atrial fibrillation, mitral regurgitation, hepatosplenamegaly	Serum AST 100 IU/l	FBC, U + E, ESR Toxoplasma and CMV serology Autoantibodies Blood/urine/marrow cultures

Conversion: SI to Traditional Units—Bilirubin 10 μmol/l ≈ 0·6 mg/100 ml.

found at the pylorus but its distal end was in the ileum about 20 cm from the ileocaecal valve. The two portions were still attached by the nylon coil that reinforces the latex of the Celestin tube (figure). The small bowel between the two portions of the tube was gathered and shortened over the nylon, and there were seven full-thickness ulcers on the mesenteric border of the 30 cm of ileum immediately proximal to the distal end of the tube. The ulcerated bowel was resected and an end-to-end anastamosis performed. At endoscopy and laparotomy there was no macroscopic evidence of residual tumour. The patient remained well for a further six months but then rapidly developed severe dysphagia due to recurrent malignancy. He died 20 months after the onset of his initial symptoms.



Two sections of Celestin tube removed at laparotomy.

Comment

Structural deterioration of Celestin tubes has been reported but only after they had been in position for more than two years.[4] In our case the tube had been in place for about six months, and probably the early deterioration was due to damage during insertion. It has subsequently been shown that the pressure exerted on the inner wall of the tube by the introducer can split the latex wall of the tube and release the inner nylon coil (L R Celestin, personal communication). Another possibility is that the tube was damaged by the high dose of irradiation the patient received. Nevertheless, this is unlikely and has never been reported after operative insertion of a Celestin tube. In view of this case care must be taken to prevent undue force on the inner wall of the Celestin tube, and perhaps other methods of inserting the tube should be considered.

We thank Dr J A Bullimore, consultant radiotherapist, for permission to publish this case.

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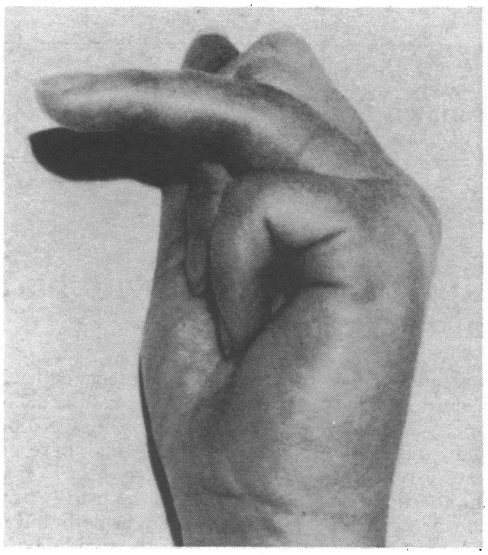
Tenolysis in juvenile diabetic cheiroarthropathy

Hand changes are not uncommon in diabetes mellitus. They are principally due to small-muscle wasting caused by peripheral neuropathy or a mononeuropathy, usually a carpal tunnel syndrome or ulnar nerve palsy.[1] Although Charcot arthropathy in the UK is now most often caused by diabetes (diabetic osteoarthropathy) it is extremely uncommon in the hand.[2] There is, however, little recognition of juvenile diabetic cheiroarthropathy, a painful arthropathy of unknown cause which affects the interphalangeal joints. It is associated with palmar flexion and is apparently specific to young longstanding insulin-dependent diabetics whose diabetes had its onset before puberty.[3] This cheiroarthropathy is usually regarded as chronic and we report here the benefit of tenolysis in this condition.

Case report

A 28-year-old woman who had had diabetes for 20 years and was controlled on twice daily soluble and isophane insulins presented with an 18-month history of stiffness and pain in the right and left ring fingers. This affected the metacarpophalangeal and proximal interphalangeal joints and was associated with dermal oedema and a sclerodermatous-like appearance. Her fingers were swollen and tender and could flex to within only 4 cm of the palm (see figure). There was no thickening or shortening of the palmar aponeurosis to suggest Dupuytren's contracture. No other joints were affected. There were no features of Raynaud's phenomenon or systemic sclerosis. Erythrocyte sedimentation rate was 7 mm in first hour (Westergen), she was negative for antinuclear factor, and the Rose-Waaler test was normal, as were radiographs of both hands. Background retinopathy was present, as was proteinuria (1·8 g/24 h) with a creatinine clearance of 77 ml/min. There were no neuropathic features in the arms or legs. Because the hand deformities were affecting her work as a music teacher, exploration of the tendon of the right ring finger was performed, which showed some fibrous thickening of flexor sheath at its palmar end. Tenolysis was carried out until the tendon could be moved freely. Histological examination of the fibrous tissue showed only a mild non-specific oedematous change. During the next nine months the pain and swelling in the joints of the right ring finger gradually disappeared. Tenolysis of the left ring finger was then performed with a similar good result. Two years later the right long finger gradually became affected and could flex to only 3 cm from the palmar surface. Tenolysis was performed six months later, and a similar operation on the left long finger was then carried out, producing free movement of the tendon. As before, the pain and swelling in the affected metacarpophalangeal and proximal interphalangeal joints gradually settled over six months. At the time of writing, the right fifth ring finger had become affected and a fifth tenolysis was to be performed.



Left hand showing diabetic cheiroarthropathy with swelling of metaphalangeal and proximal interphalangeal joints of ring finger with flexion limited to within 4 cm of the palm.

Comment

Diabetic cheiroarthropathy causes pain, stiffness, and periarticular swelling of the metacarpophalangeal and proximal interphalangeal joints, especially of the long, ring, and fifth fingers, associated with a painful palmar flexion deformity.[3] It is an uncommon condition but the evidence suggests that it is probably associated with diabetes mellitus. The differential diagnosis is from Dupuytren's contracture, again more common in diabetics,[4] which is painless and has puckering of the skin, often with a thickened nodule; at operation the palmar fascia in Dupuytren's contracture is hard and gritty whereas the palmar fascia in cheiroarthropathy is more supple.[5] Also, diabetic cheiroarthropathy must not be confused with a simple trigger finger, where there may be some swelling proximal to the fibrous constriction but