There were no obvious differences between the primigravidae and multigravidae, and no apparent association between success and the duration of pregnancy or estimated time since fetal death. Four out of six patients with a missed abortion whose uterine size was below 14 weeks underwent suction evacuation 24 hours after treatment, abortion having failed to occur. All patients were well controlled with the analgesic used; only one needed three injections, while most of those with uterine size below 29 weeks needed none or one injection immediately before abortion. Four patients vomited, two of whom had diarrhoea, and all were given 15 mg PGE_2. No other side effects were observed. There were no complications. Surgical evacuation was performed on six patients, and one retained placenta was removed manually. Four patients lost over 500 ml blood, and one required transfusion. No patient was feverish, and none developed consumption coagulopathy.

Conclusions

The results with vaginal PGE_2 gel were comparable with those obtained in this unit with extra-amniotic administration,[2] 31 patients (62%) having aborted or delivered without further uterine stimulation; in the remainder the oxytocin infusion generally rendered expulsion of the conceptus inevitable within a few hours. The vaginal route, however, is simpler than the intrauterine and avoids the risk of sepsis without reducing therapeutic efficacy. Physical and emotional distress were minimal, 11 patients not requiring analgesia and only four suffering any gastrointestinal side effects. Abortion and delivery times compared favourably with those of Southern *et al*,[4] who used repeated administration of 20 mg PGE_2 vaginal pessaries, which provoked diarrhoea in 42·7% and vomiting in 56·4%.

The 15 mg dose of PGE_2 used for a uterine size below 29 weeks seems appropriate; a larger dose, although possibly reducing the need for oxytocin, would probably provoke more side effects. Success was reduced when uterine size was 11-13 weeks; however, suction evacuation was then easily performed and seems reasonable treatment when the uterus is this small and the cervix already softened and dilated. In more advanced pregnancies the larger dose to reduce the need for oxytocin might be inappropriate. Violent labour in the presence of a dead fetus incurs the risk of amniotic fluid embolism, and any great increase in dosage might therefore be imprudent.

We thank Sister Anna Fry and Sister Sue Bradley for expert nursing care, and the medical staff of the John Radcliffe Hospital for referring patients.

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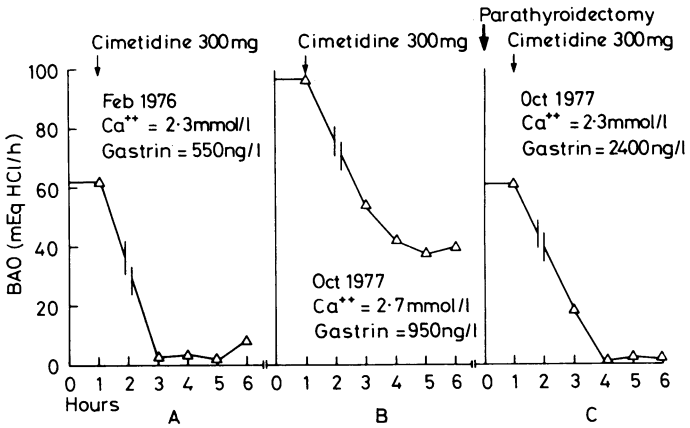
Hyperparathyroidism—a reversible cause of cimetidine-resistant gastric hypersecretion

Cimetidine has been successfully employed in the long-term treatment of gastrinoma.[1-4] We report the case of a patient in whom changing parathyroid function appeared to effect the course of acid-peptic disease and the response to the drug.

Case report

A 55-year-old woman developed hyperparathyroidism in 1953. She did well after parathyroidectomy (3 1/2 glands). In 1974 she developed Zollinger-Ellison syndrome and a non-β islet cell tumour was excised from her pancreas (tail) without return of the serum gastrin concentration or gastric hypersecretion to normal. During 1975 her serum calcium concentration rose to 3·5-4·3 mmol/l (14-17 mg/100 ml) and parathyroid tissue was excised from her mediastinum. Multiple pieces of parathyroid tissue were implanted into the left forearm in November 1975. In February 1976 dyspepsia and diarrhoea led to her admission to hospital. She had multiple peptic ulcers and watery diarrhoea (3 l/day). Her fasting serum gastrin concentration was 550 ng/l (normal <150 ng/l) and basal acid output (BAO) was 50-60 mEq/h. Her serum calcium concentration was normal (2·3 mmol/l (9 mg/100 ml). Cimetidine (300 mg) by mouth reduced BAO to less than 10 mEq/h from the second to the fifth hour after the dose[3] (fig A). She was discharged taking cimetidine 300 mg six-hourly. Six months later diarrhoea recurred. The serum calcium concentration was 2·5 mmol/l (10·2 mg/100 ml). From October 1976 to February 1977 her serum calcium concentration fluctuated between 2·2 and 2·9 mmol/l (8·8 and 11·6 mg/100 ml). In June 1977 measurements of parathyroid hormone (PTH) concentrations in the antecubital veins showed on the left 86 μg/l and on the right 0·41 μg/l (normal <0·22 μg/l), a highly significant difference. These figures established the diagnosis of systemic hyperparathyroidism and the presence of functioning tissue in the left arm. By July 1977, because of dyspepsia and diarrhoea, the dose of cimetidine was increased from 1·2 to 1·8 g/day. In October 1977 her BAO was reassessed 48 h after stopping cimetidine.[4] The effect of the drug on BAO was then recorded (fig B). Compared with February 1976 the BAO had increased by about 50%. Cimetidine (300 mg) by mouth continued to cause an absolute reduction in BAO, quantitatively similar to that achieved previously, but in the postdrug period acid secretion greatly exceeded that measured 18 months earlier. Serum gastrin concentration at this time was 800-950 ng/l, and during the test the serum calcium concentration was 2·7 mmol/l (11 mg/100 ml). The identifiable parathyroid tissue was then excised from her left forearm and the serum calcium concentration fell. On the fourth postoperative day the response to cimetidine (300 mg) was again measured (fig C). At this time the serum calcium concentration was 2·3 mmol/l and the serum gastrin concentration had risen to 2400 ng/l. In spite of the hypergastrinaemia the BAO and drug responses had returned to those of February 1976. Cimetidine 300 mg four times a day abolished diarrhoea and dyspepsia.



Effect of single oral dose of cimetidine on basal acid output. A: Three months after excision of mediastinal parathyroid tissue and forearm implantation, and 14 months after excision of islet cell tumour of tail of pancreas. B: Immediately before excision of functionally active parathyroid tissue from left forearm, 18 months after A. C: On fourth postoperative day after B. Serum gastrin concentrations on day of each test are shown in relevant panels.

Conversion: SI to traditional units—Calcium: 1 mmol/l ≈ 4 mg/100 ml.

Comment

This patient seemed to be "escaping from control" with cimetidine. Development of hyperparathyroidism and hypercalcaemia was accompanied by a sharp rise in BAO. Surgery, which corrected the hypercalcaemia, rapidly reduced BAO to the initial level and abolished symptoms. The absolute response to cimetidine did not change much over the 18-month period, but the BAO rose by 50% when the serum calcium and PTH concentrations rose. Since BAO is the result of a number of stimuli, such as acetylcholine, histamine, and gastrin, the increase in BAO, not blocked by cimetidine, may have been mediated by a stimulus other than histamine.[5] Altered response to cimetidine may be important in hyperparathyroidism. Conversely, patients with ulcer who deteriorate on cimetidine merit investigation of their parathyroid state.

We thank the staff of the Milton S Hershey Medical Center, Hershey, Pa, who contributed much to the management of the patient, particularly Drs T McGlynn and William Graham.

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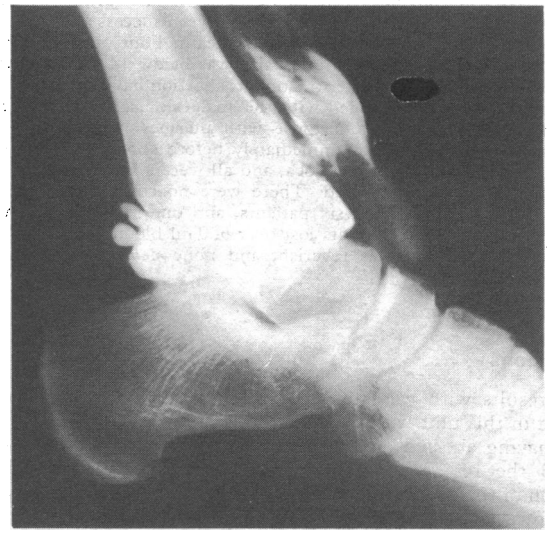

Synoviogram of left ankle joint showing immediate communication with synovial cyst.

Anterior synovial rupture of ankle joint presenting as deep venous thrombosis

Posterior synovial rupture of the knee joint with resultant oedema and swelling in the calf closely resembles deep venous thrombosis.[1] This occurs most commonly in patients with rheumatoid arthritis but occasionally in other forms of arthritis.[1 2] We report a case of monoarthritis of the ankle joint presenting with acute swelling of the ankle and calf due to anterior synovial rupture mimicking deep venous thrombosis.

Case report

A 27-year-old woman presented to the casualty department with a three-day history of painful swelling of the left ankle and calf. Deep crural venous thrombosis was diagnosed, but closer questioning disclosed a three-month history of intermittent painful swelling of the ankle after minor trauma. Three days before admission the ankle had become hot, painful, and swollen but she had continued to walk, though with difficulty, and the swelling and pain had spread to the whole leg below the knee. Examination confirmed swelling of the calf and ankle but with maximum tenderness around the ankle joint, which was warm. Minimal calf tenderness was noted, and there was pain in the calf on dorsiflexion of the foot (Homans's sign). No other abnormalities were found apart from mild genu recurvatum.

After a period of bed rest the calf swelling had diminished considerably and a fluctuant cystic swelling 5 cm in diameter could be palpated above and anterior to the ankle joint; from this 20 ml of straw-coloured fluid was aspirated, which contained 12×10^9 cells/l (12 000/mm^3), mainly neutrophils, but no crystals. Culture was negative. Synoviography of the ankle joint showed a communication between the joint and the swelling but not into the calf, with only minor leakage of contrast material (figure). We considered that the calf swelling and pain were secondary to the release of some irritant synovial fluid into the tissues of the leg consequent to joint rupture, the rest of this fluid forming the cystic swelling. No localised swelling had been previously noticed by the patient. Phlebography was not considered necessary in view of the rapid clinical improvement.

Other investigations showed haemoglobin 12·1 g/dl; white cell count 7×10^9/l (7000/mm^3); plasma viscosity 1·70 cp; and urate concentration 0·31 mmol/l (5·2 mg/100 ml) (normal 0·09-0·36 mmol/l; 1·5-6·0 mg/100 ml). An RA latex test was negative and antinuclear antibodies absent. An *x*-ray film of the ankle showed a short segment of periosteal reaction at the medial aspect of the lower tibia; sacroiliac joints were normal. HLA antigens A1 and 32 and B8 and 15 were present.

Comment

Synovial rupture in inflammatory arthritis causes the release of irritant synovial contents resulting in pain and swelling.[1] When the knee joint is affected the resulting clinical picture closely resembles that of deep venous thrombosis and is not uncommon in patients with early rheumatoid arthritis.[1] Other joints may also rupture, including the wrist, elbow, shoulder, and hip, but rupture of the ankle joint is rare.[3] Baker first reported a case of a large synovial cyst anterior to the ankle joint causing swelling in 1885.[4] Synoviography of the ankle joint in rheumatoid arthritis has shown synovial outpouches and connections with tendon sheaths but no large anterior cysts or rupture.[5]

Studies of pressure in the knee joint show that enormous rises occur during normal use when an effusion is present,[1] which are highest in patients with rheumatoid arthritis. High pressures might be expected in other weight-bearing joints such as the ankle, and it is surprising that rupture is so rare.

We are unaware of any report of synovial rupture of the ankle joint causing the degree of pain and swelling seen in this case and also of such rupture occurring so early in a monoarthritis. Anterior synovial rupture of the ankle joint must be considered in the differential diagnosis of deep venous thrombosis.

We wish to thank Dr A St J Dixon, consultant rheumatologist, for permission to report this case.

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Aneurysm after arterial puncture in Behçet's disease

Behçet's disease may be complicated by aneurysms of both central[1 2] and peripheral arteries.[3] We report a case in which arterial puncture apparently predisposed to aneurysm.

Case history

A 24-year-old Saudi Arabian soldier had suffered for eight years from recurrent episodes of oral, inguinal, and scrotal ulcers; non-deforming