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MEDICAL MEMORANDA

Caecal Rupture in Psychotic Patients

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Megacolon in psychotic patients has been sporadically documented, mainly by American workers. The details of two patients seen in this department within a short time of one another are given below; they had the additional unusual complication of ruptured caecum.

Case 1

An 82-year-old man had been admitted to hospital because of chronic schizophrenia for 42 years. Drug treatment was minimal but included benzotropine 2 mg daily. Over a period of four days he developed recurrent vomiting, and a diagnosis of subacute intestinal obstruction due to gross constipation was made. An enema was given with satisfactory result. Obstructive signs did not remit, however, and he was transferred to a general surgical ward. On examination he was dehydrated, had generalized abdominal tenderness, and an empty rectum on digital examination. Nasogastric suction and intravenous fluid replacement were started, and radiographical examination showed subdiaphragmatic gas. He died before laparotomy could be performed.

Necropsy showed a faecal peritonitis with a dilated ileum. The caecum was grossly distended, paper-thin, and had a medial perforation of 3 cm in diameter. The transverse and descending colon were also dilated, but most of the sigmoid and all of the rectum were empty. The colonic contents were soft throughout. Histological examination showed normal ganglion cells at all levels of the colon and anal canal.

Case 2

A 79-year-old woman with a 13-year history of depressive illness necessitating inpatient treatment developed alternating diarrhoea and constipation over a period of one month. Digital examination showed an empty rectum, but in view of her continued symptoms and a palpably enlarged colon numerous enemas and laxatives were given over several days with poor results. Her clinical condition deteriorated and she was admitted to a general medical ward. The diagnosis of large bowel obstruction was confirmed clinically, and in spite of further repeated attempts at evacuation by enema she developed acute pulmonary oedema, tachycardia, and hypotension, and she died soon afterwards.

At necropsy the colon was grossly dilated throughout its length, the anal canal alone being unaffected. The caecum was especially distended and thinned, and there was a slit-like 1 cm anterior perforation with a faecal peritonitis. The faeces were soft and foamy and there was no organic obstruction. As in case 1 ganglion cells were present at all levels of the large bowel and anal passage.

Comment

Megacolon has previously been noted in both psychoses (Ehrenthel and Wells, 1955) and Parkinson's disease (Lewitan et al., 1951). The aetiology is not clear but in Parkinson's disease anticholinergic drugs may be a factor. The patient in case 1 had been taking an anticholinergic agent (benztropine) for a considerable period up to the time of his admission to a surgical ward, but there was no such history in case 2. Chlorpromazine has been incriminated as a rare cause of severe constipation (Oettinger, 1958), and Zimmerman (1962) was able to produce an animal model with striking similarities to the human syndrome by the intraperitoneal injection of large doses of this drug in rats. Though both patients reported here had chlorpromazine at some time during their illnesses neither had ingested the drug in large amounts or for a prolonged period before the appearance of gastrointestinal symptoms. Since iatrogenic causes now seem to be unlikely it is probable that most cases of megacolon are essentially psychiatric in origin, as proposed by Ehrenthel and Wells (1955).

Whatever the cause these patients have soft faeces and markedly decreased bowel and sphincter tonus, and evacuation, either by enema or spontaneously, is difficult or ineffective. Such are the difficulties in management that Watkins and Oliver (1965) have recommended subtotal colectomy, apparently with gratifying results.

No cases previously described have developed caecal rupture though this is the most distensible part of the colon with a bursting pressure of about 50 mm Hg in the normal viscus (Burt, 1931). Noveroske (1972) confirmed this and pointed out that, as with all distensible tubes, the colon must obey the law of Laplace. It follows that a caecum which is already distended and thinned will tolerate a proportionately smaller increase in intraluminal pressure before splitting, and it is significant that both these patients had enemas, multiple in one case, with inevitable increase in intraluminal pressure immediately before sudden physical deterioration.

It seems, therefore, that colonic rupture must take a place alongside the other often forgotten dangers of enema, which include chemical colitis (Hirschman, 1927; Barker, 1945; Pike et al., 1971), anaphylactoid reaction (Smith, 1967; Egdell and Johnson, 1973), and direct traumatic damage (Bendit, 1945). It is extremely unlikely that this complication is as rare as its absence from the literature suggests. Megacolon in psychotics is relatively common, but necropsy in mental hospital patients is not as a rule routine. Awareness of the condition and an

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increased index of suspicion will probably uncover more cases.

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Necrotizing Vasculitis and Ulcerative Colitis

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Skin lesions are frequent extracolonic manifestations of ulcerative colitis, the incidence of such lesions ranging from 6% to 34% of cases (Bockus *et al.*, 1965). We describe a patient with severe ulcerative colitis who developed extensive necrotic skin lesions due to cutaneous vasculitis and survived.

Case Report

The patient, a 26-year-old man had enjoyed good health till May 1972, when a superficial thrombophlebitis occurred in the left popliteal fossa. This healed spontaneously within a few days. From that time on he suffered from a migrating thrombophlebitis affecting alternately the knees and elbows. Five months later his general condition deteriorated; he felt tired and weak and developed low-grade fever. Extensive physical, biochemical, radiological, and serological examination showed nothing abnormal, and skin biopsy of the left elbow showed no underlying disease. Treatment with coumarin was given from December 1972 to July 1973, during which no relapse of migrating thrombophlebitis occurred.

The second day after stopping the coumarin the patient became feverish and developed painful blue spots on both thighs which disappeared spontaneously within a day. Two days later a more extensive bluish area appeared on the inner aspect of the left thigh which did not resolve but instead central haemorrhagic necrosis of the skin developed. Again extensive examination was carried out for a systemic disease but no specific abnormalities were found. Treatment with coumarin was reintroduced but was stopped after three days because of haematuria and bloody stools. At that time the patient also suffered a short-lasting attack of arthritis of the jaw joints. X-ray studies of the thorax and colon and an intravenous pyelogram were thought to show nothing abnormal. At sigmoidoscopy the rectal mucosa appeared to be haemorrhagic. On 16 August two biopsy specimens were taken from indurated remnants of healed thrombophlebitis on the back of the left thigh and upper calf. That evening a large, painful, bluish skin discoloration appeared centred around the previous already necrotic lesion on the left thigh. On 18 August he was transferred to this hospital for investigation of blood-clotting abnormalities.

On admission he appeared ill and pale-looking. Temperature 38.9°C. Examination of the head, neck, lungs, and heart showed nothing abnormal. His abdomen was slightly tender on palpation

but was otherwise normal. On the anterior aspect of the left thigh there was an extensive slightly raised area of bluish purple discoloration. The lesion was sharply and serpigiously circumscribed and surrounded by an erythematous zone. Small vesicles and a few blisters filled with haemorrhagic fluid were present within this area, which surrounded a centrolateral black necrotic patch (fig. 1 a, b). The left side of the scrotum was black and necrotic and surrounded by an erythematous zone. All the peripheral arteries pulsated normally.

FIG. 1—(a) Necrotic skin lesion on anterior aspect of left thigh and scrotum on admission. (b) Small vesicles and blisters filled with haemorrhagic fluid within bluish black skin. (c) and (d) Microscopical appearances of colonic mucosa before treatment. Severe inflammation was confined to mucosal layer, with cryptitis, crypt abscesses, and depletion of mucus. No evidence of vasculitis was present in submucosa (arrows). ($\times 50$). (e) Microscopical appearances of mucosa of colon after one and a half months of treatment. Striking regression of inflammatory reaction had occurred. Epithelium was of normal appearance. ($\times 50$)

Urine analysis and blood chemistry showed nothing abnormal throughout the illness. The E.S.R. was 73 mm in the first hour, haemoglobin 9 g/100 ml, and W.B.C. 20,000/mm³. Fibrin degradation products were increased and the Thrombotest time (Owren) was prolonged; all other coagulation values were normal. No cryoglobulins were detected. Widal, Bang, and Yersinia agglutination reactions were negative. The cytomegalic virus complement fixation titre was 1/16. The antistreptolysin titre was 100 U and the Rose-Waaler and Latex test results were negative, as was the Wasserman reaction. Blood cultures remained sterile on seven occasions. Stool cultures were negative for salmonella, shigella, yersinia, ova, and parasites. Faecal examination for occult blood using benzidine was strongly positive.

The first day in hospital the patient developed arthritis and periarthritis of the right elbow, and 12 hours later the same symptoms appeared in the left elbow with concomitant swelling and oedema of the upper arm. Next day he experienced sharp

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