MEDICAL MEMORANDA

Oxygen Therapy for Pneumatosis Coli

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Pneumatosis coli is an uncommon condition of unknown aetiology which is characterized by multiple gas-filled cysts in the submucosa of the colon. The cysts vary in size from a few millimetres to one or two centimetres and may obliterate the bowel lumen.

We describe here a patient with incapacitating symptoms from rectosigmoid pneumatosis coli who was treated with a high concentration of oxygen, a form of simple, safe, and effective therapy not widely known.

Case Report

This patient had successfully undergone aortic and mitral valve replacement for rheumatic valve disease in February 1971 when she was 55 years old. Apart from the injection of bleeding haemorrhoids with sclerosant twice in 1972 she had had a normal bowel habit.

In May 1973 she complained of passing loose motions two or three times a day and excessive flatus per rectum. Sigmoidoscopy and a barium enema examination showed nothing abnormal. Four months later she complained of faecal soiling of her underclothes and by February 1974 she was wearing pads day and night because of the involuntary passage of much frothy mucus and flatus. She needed to defaecate often and urgently, and when she got the call to stool was unable to prevent faecal incontinence, which made her frightened to leave the house.

On examination there was no evidence of anaemia or weight loss. Abdominal examination showed nothing abnormal, but a large polypoid mass about 6×4 cm could be felt low in the rectum extending almost to the anal canal. On proctoscopy the lower limit of this mass could be seen bulging beneath a pale but non-ulcerated mucosa. A biopsy specimen was taken for histological analysis, and the patient admitted with a clinical diagnosis of carcinoma of the rectum.

Blood test results were normal and no pathogens were isolated from the stool, which grew normal intestinal organisms on three occasions. Histological examination of the rectal biopsy specimen showed gas-filled cystic spaces immediately beneath the muscularis mucosae. The cysts were lined by large macrophages with eosinophilic cytoplasm. Some of these macrophages were multinucleate. The connective tissue between the cysts showed a mild inflammatory infiltrate and the overlying mucosa was normal. These changes are characteristic of those described in pneumatosis coli (Morson and Dawson, 1973). The diagnosis was confirmed by examination of plain abdominal radiographs and barium enema films. Gas cysts were seen within the bowel wall extending from the lower rectum to the distal part of the transverse colon. The proximal colon appeared to be normal.

Because her symptoms were severe oxygen therapy was started in an attempt to avoid proctocolectomy. Initially, humidified oxygen was delivered at 8 l/min via a Mary Catterall (M.C.) mask,

but this produced an arterial oxygen tension (PaO_2) of only 26.40 kPa (198 mm Hg) which was below that recommended by Forgacs *et al.* (1973). The combination of 8 l/min of humidified oxygen via an M.C. mask and 4 l/min via nasal catheters produced a PaO_2 of 51.73 kPa (388 mm Hg). The recommended PaO_2 of about 40 kPa (300 mm Hg) was achieved with humidified oxygen 6 l/min via the M.C. mask together with 4 l/min via nasal catheters. The oxygen was administered continuously overnight followed by a one-hour break for toilet, washing, and breakfast. It was continued for at least four hours in the morning and after another break in the middle of the day she had a further four hours of oxygen in the afternoon. One further break was allowed in the evening. The patient suffered no untoward effects from this treatment, which was continued for six days. At sigmoidoscopy eight days after beginning the treatment the rectal mucosa was healthy but redundant where the cysts had deflated. A further rectal biopsy specimen taken from the same area as the first showed a mild mononuclear inflammatory infiltrate with a peppering of neutrophils and eosinophils, but no cysts were seen. The plain radiograph of the abdomen showed a marked improvement though there were possibly a few residual cysts in the sigmoid colon. The patient was discharged home asymptomatic.

When she was seen for review one month later her bowels were opening normally once a day and she had had no faecal incontinence. Radiological examination showed no evidence of any cysts and sigmoidoscopy showed normal rectal and colonic mucosae.

Comment

The term pneumatosis coli should be used when the colon alone is affected and pneumatosis cystoides intestinalis reserved for those cases that affect the small bowel. Most patients with pneumatosis coli are middle-aged and present with mucous diarrhoea and excessive flatus (Hughes *et al.*, 1966), which progressed to faecal incontinence in our patient. Because these are common symptoms of carcinoma of the rectum and other colonic diseases the correct diagnosis is only occasionally made in the first instance (Smith *et al.*, 1958). The plain abdominal radiograph is characteristic (Kenny, 1963) and the diagnosis can be confirmed by barium enema examination, sigmoidoscopy (Pemberton *et al.*, 1957), or colonoscopy (Wyatt, 1972; Varno and Bonanno, 1973).

In the past a "wait and see" approach has been adopted to manage patients with pneumatosis coli unless severe symptoms have necessitated surgical resection. In two major series 11 out of 27 (Smith *et al.*, 1958) and three out of eight patients (O'Reilly, 1973) required surgical resection, and in our patient proctocolectomy would have been necessary. In 1973, however, Forgacs *et al.* (1973) predicted that the cysts, which contain variable proportions of nitrogen, carbon dioxide, hydrogen, and oxygen, could be deflated if the total pressure of the gases in venous blood was lowered by prolonged breathing with a high concentration of oxygen. This alters the balance of diffusion of gases between the cyst and the surrounding tissues in favour of absorption and has now been used successfully to restore normal bowel function and contour in both our patient and the three reported by Forgacs *et al.* (1973). Oxygen therapy represents a major advance in the management of pneumatosis coli.

The most satisfactory method of administering the oxygen has not yet been worked out. We found that a satisfactory PaO_2 of about 40 kPa (300 mm Hg) could be achieved with 6 l/min of humidified oxygen via a standard M.C. mask together with 4 l/min via nasal catheters. The oxygen has to be given intermittently to guard against lung toxicity (Pratt, 1965) but should be continued without a break for periods of at least four hours to allow the PaO_2 to be maintained. As Leigh (1970) has shown that the PaO_2 is subject to individual variation with flow rates greater than 4 l/min via an M.C. mask we

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recommend that the PaO_2 should be checked about two hours after starting the first period of therapy and the flow rate adjusted accordingly. Follow-up of patients for up to two years has shown that they are still asymptomatic and free of disease (P. Forgacs, personal communication).

For many years the aetiology of pneumatosis coli has been a matter for debate. The introduction of oxygen as a successful form of therapy leads us to suggest that the cysts are created and maintained by a fastidious anaerobic gas-forming organism which produces gas at a rate which exceeds the rate of absorption until an equilibrium is reached. The high tissue PaO_2 achieved with oxygen therapy kills the organisms and the gas is then reabsorbed in the same way as gas contained within any natural or artificially created space in the body. Hence, the cysts do not recur. We are currently undertaking studies with the electron microscope in an attempt to substantiate this hypothesis.

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Acute Disseminated Encephalomyelitis in Typhoid Fever

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Encephalopathy with focal neurological signs and altered consciousness is a rare complication of typhoid fever and carries a high mortality (Roger and Gastaut, 1945; Pohowalla and Ghai, 1957; Zellweger and Idriss, 1960; Aita, 1972). The pathological changes in the brains of such patients, however, are poorly documented in the literature.

We report a case of symptomatic typhoid fever in which encephalopathy with clinical features of brain-stem involvement was found to be due to acute disseminated encephalomyelitis (perivenous demyelinating leucoencephalitis) of the midbrain and pons.

Case Report

A 25-year-old man was admitted to hospital with intermittent fever of one month's duration. A clinical diagnosis of typhoid fever was made. There was a good response to co-trimoxazole with disappearance of the toxæmia and abdominal signs. Widal's reaction with *Salmonella typhi* H gave a titre of 1/250, O-negative, and with *S. paratyphi* A-H 1/50. Blood culture was sterile.

On the 11th day the fever returned with mild toxæmia and confusion. The patient appeared ill and blood culture grew *S. typhi*. Widal's reaction remained unaltered. The fever and toxæmia subsided over the next 48 to 72 hours and there was clinical improvement. Next day, however, he rapidly lapsed into deep coma with fixed, semi-dilated pupils, tachycardia, hyperpnoea, quadriparesis, and spasticity of all four limbs with exaggerated reflexes and bilateral extensor plantar responses. There were intermittent spasms of extensor rigidity and clonic fits in the arms. A clinical diagnosis of typhoid encephalopathy was made and he was given dexamethasone, hydrocortisone, hypertonic dextrose, and intramuscular chloramphenicol. The ocular fundi were normal. The C.S.F. was clear and not under tension and contained sugar

3 mmol/l (54 mg/100 ml), protein 0.25 g/l, and chloride 1040 mmol (mEq) and had a cell count of 2×10^6 lymphocytes per l. C.S.F. culture was sterile and the Widal reaction remained unaltered. During the next seven hours he developed an axillary temperature of 38°C and his pupils became small and non-reacting. The tachycardia persisted and over the next few hours he ran a temperature of 41.1°C and had stertorous breathing. He died within 24 hours of the onset of the neurological manifestations.

Necropsy, five hours after death, showed congestion and superficial ulceration of the terminal 20 to 25 cm of the ileum with enlargement of the mesenteric lymph nodes. The spleen was not enlarged and the brain appeared normal. Histologically there was thinning of the mucosa of the terminal ileum with loss of villi and partial loss of glands. There was congestion of the submucosal vessels and cellular infiltration of the submucosa by lymphocytes. The mesenteric lymph nodes showed a marked histiocytic cellular proliferation in the subcapsular, cortical, and medullary sinuses with generalized congestion, as is characteristically seen in typhoid fever. In the brain the lesions were confined to the midbrain and pons. There was demyelination in an eccentric zone around the small vessels and microglial cells with pleomorphic nuclei surrounding the veins. Perivascular cuffing of small vessels by lymphocytes was seen. The appearance was of an acute disseminated encephalomyelitis with demyelination of the midbrain and pons (figs. 1 and 2).

FIG. 1—Histological section of brain showing dense eccentric infiltration by phagocytic cells to left of a venous channel. (H. and E. $\times 350$.)

Comment

The pathological process in the brain causing typhoid encephalopathy in this patient was an acute disseminated encephalomyelitis characteristically observed in viral infections such as chickenpox, mumps, and measles and after prophylactic

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