Case 4—A 56-year-old man with alcoholic cirrhosis of the liver had an oesophageal transection with the EEA staple gun for oesophageal varices. A day later a radiograph showed a complete staple ring and a barium swallow confirmed no oesophageal leakage. Further radiographs showed an enlarging break in the staple ring. He was discharged on the 14th day but was readmitted three days later with further bleeding, which was due to a varix at the site of the open staple ring.

Discussion

Two complete rings of full-thickness bowel in the cartridge indicates a satisfactory stapled anastomosis and in our experience has been associated with a complete staple "ring" observed by plain radiographs in the early postoperative phase. In these four cases, however, this did not ensure an uncomplicated recovery. In each case clinical complications were associated with late disruption of the circular staple ring seen on a plain radiograph. In the three colorectal anastomoses the site of leak corresponded with the area of staple dehiscence. It is interesting that two patients had rectal bleeding before the breaks in the staple rings were detected. This late complication has not been reported before and is unlikely to be caused by technical difficulties but might result from pelvic sepsis.

We emphasise that a complete staple ring in the early postoperative period does not necessarily imply that late dehiscence will not occur. Furthermore, if a break in the circular staple rings is seen on *x*-ray examination, even after an apparently successful anastomosis, the surgeon should expect complications.

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Small-bowel gangrene caused by Yersinia enterocolitica III

The most common symptoms of *Yersinia enterocolitica* infection are diarrhoea and fever, often with acute abdominal pain mimicking appendicitis. At operation mesenteric lymphadenitis, terminal ileitis, or catarrhal or suppurative appendicitis may be found. Occasionally enteritis persists for months, simulating a chronic inflammatory bowel disease. *Yersinia* may also cause intestinal ulceration, perforation, and peritonitis, sometimes with a fatal outcome.

We report a case of massive mesenteric lymphadenitis caused by *Yersinia enterocolitica* III, resulting in superior mesenteric vein thrombosis and gangrene of the small intestine.

Case report

A 27-year-old healthy man had a two-week history of fever and intermittent abdominal pain. On admission to Turku University Central Hospital he had a temperature of 37·3°C and generalised abdominal tenderness to deep palpation. Other findings, including rectal examination, were normal. Urine analysis, serum and urine amylases, serum glutamic-oxalacetic transaminases, serum alkaline phosphatase, and serum bilirubin concentrations were normal. The white blood cell count was 12×10^9/l (12 700/mm^3). After two days severe abdominal pain developed, the abdomen was protuberant, and bowel sounds were absent. The white blood cell count increased to 30×10^9/l (30 000/mm^3). Laparotomy revealed a litre of reddish serous fluid in the peritoneal cavity. The entire mesentery was massively thickened, mesenteric lymph nodes were enlarged, and 80 cm of the proximal part of the small bowel was necrotic and was resected, starting from the ligament of Treitz. All the veins in the mesentery of the resected part were thrombosed. The appendix and other abdominal organs were normal. After the operation intravenous ampicillin 2 g six hourly was started. The patient failed to improve. Operated on again the next day his entire small bowel was necrotic and only 15 cm of it could be spared at resection. Cultures of blood and stool, taken after the operation and initiation of antibiotic treatment, were sterile. Measurements of IgA, IgG, and IgM against *Yersinia enterocolitica* III were 35%, 65%, and 72% of a high positive standard serum.[1] Two weeks later they were 20%, 10%, and 25%, respectively. Six weeks' later they were absent. The findings strongly suggested recent *Yersinia* infection. All other results of bacterial and viral examinations were negative, including antibodies against salmonellae and brucellae and various autoantibodies. Lymphocyte responses to phytohaemagglutinin and concanavalin A were normal. After six months his general condition remained poor and he had severe nutrition problems.

The histological changes were identical in the specimens from the two operations. Haemorrhagic necrosis affected all the three layers of the jejunum. The mucosa was almost totally destroyed and the villi appeared as haemorrhagic "ghosts" with no viable epithelial cells. The deeper parts of the necrotic mucosa and the venous walls were infiltrated by neutrophils and mononuclear inflammatory cells. Most small veins and capillaries were thrombosed; the arteries were relatively intact. Sections from the first resection margins showed slight submucosal thrombophlebitis suggesting subsequent extension of the necrotising process.

Comment

Usually *Yersinia* infection is self-limiting and resolves without antibiotic treatment. Mortality is high only in cases with intestinal perforation or sepsis.[2–5] Mesenteric lymphadenitis is a typical lesion, but venous thrombosis caused by *Yersinia* has not been described. It is not clear whether thrombophlebitis or mechanical obstruction of the superior mesenteric vessels led to their thrombosis. It is also not clear whether early intensive antibiotic treatment would have prevented the extensive damage. This case shows that *Yersinia* may cause rapidly progressing serious infections with potentially lethal complications.

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Necrotising fasciitis due to Streptococcus pyogenes

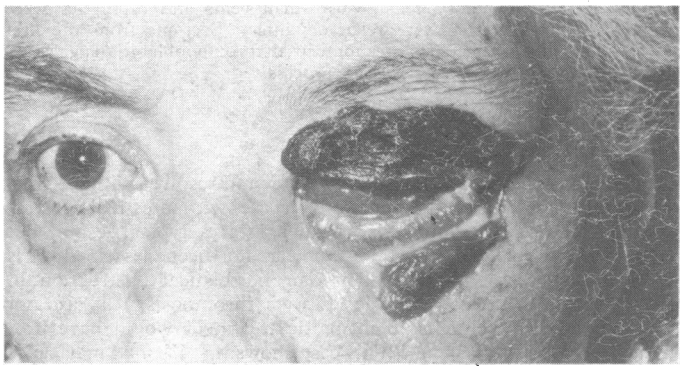
Three cases of *Streptococcus pyogenes* gangrene occurred over a period of two months. All patients survived with antibiotic treatment, although they later required reconstructive surgery. None had early surgical debridement.

Case reports

(1) A 45-year-old office worker, previously well, was admitted with severe right orbital cellulitis and glycosuria. He had no history of injury. An eye swab grew a pure, heavy growth of *S pyogenes* that was still present one week later after large doses of benzylpenicillin. At that time there was little resolution and both eyelids and surrounding tissues were swollen, purulent,

and discharging, but deep necrosis did not occur. There was much oedema in the right cheek extending to the neck. A large black scab formed over the upper lid while the lower lid became ulcerated and severely damaged, needing reconstruction.

(2) A 71-year-old woman, previously well, was admitted with severe cellulitis of the left face and left eyelids. A pure, heavy growth of *S pyogenes* was obtained from an eye swab and was present five days later. She developed severe left orbital cellulitis despite benzylpenicillin 20 megaunits daily. A pansystolic murmur of mitral incompetence occurred over the next week, despite an absence of growth in blood cultures, and she was considered to have acute streptococcal endocarditis. She developed a similar black scab on the upper lid with destruction of both upper and lower lid tissues and cicatrical ectropion (figure) requiring reconstruction.



Left orbital necrotising fasciitis due to *Streptococcus pyogenes*.

(3) A 41-year-old lorry driver was admitted febrile with an inflamed left elbow. He had no history of injury. He was treated with cephradine intravenously but within three days had developed blistering cellulitis, spreading up his arm, from which no bacteria were grown. He was treated additionally with erythromycin intravenously, but remained febrile. Eighteen days after admission a 2-in (5-cm) incision was made over his elbow to release 200 ml of pus. This yielded no bacterial growth. The wound was irrigated with Savlon (cetrimide and chlorhexidine). He remained febrile with necrotic material discharging from his wound. At a second operation after 16 days a vast loculated cavity was found, in which four corrugated drains were placed. Ten days later split skin grafts were placed over a large area of granulation tissue, which healed satisfactorily.

Comment

Necrotising fasciitis was described by Meleney in 1933 as a separate entity from erysipelas and synergistic gangrene.[1] The legs are usually affected and periorbital tissues[2] and elbows[3] are affected uncommonly. Surgical intervention with removal of all necrotic tissue is the treatment of choice.[1] Despite high doses of antibiotics our three patients still developed blistering and necrosis of the affected tissues. Debridement might have shortened the course of the disease but the decision is difficult when important tissues such as eyelids are affected.

All three patients had low antistreptolysin O titres (80-400) but high anti-desoxyribonuclease B titres (1200->9600), which is usual in skin infection with *S pyogenes*. All three had normal (<10 to 40) antibody titres to M-associated protein (MAP) type I.[4] High titres (>80) are observed in patients with rheumatic fever and uncomplicated infection due to certain opacity factor-negative serotypes associated with throat infection. There was a small rise in titre both to MAP II[4] (<10 to 20) and to the opacity factor (OF)[5] of M-type 75 (5 to 10) in the third patient, and a similar titre to M-type 75 OF was present in the second patient. M-type 75 streptococcus is often found in skin lesions in tropical countries but is uncommon in the UK, while anti-OF to M-type 75 occurs with low frequency (<3%) in the British population. These results confirm recent streptococcal infection in the third case and suggest that the patients had been infected with a "skin" strain. M-type 52 streptococcus, a "skin" strain, has been reported as the cause of one case of necrotising fasciitis.[3] It needs to be established whether this condition is due to "skin" rather than "throat" strains of *S pyogenes*.

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Pulmonary oedema precipitated by nifedipine

Nifedipine (Adalat), a calcium antagonist, is useful in the treatment of angina pectoris, particularly when beta-blockers are contraindicated, and in suspected cases of coronary artery spasm. The drug has negative inotropic effects and theoretically may precipitate cardiac failure, although this has not been reported.[1 2] We describe a case of acute pulmonary oedema precipitated by nifedipine.

Case report

A 71-year-old man with symptoms of severe angina pectoris and a clinical diagnosis of moderate aortic valve stenosis and probable coronary artery disease was admitted for cardiac catheterisation. He had no signs of congestive cardiac failure. His electrocardiogram showed controlled atrial fibrillation. His chest radiograph showed a cardiothoracic ratio of 55% and no pulmonary venous congestion. His medication on admission was digoxin 0·25 mg daily, Dyazide (hydrochlorothiazide and triamterene) 1 tablet daily, and perhexiline maleate 100 mg twice daily. Cardiac catheterisation showed severe aortic valve stenosis (peak systolic gradient = 124 mm Hg and aortic valve area = 0·4 cm^2). The mean pulmonary capillary wedge pressure was 32 mm Hg and there was no mitral valve disease. Selective coronary arteriography showed diffuse triple vessel disease. He was not accepted for cardiac surgery and an attempt was made to control his symptoms medically. He was discharged taking the same medication but isosorbide dinitrate replaced the perhexiline and the dose of Dyazide was doubled.

When seen a month later he complained of increasing angina. There were no alterations in his physical signs and his electrocardiogram and chest radiograph were unchanged. His raised left ventricular end diastolic pressure contraindicated beta-blockers and we thought nifedipine might benefit him. He took the first 10 mg capsule that evening. Half an hour later he had extreme dyspnoea, which lasted several hours. The next morning he took a second capsule, which precipitated acute pulmonary oedema. He was admitted to hospital in extremis with most florid clinical and radiographic features of pulmonary oedema and no alteration in his electrocardiogram. He was treated conventionally with oxygen, morphine, and frusemide. Intravenous calcium chloride was also given to counteract the calcium-antagonist effect of nifedipine. He made a steady recovery and was symptomatically improved on discharge several days later taking digoxin, frusemide, and isosorbide dinitrate.

Comment

The negative inotropic effect of nifedipine does not usually reduce cardiac output or provoke cardiac failure, because of its potent vasodilatory effect on resistance vessels.[3] The drug has been recommended in the treatment of acute pulmonary oedema in cases in which left ventricular after-load reduction is desirable.[4] As this case shows, in aortic stenosis impedance to left ventricular ejection is fixed