CASE REPORTS

Acute colorectal ischaemia after anaphylactoid shock

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Abstract

A 29 year old woman is reported with bloody diarrhoea three hours after developing anaphylactoid shock. Sigmoidoscopy, barium enema, and histology showed rectal and colonic ischaemia to the splenic flexure. Recovery was complete. There was no history of vascular disease but the patient was taking an oral contraceptive. Thirty one other cases of spontaneous ischaemic proctitis are reviewed.

Rectal ischaemia has been described as 'so rare as to be clinically unimportant.'[1] The rectum is effectively an arterial sanctuary protected by communications between the inferior mesenteric, internal iliac, and internal pudendal arteries.[2] Spontaneous rectal ischaemia has been reported in 31 patients,[3-20] of whom the youngest was 36 years old, with widespread vascular disease in 81%. Ischaemic colitis, on the other hand, is common,[21 22] often affecting the region of the splenic flexure at the watershed of the middle and left colic arteries. It remains, however, extremely uncommon under the age of 30.[23] We describe a patient aged 29 years with no sign of cardiovascular disease who developed rectal and colonic ischaemia as a result of hypotension from anaphylactoid shock.

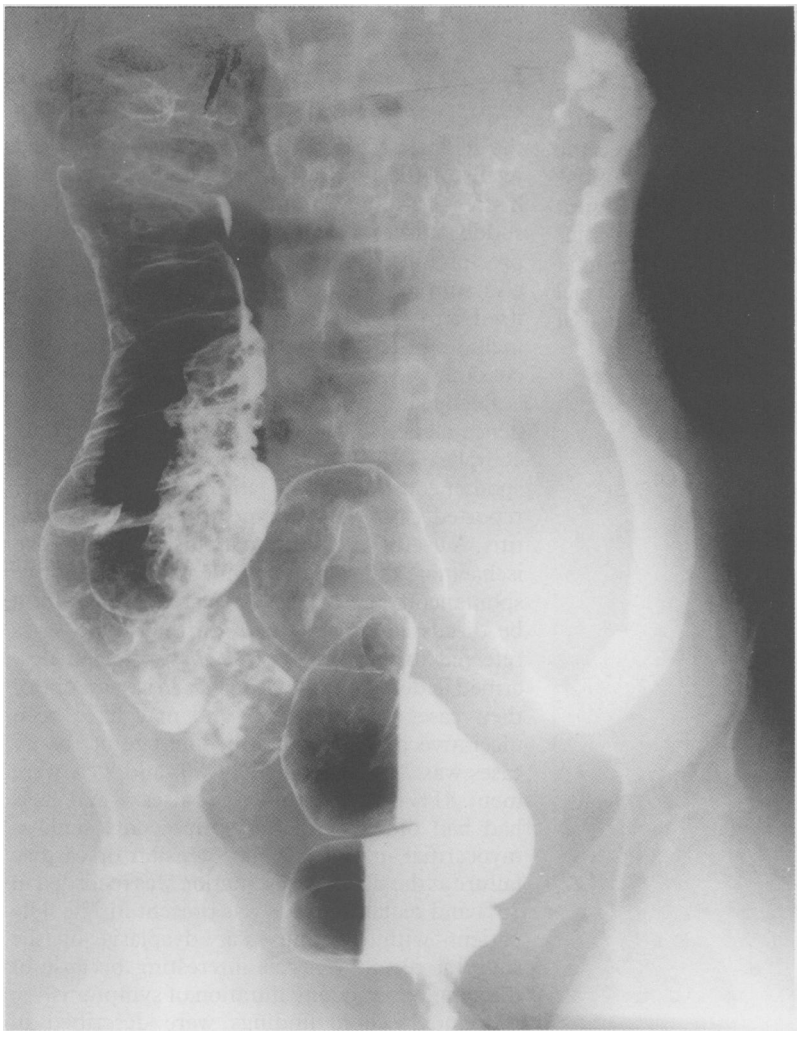

Figure 1: Barium enema 36 hours after admission. This picture shows thumb printing up to the splenic flexure but not the mucosal ulcers extending down to 7 cm from the anal margin.

Case report

A 29 year old housewife ate a cheddar cheese sandwich before going to bed. Forty five minutes later she awoke itching, with swelling of the lips and a blotchy rash over the whole body. She then experienced generalised abdominal discomfort and fainted three times before arriving in hospital two hours after the onset of symptoms. She had previously been well apart from an uncertain history of penicillin allergy. She had taken Microgynon 30 (0·03 mg ethinyl oestradiol, 0·15 mg levonorgestrel) for 20 months and was a non-smoker. Nobody else in the family had eaten the cheese which had been bought, prewrapped, from a major supermarket 48 hours before.

The blood pressure was unrecordable on admission. She was conscious but disoriented. There was a widespread erythematous rash, labial oedema, and lower abdominal tenderness. There was no stridor or wheeze (peak flow 420 lpm) and no tampon in situ. Adrenaline (1 ml of 1 in 1000) was given intramuscularly with 200 mg intravenous hydrocortisone and 10 mg chlorpheniramine; 1000 ml polygeline was infused rapidly before a systolic pressure of 90 mmHg was recorded 45 minutes after admission. Her normal blood pressure (130/70 mmHg) returned 30 minutes later, so the maximum duration of hypotension could have been 3 hours 15 minutes but was probably substantially shorter.

Rectal bleeding began within three hours of the onset of symptoms and lasted for 30 hours. The haemoglobin fell from 162 g/l to 125 g/l without transfusion. The white cell count was $16{\cdot}3\times10^9$/l with a normal platelet count and clotting studies and no detectable fibrinogen degradation products. Sigmoidoscopy on admis-

Spontaneous ischaemic proctitis: reported cases of rectal ischaemia

Age	Sex	Site	Predisposing factors	Duration*	Outcome	Reference
29	F	R-D	Anaphylactoid shock, oral contraceptive	3 h	Recovery	Current
78	M	A-S	Diabetes, hypertension	30 min	Recovery	4
52	F	R-C	Rheumatoid arthritis	10 days	Colectomy	5
71	F	R-S	Rectal prolapse, fibromuscular dysplasia	4½ yr	Stricture	6
77	M	R	Myocardial infarction, cardiovascular accident	-	Colectomy	7
71	M	R-D	Aortic aneurysm	2 days	Colectomy	8
67	M	R-S	Cardiovascular accident, diabetes	-	Died	9
55	M	A-S	Rectal prolapse	4 days	Stricture	10
74	F	R	Diabetes, myocardial infarction	1 day	Recovery	11
57	M	R	Vascular disease	3 wk	Stricture	12
62	M	A-R	Aortoiliac atheroma	5 wk	Recovery	13
67	M	A-T	Aortoiliac atheroma	5 days	Stricture	13
74	F	R-S	Diabetes, myocardial infarction, cardiac failure	2 days	Recovery	13
68	M	R	Myocardial infarction, cardiac failure	4 wk	Recovery	13
74	M	R-S	Cardiovascular accident, aortic atheroma	-	Recovery	13
62	M	R-S	Hypertension	1 day	Recovery	13
74	M	R-T	Myocardial infarction, aortic aneurysm	2 days	Died	13
71	M	R-C	Myocardial infarction, cardiac failure	2 mth	Died	14
77	M	R-S	Aortic atheroma	2 days	Died	14
80	F	R-D	Aortic atheroma, septic shock	2 days	Died	14
40	F	R	Ruptured ectopic pregnancy	6 days	Stricture	15
72	M	A-T	Dissecting aortic aneurysm	-	Died	16
75	M	R-D	Aortic aneurysm, popliteal embolus	1 day	Stricture	17
75	M	R-S	Aortic atheroma	-	Recovery	18
72	M	R-D	Vascular disease	3 days	Died	19
73	M	R-D	Aortic aneurysm	-	Died	19
36	M	R	Severe hypertension	6 mth	Died	20

A=anus; R=rectum; S=sigmoid; D=descending colon; T=transverse colon; C=caecum.
*Duration of symptoms at presentation.

sion showed altered blood coming from beyond the rectosigmoid junction with an apparently normal rectal mucosa. A plain abdominal film showed a thickened bowel wall with thumb printing in the descending colon. A barium enema, 36 hours after admission, confirmed the characteristic appearances of ischaemic colitis[24] (Fig 1) extending to the splenic flexure. Repeat rigid and flexible sigmoidoscopy four days after admission showed linear ulcers in the rectum at 7 cm, separated by macroscopically normal mucosa. These ulcers, covered by mucosal slough, became confluent at 12 cm and extended beyond 30 cm. A biopsy specimen taken at this time showed necrotic mucosal glands, occlusion of some capillaries with proteinaceous material, some viable glands, and polymorphs on the surface (Fig 2). Three weeks later the ulcers were healing and a rectal biopsy showed a light neutrophil infiltrate with occasional dilated and distorted glands with fibrosis (Fig 3). Colonoscopy at eight weeks was macroscopically normal, although mild inflammatory changes persisted in the most distal biopsy specimens. After 20 weeks the rectal mucosa was macroscopically and microscopically normal. The histological interpretation was acute ischaemic necrosis with healing changes thereafter, leading to complete resolution.

Once the bleeding settled the patient made an uneventful recovery. Stool and blood cultures grew no pathogens. Other results included normal concentrations of complement and negative tests for C1 esterase inhibitor. No antinuclear or anticardiolipin antibodies were found. The cheese was sent for microbological and chemical analysis but no pathogens were grown and a histamine content of 0·9 mg/100 g was not excessive. The colonic vasculature was not investigated because it was considered unethical to perform contrast studies after the complete recovery from a presentation with anaphylaxis and Doppler studies of the inferior mesenteric artery were technically impossible.

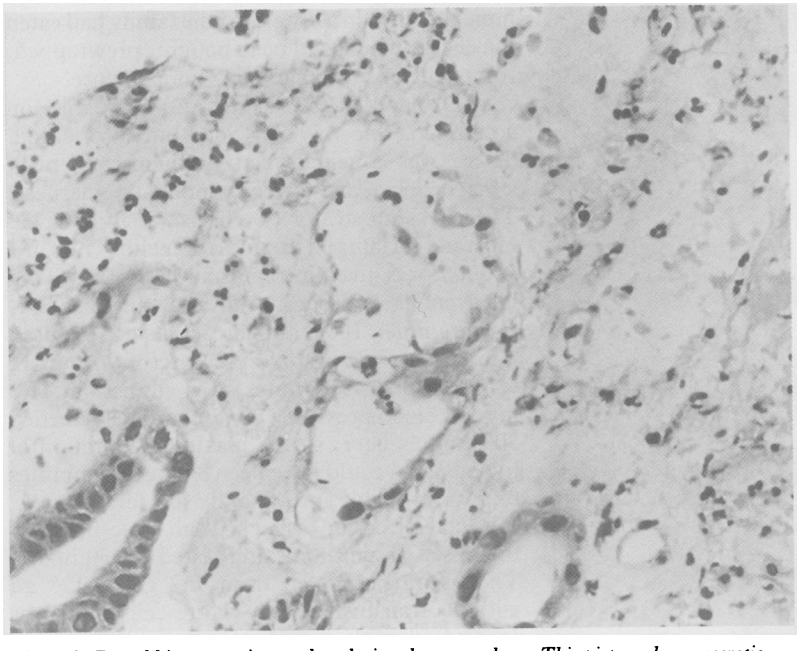

Figure 2: Rectal biopsy specimen taken during the acute phase. This picture shows necrotic large bowel mucosa with an acute inflammatory exudate on the surface. The deep portion of the mucosa shows viable epithelium. (Haematoxylin and eosin, original magnification ×400.)

Discussion

Spontaneous rectal ischaemia is so unusual that the diagnosis was only considered established as a result of the typical barium, endoscopic,[25] and histological[26] appearances of ischaemia. The sudden onset with hypotension as an identifiable precipitating factor and subsequent resolution also support the diagnosis. Ischaemic proctitis itself and the aetiological factors in this case, including the oral contraceptive pill, merit discussion.

Rectal and colonic ischaemia differ in incidence alone. Rectal ischaemia more commonly complicates aortoiliac surgery[3 27] than occurs spontaneously. The table summarises 27 reported cases of spontaneous ischaemic proctitis. A useful study of 16 patients with colorectal ischaemia[3] did not discriminate between five spontaneous and 11 surgery related cases and will be discussed separately. A further three cases referred to by Kilpatrick *et al*[13] were not described in detail but the diagnosis in at least one of their cases was debatable and may have been ulcerative proctitis. The median age of the 27 cases was 71 years (range 29–80) and 74% were men; 81% had atheromatous disease and 33% had had other focal ischaemic events such as myocardial infarction. Hypotension or cardiac failure at the time of presentation was recorded in 26% and rectal prolapse was present in 7%. The patient with fibromuscular dysplasia of the superior rectal artery[6] is interesting because of the aetiology and long duration of symptoms.

Sigmoidoscopic findings were described in detail in 12 patients; 50% had discrete linear ulcers, 42% had punctate ulcers, and one had a

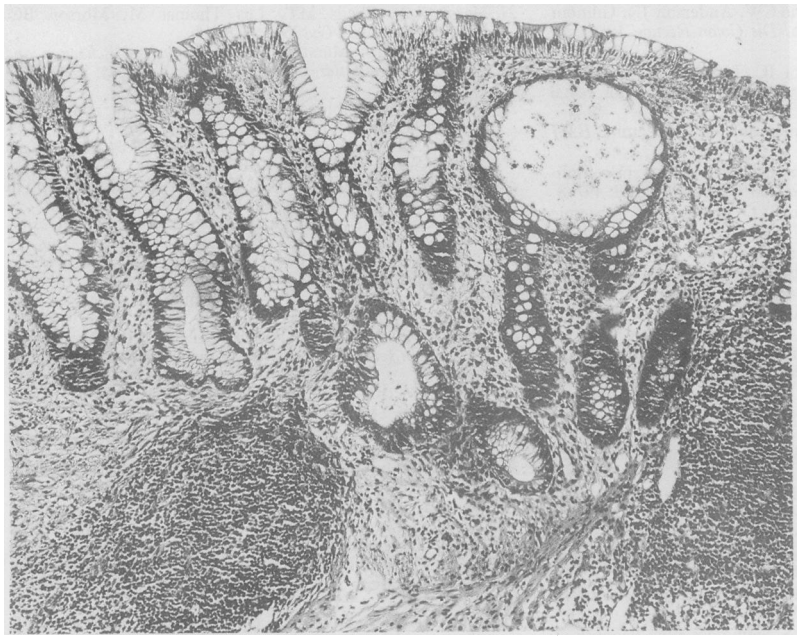

Figure 3: Rectal biopsy specimen taken three weeks after the illness. This picture shows intact large bowel mucosa with a dilated gland. Only scanty acute inflammatory cells remain. A subsequent biopsy specimen was normal. (Haematoxylin and eosin, original magnification ×250.)

solitary ulcer. The appearances may be confused either with Crohn's disease or with ulcerative colitis if the ulcers are confluent. Pseudopolyps were seen in 58% and mucosal friability was almost universal. Rectal gangrene occurred in 19% and 74% had associated ischaemic colitis. Histological features were reported too infrequently to compare cases, but haemosiderin laden macrophages were notably absent in several chronic cases[6 13] and may be less characteristic than previously described.[21 26] The histological features of advanced pseudomembranous colitis are similar to acute ischaemia, but they may be distinguished in the early stages.[28] The overall mortality was 33%, although this includes three patients from an early necropsy study,[14] so the mortality in clinical practice is probably lower. A stricture developed in 22% and 33% recovered spontaneously without surgery. The prognosis is the reason for distinguishing between spontaneous and surgery related ischaemic proctitis which is well illustrated by considering the 16 patients described by Boot *et al*[3]: 11 of their patients were postoperative and the overall mortality was 63%. This probably reflects a greater incidence of hypotension as well as other complications of aortoiliac surgery in unfit patients.[29]

In our patient the cause of the hypotension was attributed to an anaphylactoid reaction to cheese. The term anaphylactoid is used in preference to anaphylactic because an immune basis appears unlikely.[30] Cheese was implicated because eating a cheese sandwich was the only event that preceded the catastrophic reaction. There was, however, no bacteriological or chemical evidence of contamination nor any history to suggest cows' milk protein allergy. Toxin mediated hypotension was considered, especially in view of the absence of bronchospasm, but again no evidence could be found. The alternative hypothesis that the labial oedema, rash, and hypotension were due to vasoactive peptides released by spontaneous colorectal ischaemia is implausible because of the temporal sequence of events.

Several factors could have contributed to the ischaemic damage. If damage was due to hypotension alone then it is difficult to understand why only the left side of the colon and rectum were involved and other organs were spared. Experimental studies in dogs showed that hypovolaemia alone did not produce ischaemic colitis without arterial obstruction.[31] Hypotensive ischaemic colitis seems to follow a different pattern to that described by Marston *et al*.[21] The right side of the colon was affected alone in 68% of 19 cases[32] attributed to hypotension. Although the evidence for hypotension was circumstantial in some of these patients, in six other cases associated with haemorrhagic shock[33] there were no cases of purely left sided ischaemic damage. Other factors that could have contributed to the damage in our patient are an anatomical anomaly, intestinal involvement in the anaphylactoid process, vasospasm, and venous thrombosis. An anatomical anomaly can not be excluded. The histological appearances did not support direct anaphylactoid intestinal damage, although the biopsies were carried out four days after the injury. Vasospasm is likely to have occurred while the cardiac output was redistributed to other vital organs but why the distal colon and not the entire mesenteric bed should be affected is unclear. The final common pathway of ischaemic mucosal damage is almost certainly through the generation of free radicals in an ischaemic-reperfusion injury,[34] but this does not explain localised left sided damage without associated arterial insufficiency.

The contribution of the oral contraceptive pill is worth considering. Several cases of ischaemic colitis related to the pill have been described[35–37] but the evidence remains one of association. The mechanism is uncertain. It is possible to produce venous lesions in the colon which resemble ischaemic colitis radiologically and histologically but which differ macroscopically,[38] but venous thrombosis still does not readily explain rectal involvement. Even so, our advice was to stop the pill. The rapid onset of bleeding and superficial ulceration tend to favour arterial ischaemia in our patient.

Colorectal ischaemia remains an uncommon cause of rectal bleeding in young people. The clinical, macroscopic, and histological features of ischaemic proctitis are the same as ischaemic colitis, apart from the site of disease. Spontaneous ischaemia seems to have a better prognosis than ischaemic proctitis related to surgery. Both hypotension and the oral contraceptive pill should be considered risk factors in the young.

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1 Marston A. Ischaemia of the gut. In: Misiewicz JJ, Pounder RE, Venables CW, eds. *Diseases of the gut and pancreas.* Oxford: Blackwells, 1987: 1029–43.
2 Church JM, Raudkivi PJ, Hill GL. The surgical anatomy of the rectum – a review with particular relevance to the hazards of rectal mobilisation. *Int J Colorectal Dis* 1987; **2**: 158–66.
3 Boot H, Rauwerda JA, Hoitsma HFW, Bosma A, Meuwissen GM. Ischaemic colitis with rectal involvement: clinical features, endoscopic assessment and management. *European Journal of Gastroenterology and Hepatology* 1989; **1**: 69–72.
4 Andersson R, Uvelius B, Willen R. Transient ischemic colitis. *Acta Chir Scand* 1987; **153**: 151–3.

5 Welch GH, Shearer MG, Imrie CW, Anderson JR, Gilmour DG. Total colonic ischemia. *Dis Colon Rectum* 1986; **29**: 410–2.

6 Quirke P, Campbell I, Talbot IC. Ischaemic proctitis and adventitial fibromuscular dysplasia of the superior rectal artery. *Br J Surg* 1984; **71**: 33–8.

7 Weaver M. Atherosclerotic infarction of the rectum. *BMJ* 1984; **288**: 684.

8 Stenberg B, Risberg B. Abdominal aortic aneurysm complicated by rectal necrosis. *Acta Chir Scand* 1984; **150**: 497–8.

9 Hagihara PF, Parker JC, Griffen WO. Spontaneous ischaemic colitis. *Dis Colon Rectum* 1977; **20**: 236–51.

10 Kawarda Y, Satinsky S, Matsumoto T. Ischemic colitis following rectal prolapse. *Surgery* 1974; **76**: 340–3.

11 Parks TG, Johnston GW, Kennedy TL, Gough AD. Spontaneous ischaemic proctocolitis. *Scand J Gastroenterol* 1972; **7**: 241–6.

12 Ganchrow MI, Clark JF, Ferguson JA. Ischemic proctitis with obliterative vascular change. *Dis Colon Rectum* 1970; **13**: 470–4.

13 Kilpatrick ZM, Farman J, Yesner R, Spiro HM. Ischemic proctitis. *JAMA* 1968; **205**: 74–80.

14 McGovern VJ, Goulston SJM. Ischaemic enterocolitis. *Gut* 1965; **6**: 213–20.

15 Wald M. Gangrene of the distal two thirds of transverse colon, left colon, rectum and anal canal due to superior mesenteric vascular insufficiency. *Dis Colon Rectum* 1964; **7**: 303–5.

16 Curr JF. Rectal stricture due to ischaemia following ruptured ectopic gestation. *Gut* 1967; **8**: 178–9.

17 Shanahan MX, Steedman PK. Inferior mesenteric artery occlusion. *Br J Surg* 1963; **50**: 533–4.

18 Littman L, Boley SJ, Schwartz S. Sigmoidoscopic diagnosis of reversible vascular occlusion of the colon. *Dis Colon Rectum* 1963; **6**: 142–6.

19 Carter R, Vannix R, Hinshaw DB, Stafford CE. Inferior mesenteric vascular occlusion: sigmoidoscopic diagnosis. *Surgery* 1959; **46**: 845–6.

20 Shearburn EW, Connell J, Hopkins JE. Rectal ulceration due to ischemic necrosis. *Ann Surg* 1955; **141**: 563–6.

21 Marston A, Pheils MT, Lea Thomas M, Morson BC. Ischaemic colitis. *Gut* 1966; **7**: 1–15.

22 Marston A. In: Marston A, ed. *Splanchnic ischaemia and multiple organ failure.* London: Edward Arnold, 1989: 300–21.

23 Duffy TJ. Reversible ischaemic colitis in young adults. *Br J Surg* 1981; **68**: 34–7.

24 Thomas ML. Radiology of ischaemic colitis. *Proceedings of the Royal Society of Medicine* 1968; **61**: 341–2.

25 Scowcroft CW, Sanowski RA, Kozarek RA. Colonoscopy in ischaemic colitis. *Gastrointest Endosc* 1981; **27**: 156–61.

26 Morson BC. The pathology of ischaemic colitis. *Clin Gastroenterol* 1972; **1**: 745–63.

27 Nelson RL, Schuler JJ. Ischemic proctitis. *Surg Gynecol Obstet* 1982; **154**: 27–33.

28 Price AB, Davies DR. Pseudomembranous colitis. *J Clin Pathol* 1977; **30**: 1–12.

29 Travis SPL. Prognosis and management of rectal ischaemia. *European Journal of Gastroenterology and Hepatology* 1990; **2**: 163.

30 Fisher MM, Baldo BA. Acute anaphylactic reactions. *Med J Aust* 1988; **149**: 34–8.

31 Mathews JGW, Parks TG. Ischaemic colitis in the experimental animal. *Gut* 1976; **17**: 677–84.

32 Sakai L, Keltner R, Kaminski D. Spontaneous and shock-associated ischemic colitis. *Am J Surg* 1980; **140**: 755–60.

33 Byrd RL. Cunningham MW, Goldman LI. Nonocclusive ischaemic colitis secondary to haemorrhagic shock. *Dis Colon Rectum* 1987; **30**: 116–8.

34 Haglund U, Bulkley GB, Granger DN. On the pathophysiology of intestinal ischemic injury. *Acta Chir Scand* 1987; **153**: 321–4.

35 Kilpatrick ZM, Silverman JF, Betancourt E, Farman J, Lawson JP. Vascular occlusion of the colon and oral contraceptives. *N Engl J Med* 1968; **278**: 438–40.

36 Cotton PB, Thomas ML. Ischaemic colitis and the contraceptive pill. *BMJ* 1971; iii: 27–8.

37 Heer M, Repond F, Hany A, *et al.* Acute ischaemic colitis in a female long distance runner. *Gut* 1987; **28**: 896–9.

38 Marcuson RW, Stewart JO, Marston A. Experimental venous lesions of the colon. *Gut* 1972; **13**: 1–7.