Short reports

Late stage congestive gastropathy

Ornella Leone, Magda Zanelli, Simonetta Piana, Donatella Santini, Domenico Marrano

Abstract

Gastric mucosal abnormalities resulting from portal hypertension are defined as "congestive gastropathy". A case of congestive gastropathy with unusual features, in a 63 year old man with a history of excessive alcohol intake and cirrhosis, is described. The patient underwent a subtotal gastrectomy because of profuse bleeding from a gastric ulcer, providing a large surgical specimen for examination. Unusual gross and histological findings included prominent arterial intimal hyperplasia, and diffuse duplication and focal fragmentation of the internal lamina elastica. The differential diagnosis of this condition includes primary angiodysplastic gastropathy such as Dieulafoy's disease. The similarity with Dieulafoy-like angiodysplasia emphasises that clear cut criteria to define gastric vascular lesions do not yet exist.

(J Clin Pathol 1997;50:350-358)

Keywords: congestive gastropathy; cirrhosis; primary angiodysplasia

Congestive gastropathy and mucosal vasculopathy¹ are the terms currently used to define gastric mucosal abnormalities occurring in portal hypertension. The endoscopic appearance² of this condition has been widely studied and the macroscopic mucosal changes have been categorised as fine pink speckling, superficial reddening, and, the most common, a mosaic or snake-skin pattern. The histological hallmark³ related to these endoscopic aspects is a prominent vascular ectasia in the lamina propria with an almost complete lack of inflammatory cells. In addition, submucosal extensive oedema, thickened arterioles and veins showing features of arterialisations have been described.⁴

To the best of our knowledge, reports of the morphological patterns of vascular abnormalities in portal hypertension have been described only in gastric biopsies. Despite the original study of Moore *et al*⁵ in 1976, further modified by Lewi⁶ and Fowler,⁷ the classification of gastrointestinal vascular malformations and the related morphological findings remain unclear. Recent interest has focused on angiodysplasia,⁸ a still undefined condition called vascular ecta-

sia, arteriovenous malformation, haemangioma or angioma.⁹

We report a case of congestive gastropathy in a patient with portal hypertension focusing on the histopathological aspects of a long standing disease.

Case report

A 63 year old man, with a long history of excessive alcohol intake and with symptomatic cirrhosis, was admitted urgently to our institution suffering from haematemesis and melaena. At admission, his haemoglobin concentration was 69 g/l, red blood cell count $2200 \times 10^9/l$, packed cell volume 0.2, mean cell volume $91.3 \mu^3$, white blood cell count $8.82 \times 10^9/l$, and platelet count 68 000 per litre. The gastroscopy, urgently performed, disclosed an ulcer (1.5 cm in diameter) on the posterior wall of the stomach. After three days, owing to a second episode of haematemesis, the patient was transferred urgently to the surgical department. A further gastroscopy showed an active, profuse bleeding from the gastric ulcer prompting a subtotal gastrectomy.

Figure 1 Necrosis of the superficial and glandular epithelium and haemorrhagic effusion in the lamina propria, related to ischaemic damage.

Institute of Anatomic Pathology, University of Bologna, Ospedale Sant' Orsola, Via Massarente 9, 40138 Bologna, Italy
O Leone
M Zanelli
S Piana
D Santini

Institute of Surgery I
D Marrano

Correspondence to:
Dr Ornella Leone.

Accepted for publication
7 May 1996

Figure 2 Artery showing intimal hyperplasia and aspects of vascular neoformation.

PATHOLOGICAL FINDINGS

The stomach measured 14.5 cm along the small curvature and 23 cm along the great curvature. The mucosa was brownish and widely haemorrhagic. On the posterior wall, in the distal part of the gastric body, there were four ulcers, ranging in diameter from 1.9 cm to 0.8 cm. One of the ulcers was sutured. Close to the proximal resection margin, on the anterior wall, there was an area (3.5 cm in diameter) of greyish mucosa and haemorrhagic speckles. All of the ulcers along the above mentioned area were sampled.

The specimens were fixed in 10% buffered formalin and paraffin embedded; 2 µm sections were stained with haematoxylin and eosin, van Gieson, and Masson's trichrome methods.

Microscopic examination confirmed the presence of multiple acute ulcers; the surrounding mucosa showed features consistent with ischaemic damage, such as necrosis of the superficial and glandular epithelium, and haemorrhagic effusion in the lamina propria (fig 1). The remaining mucosa showed chronic,

focally atrophic, gastritis with diffuse intestinal metaplasia and foveolar hyperplasia.

The submucosa showed marked oedema and vascular anomalies involving both the arteries and veins.

The arteries were enlarged and showed different stages of intimal hyperplasia up to a complete obliteration of the lumen with aspects of vascular neoformation (fig 2); the internal lamina elastica was diffusely reduplicated and focally fragmented. The parietal muscle layer was hypertrophic and the fibres appeared intermingled with fibrous tissue (fig 3).

The veins were also enlarged; some showed a circumscribed thickening with non-concentric muscularisation (fig 4), and others, in absence of muscularisation, had a marked derangement of the structure, with fragmentation of the elastic fibres. The more the elastic fibres were dissociated, the less the muscularisation was prominent. All of these vascular abnormalities were present throughout the stomach, but more prominent in the ulcerated areas. Finally, the gastric muscle coat appeared somewhat reduced and focally interrupted by connective tissue.

Discussion

To date, the term "congestive gastropathy" has been used to identify a clinicopathological entity related to portal hypertension. On endoscopy, the features consistent with congestive gastropathy are quite well known and usually described as "mosaic pattern" or "snake-skin pattern". The histopathological findings, such as vascular ectasia in the lamina propria, submucosal oedema, thickened arterial wall, and arterialisation of the veins, have also been described, but only on bioptic fragments and in a relatively early stage of portal hypertension.

In our case, owing to the evaluation of a surgical specimen, we have been able to identify and describe the prominent parietal wall vascular abnormalities occurring in a long standing portal hypertension. Both veins and arteries were involved. The diffuse and conspicuous venous muscularisation is in keeping with a long standing portal hypertension. In the arteries, the prominent intimal hyperplasia with scattered oedema could be the expression

Figure 3 Fibrous tissue within the parietal muscle layer.

Figure 4 Vein showing non-concentric muscularisation.

of the arterial involvement secondary to a long standing venous congestion, actually representing a more advanced stage of the disease. Indeed, the presence of fibrous tissue within the gastric muscle coat is likely to represent the dystrophic effect due to the vascular abnormalities.

The acute ulcers and the superficial mucosal necrosis could be an expression of either alcohol dependent toxicity or ischaemic damage secondary to the prominent vascular alterations. Features in keeping with the latter pathogenesis are mucosal atrophy, submucosa fibrosis, and the presence of fibrous tissue within the gastric muscle.

In addition, we noted the similarity of some arterial abnormalities—intimal hyperplasia up to total lumen obliteration, diffuse duplication, and focal fragmentation of the internal lamina elastica—with the arterial anomalies described in primary angiodysplasia such as Dieulafoy's disease.¹⁰ Nevertheless, in the present case the clinical presentation in a cirrhotic patient with portal hypertension was not in keeping with the hypothesis of a Dieulafoy-like angiodysplasia that usually occurs in middle aged and elderly men without any relevant family history, excessive alcohol or aspirin intake. In addition, in Dieulafoy's disease the mucosal ulcer, localised in the upper stomach, is usually single, small, and shallow, whereas in our case the ulcers were multiple and deep penetrating. Finally, the prominent muscularisation of the veins was

the main histological feature supporting a late stage congestive gastropathy rather than an angiodysplasia.

In conclusion, our study focuses on the large spectrum of microscopical vascular alterations present in a late stage congestive gastropathy, of which, to the best of our knowledge, this is the first description. The similarity with Dieulafoy-like angiodysplasia emphasises that clear cut criteria to define gastric vascular lesions do not exist yet.

- 1 Sarfeth IJ, Tarnawski A, Hajduczek J, Stachura J, Bui HX. Gastric mucosal vasculopathy in portal hypertension. *Gastroenterology* 1987;92:1129–31.
- 2 Sarin SK, Misra SP, Singal A, Thorat V, Broor SL. Congestive gastropathy in portal hypertension: variations in prevalence. *Am J Gastroenterol* 1988;83:1235–9.
- 3 Quintero E, Pique JM, Bombi JA, Bordas JM, Sentis J, Montserrat E, et al. Gastric mucosal vascular ectasias causing bleeding in cirrhosis. *Gastroenterology* 1987;93:1054–61.
- 4 Triger DR, Hosking SW. The gastric mucosa in portal hypertension. *J Hepatol* 1989;8:267–72.
- 5 Moore JD, Thompson NW, Appelman HD, Foley D. Arteriovenous malformations of the gastrointestinal tract. *Arch Surg* 1976;111:381–9.
- 6 Lewi HJE, Gledhill T, Gilmour HM, Buist TAS. Arteriovenous malformations of the intestine. *Surg Gynecol Obstet* 1979;149:712–16.
- 7 Fowler DL, Fortin D, Wood WG, Pinkerton JAJr, Koontz PG Jr. Intestinal malformations. *Surgery* 1979;86:377–85.
- 8 Scully R, Mark EJ, Mc Neely WF. Case records of the Massachusetts General Hospital. *N Engl J Med* 1991;10:1086–96.
- 9 Tivadar L M, Vilmos AT. The caliber persistent artery of the stomach: a unifying approach to gastric aneurysm, Dieulafoy's lesion, and submucosal arterial malformation. *Hum Pathol* 1988;19:914–21.
- 10 Leone O, Zanelli M, Santini D, Minni F, Marrano D. Dieulafoy's disease associated with early gastric cancer. *J Clin Pathol* 1995;48:267–70.

Inflammatory pseudotumour of the liver

G Kafeel, P U Telesinghe

Abstract

Inflammatory pseudotumour is not a common lesion. The first series of 12 cases was described in 1986, to which 37 more cases have now been added. The histology, differential diagnosis, and prognosis of this lesion have been described in detail, but the aetiology is unknown and the mode of treatment remains controversial. A new case is presented and compared with the previously reported cases. Fine needle aspirate yielded a growth of klebsiella organisms. The possibility of this infection as an aetiological agent is considered.

(*J Clin Pathol* 1997;50:352–353)

Keywords: inflammatory pseudotumour; liver; klebsiella

Case report

A 70 year old female was referred to RIPAS Hospital, Brunei with a history of intermittent low grade fever, progressive weight loss and weakness, and vague abdominal pain on the

right side. She looked sick and weighed only 32 kg. Her body temperature was raised at 38.4°C. A tender mass was felt in right iliac fossa. Ultrasonography of the abdomen showed a necrotic mass of 4.0 cm diameter on the inferolateral aspect of the right lobe of the liver displacing the hepatic flexure downwards. Except for a raised erythrocyte sedimentation rate (ESR) of 30 mm in the first hour the rest of her haematological and biochemical profile, including the liver function tests, was within the normal range. α -Fetoprotein (AFP) and carcinoembryonic antigen (CEA) were not raised.

Ultrasound guided fine needle aspiration of the mass yielded about 2 ml of turbid fluid which showed abundant neutrophils, fibrin, and few degenerating liver cells on a necrotic background. No malignant cells, fungal elements, parasites, or acid-fast bacilli could be seen. *Klebsiella* spp were cultured from the aspirated material. The lesion was considered to be inflammatory. After six weeks of treat-

Department of
Pathology, State
Laboratory, RIPAS
Hospital, BSB,
Darussalam, Brunei
G Kafeel
P U Telesinghe

Correspondence to:
Dr G Kafeel.

Accepted for publication
10 December 1996