Case Report

Watermelon stomach. An unusual cause of recurrent upper GI tract bleeding in the uraemic patient: efficient treatment with oestrogen–progesterone therapy

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Introduction

The gastrointestinal tract is frequently the source of occult blood loss in patients on long-term haemodialysis. Among the many causes of bleeding, angiodysplastic lesions are encountered more frequently in uraemic than in non-uraemic patients [1–5].

We report a patient on chronic haemodialysis who developed a rare form of angiodysplastic lesions localized in the stomach. Its unique appearance on endoscopy has justified the name of watermelon stomach (WS) [6]. Its recognition is of interest as it responds well to an oestrogen-progestative treatment with eventual disappearance of repeated bleeding episodes.

Case report

A 63-year-old man on chronic haemodialysis was admitted to our hospital in June 1994 for severe persistent anaemia.

Haemodialysis had been initiated in 1988 for end-stage renal failure due to chronic glomerulonephritis. The patient had a long history of severe hypertension and suffered from peripheral vascular disease. Soon after the onset of dialysis, he developed severe anaemia resistant to the prescription of iron supplements and erythropoietin and requiring repeated blood transfusions (Figure 1). Upper GI endoscopy disclosed, on several occasions, antritis despite the administration of ranitidine (300 mg o.d.) and subsequently omeprazole (20 mg o.d.). In November 1991, antral vascular ectasia typical of WS aspect were diagnosed (Figure 2). Repeated courses of omeprazole (40 mg o.d.) and sucralfate (1 g t.d.s.) did not prevent further digestive blood loss and anaemia (Figure 1). An extensive work-up in April 1994 including gastroscopy, barium enema, rectoscopy, and sigmoidoscopy confirmed WS and failed to identify another gastrointestinal lesion.

On clinical examination, blood pressure was 160/90 mmHg. A murmur was present on the right femoral artery and peripheral pulses were absent. Clinical examination was otherwise unremarkable. Relevant blood tests disclosed anaemia (Hb: 6.3 g/dl, mean globular volume: 95.4 μm3) and iron deficiency (iron, 38 μg/dl; iron binding capacity, 347 μg/dl; ferritin, 13 ng/ml, normal, 10–300) despite recent blood transfusions. Gastroscopy confirmed the existence of WS. Gastric mucosal histology disclosed typical dilated mucosal capillaries, focal thrombosis (Figure 3a) as well as fibromuscular hyperplasia (Figure 3b). Hormone therapy (norethisterone 1 mg and ethynylestradiol 0.05 mg; Ovismen R, Cilag, daily) was initiated. Within 3 months haemoglobin steadily increased, without further evidence of bleeding. Tolerance to the treatment was excellent. One year later, no complication has been observed except a single episode of clotting in the dialysis circuit requiring an increased heparin dosage. The appearance of a slight gynaecomastia led to an alternate-day prescription of the hormones in November 1994, without any evidence of recurrent bleeding (Figure 1).

Discussion

The watermelon stomach has rarely been reported as a cause of digestive bleeding in uraemic patients [2–5]. It refers to a localized form of angiodysplasia confined to the gastric antrum. As shown by the present report, its diagnosis is of critical importance as the prescription of an oestrogen–progesterone preparation may provide a sustained cure.

Angiodysplasia is a vascular lesion of the gastrointes-
Fig. 1. Blood transfusion requirements and serial haemoglobin concentrations before and after hormone treatment.

Fig. 2. Endoscopic photograph of the gastric antrum showing longitudinal folds of dilated vessels radiating from the pylorus and resembling the skin of a watermelon.

A peculiar form of angiodysplastic lesion confined to the gastric antrum was first described in 1953 by Rider et al. and named antral vascular ectasia [9]. The term watermelon stomach was coined in 1984 by Jabbari, who was struck by its endoscopic features: longitudinal gastric antral folds containing visible

Fig. 3. a Microscopic features of WS: gastric antral mucosa with capillary ectasia focally thrombosed, and b fibromuscular hyperplasia in the lamina propria.
vessels radiating from the pylorus and resembling the skin of a watermelon [6]. The same lesions of antral vascular ectasia may extend diffusely throughout the gastric antrum, with circumferentially distributed red-dish spots, coalescent in some areas [10]. The two forms differ only on endoscopic appearances since histological aspects are similar. Progression from the localized WS aspect to the diffuse form has been reported [6].

Endoscopic diagnosis may prove difficult for an investigator unfamiliar with this pattern. On biopsy, however, histological evidence for antral vascular ectasia is characteristic, as in our patient: hyperplastic antral mucosa, dilated capillaries in the submucosa and in the lamina propria, some of them with fibrin thrombi, fibromuscular hyperplasia of the muscularis mucosae in the lamina propria [6]. The absence of signs of gastric inflammation rules out haemorrhagic antritis, a frequent misdiagnosis [11,12]. As biopsy carries only a minimal risk of haemorrhage, it should always be performed.

The aetiology of the WS remains obscure. Portal hypertensive mucosal vasculopathy [13], degenerative lesion related to age [14], antral prolapsus [6,14], achlorhydria with hypergastrinaemia [15,16] have all been considered.

While angiodysplastic lesions are incriminated in 1.2–8% of GI tract bleeding episodes in patients with normal renal function [8], several retrospective reports suggest that they are responsible for 19–32% of the GI tract bleeding episodes observed in patients with chronic renal failure [2–5]. These lesions are located mainly in the stomach and the duodenum but the jejunum and the colon can also be affected [2,4,5]. The actual frequency of antral vascular ectasia and watermelon stomach in uraemic patients remains to be determined. This diagnosis is clearly not unusual: in a recent series of 45 patients with typical WS, six had chronic renal failure [17].

To the best of our knowledge, this is the first report demonstrating the beneficial effect of hormone therapy on WS in an uraemic patient. Oestrogen–progesterone therapy dramatically improved our patient’s condition: blood transfusions could be interrupted and haemoglobin returned to the target value of 10 g/dl without significant changes in erythropoietin prescription. No recurrence has been observed 12 months later despite a reduction in oestrogen–progesterone dosage.

Interestingly, hormone therapy was first successfully initiated in seven uraemic patients with bleeding GI angiodysplastic lesions, four of which being located in the stomach [18]. Its effectiveness was demonstrated in a double-blind, placebo-controlled, cross-over trial involving 10 non-uraemic subjects with chronic haemorrhage from GI angiodysplastic lesions (7 in the stomach) [19–21]. Successful hormonal treatment of antral vascular ectasia has recently been reported in non-uraemic patients [22–24]. The mechanism of action of this therapy remains unknown. A trophic effect on the mucosa [25], a primary effect on haemostasis [26], on the gastric microcirculation [27] or on oestroprogestative receptors located on vascular ectasia [28] have all been considered.

Hormone therapy provides an attractive alternative to currently available treatments. Antrectomy, indeed, offers definitive cure but carries significant morbidity and an operative mortality of 5–10% [12,16]. Conservative treatment with repeated blood transfusions, iron replacement, H2 antagonists, sucralfate, or steroids have all been applied with limited success [6,11,12,14,29]. Although reported to be safe and effective [17], endoscopic laser coagulation is made difficult by the diffuse location of the lesions, and requires repeated sessions [12,14,17].

In conclusion, we report an unusual cause of chronic GI haemorrhage in a dialysed patient, the watermelon stomach, and illustrate the long-term therapeutic value of an oestroprogestative combination.

References

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