

# A young woman with upper gastrointestinal bleeding

A young, previously healthy woman was admitted to the department of internal medicine with haematemesis and haemodynamic instability. Her haemoglobin level was low, and a rapid infusion of fluid and a blood transfusion were necessary. The first gastroscopy did not identify any clear focus of bleeding. The condition worsened, which created a demanding situation in the intensive care department and the gastrointestinal laboratory – only to result in a surprising diagnostic clarification.

**Vikas Kumar Sarna**  
v.sarna@hotmail.com  
**Njaal Stray**  
Department of Medicine  
Diakonhjemmet Hospital

*A young woman was admitted to the department of internal medicine after vomiting small amounts of fresh blood mixed with coagula, and with a haemoglobin level of 7.7 g/100 ml (reference value 11.7–15.3 g/100 ml). The previous evening she had been at a party and had consumed some alcohol. On the morning of the day of admission she felt unwell, and on her way to the toilet she suffered a near syncope. She contacted the emergency medical service. When the ambulance came, she fainted while trying to get out of bed. She had had no previous serious illness and was taking no regular medications.*

*On admission (day 0) she was pale with a slightly reduced general condition, blood pressure of 102/70 mm Hg which fell to 88/44 mm Hg, and a pulse of between 105 and 122 beats per minute. The clinical examination was inconspicuous, including negative abdominal findings. The stools appeared normal, but a test for occult blood showed an immediate positive result. Blood tests showed Hb 6.2 g/100 ml and MCV 72 fl (reference value 82–98 fl). Other blood tests were all normal.*

Important elements of the initial anamnesis were questions about comorbidity and use of medication (NSAID drugs, acetylsalicylic acid and anti-coagulants). Our patient had previously felt completely well and had not used any medication. Furthermore it was important to obtain an overview of the assumed volume of blood loss. With a current haemoglobin value of 6.2 g/100 ml and an assumed initial value of approx. 13 g/100 ml, it was possible to gauge an approximate

blood loss of at least 40 % of her total blood volume. This presupposed that compensatory haemodilution had been able to occur. If a blood loss of this magnitude had occurred acutely, it would have resulted in haemodynamic instability. If this volume of blood is lost over a period of time, the body can manage to initiate corrective measures. In this type of compensated situation an acute smaller amount of bleeding can be serious. Our patient came in with an anamnesis of light gastrointestinal bleeding with low MCV value, but with an abnormally low haemoglobin level and additionally signs of haemodynamic instability. This suggested that she had perhaps had occult blood loss over a period of time and now had acute bleeding.

Haematemesis always indicates that the bleeding is located in the upper gastrointestinal tract, i.e. proximal to the ligament of Treitz. Tentative diagnoses were peptic ulcer bleeding, Mallory-Weiss lesion (tears in the mucosa at the junction of the oesophagus and stomach induced by retching), tumours and vascular malformation. In a young person with no sign of liver disease, bleeding varices were a less relevant differential diagnosis.

*The patient was admitted to the intensive care department and received parenteral fluid and a total of four bags of SAGMAN blood on the first day. In addition, treatment was started with intravenous pantoprazole bolus dose 80 mg, followed by a maintenance infusion of 8 mg/hr, because of a suspected bleeding ulcer.*

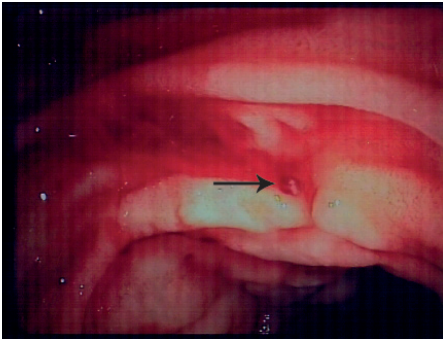
*The hospital had no staff on call to perform gastroscopy at the time the patient was admitted. Collaborating hospitals that had such staff on call were not contacted since the condition rapidly stabilised after the patient was moved to the intensive care department. During the first night moderate amounts of melena were produced regu-*

*larly, but no new haematemesis. In the morning (day 1) the haemoglobin value was 8.5 g/100 ml. Gastroscopy showed no traces of blood down to the lower portion of the duodenum, and neither were any potential sources of bleeding detected. The only findings were low-grade reflux oesophagitis, a small hiatus hernia and a small superficial ulcer (< 4 mm) in the antrum with no bleeding stigmata. A rapid urease test with a view to *Helicobacter pylori* was negative.*

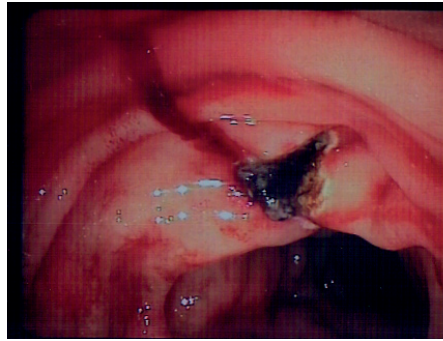
It was somewhat surprising that gastroscopy showed no potential lesions or traces of blood in the upper gastrointestinal tract. However, it was possible that the lesion was so small (or located in such a way) that it was overlooked. The possibility was also considered that there were two lesions, a minimal lesion in the upper gastrointestinal tract, and a larger one distal to the duodenum. A repeat gastroscopy prior to a possible colonoscopy was therefore called for.

*Because of her stable haemoglobin value, the patient was moved to a ward. The same evening the haemoglobin value fell to 6.5 g/100 ml. Persistent low-grade melena was unchanged. She was given two further bags of SAGMAN blood and more fluids. Early the next morning (day 2) a repeat gastroscopy was performed. Again conditions were normal down to the lower portion of the duodenum. But as the scope was being pulled back, heavy bleeding commenced from the duodenum. The view was quickly and significantly obscured. The suspected focus of bleeding was localised to the medial wall immediately distal to the duodenal bulb. Diluted epinephrine (0.1 %) was injected in several depots and haemostasis was gradually achieved. Polidocanol 1 %, 0.5 ml was also injected in each depot to address the assumed focus of bleeding. However, there were no visible lesions in the mucous membrane.*

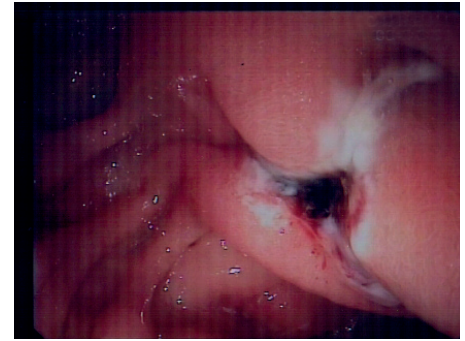
*The patient was given two further bags of SAGMAN blood, and treatment was also started with tranexamic acid 1.5 g × 4 intravenously. A repeat gastroscopy on the afternoon of the same day showed continued fresh bleeding from the same area of the duodenum. Diluted epinephrine was again injected, which resulted in a diminution of bleeding. To obtain a better overview, an ERCP scope was used (optics to the side). It was thereby possible to see capillary bleeding from a point with no wound bed (Figure 1). Polidocanol*



**Figure 1** Persistent bleeding from a point [arrow] in the mucous membrane with no visible wound bed



**Figure 2** Haemostasis following injection of Aetoxysklerol and application of argon plasma coagulation



**Figure 3** Surveillance gastroscopy the day after therapy shows that bleeding has stopped

1% was injected, total 4.5 ml at 0.5 ml per injection, and afterwards argon plasma coagulation (APC) was carefully applied to the point of bleeding until full haemostasis was achieved (Figure 2).

Arterial bleeding from a macroscopically normal mucous membrane provided diagnostic clarification. The finding was consistent with Dieulafoy's lesion (exulceratio simplex), i.e. bleeding from a dilated submucous arteriole.

The patient's haemoglobin level was stable at 7.7 g/100 ml until the following day (day 3) and continued to rise throughout the day (8.4 g/100 ml) without any further blood transfusion. There was no stool/melena in the morning or afternoon. A repeat gastroscopy (no. 4) confirmed that the bleeding had stopped (Figure 3). Because of information in the case history about possible low-grade haemorrhagic diathesis, a haematologist was conferred with who recommended testing for von Willebrand disease during remission. A rheumatologist was also conferred with, who found no indications of vasculitis. A CT scan of the abdomen revealed only slight oedema of the duodenal wall.

The patient received intravenous iron infusion with a transition to peroral iron treatment with pantoprazole per os. She was discharged with a haemoglobin value of 9.7 g/100 ml after five days of hospitalisation. A gastroscopy check-up was scheduled for four weeks later.

At the four-week check-up the patient was able to state that she had had no further episodes of bleeding. Gastroscopy performed with an ERCP scope detected no pathological signs in the duodenal mucous membrane.

Blood tests taken after four months showed normalisation of haematological variables (Hb 14 g/100 ml, MCV 94 fl, ferritin 102 mcg/l [reference value 10–200 mcg/l]), including normal von Willebrand tests.

## Discussion

Dieulafoy's lesion is named after the French surgeon Paul Georges Dieulafoy (1839–

1911). The bleeding is thought to emanate from an abnormally dilated submucosal arteriole of 1–3 mm (1). The aetiology is unknown. One hypothesis is that the blood vessel defect is congenital and that the blood vessel is unable to be provided with calibre-reducing bifurcations (2). Bleeding occurs secondary to a macroscopically invisible erosion of the overlying mucous membrane. The diagnosis is made endoscopically by the presence of active arterial bleeding, visible blood vessel or a fresh adherent thrombus in macroscopically normal mucous membrane, or a small defect in the mucous membrane of a maximum of 3 mm (3).

Our patient had signs of chronic occult gastrointestinal bleeding, with acute deterioration. Only with the second gastroscopy was bleeding detected from an area of the duodenum which is not normally viewed properly with a gastroscopy. With the aid of an ERCP scope a focus of bleeding was then detected that was consistent with Dieulafoy's lesion. This is a rare but important condition that must be considered when faced with unexplained chronic or acute gastrointestinal bleeding. In up to 70% of cases the lesion is discovered after the first gastroscopy (1). In the remainder of cases, as with our patient, repeat gastroscopies will normally be successful. Endoscopic ultrasound examination may be a diagnostic tool (2). The reason why the focus of bleeding is not found with the first gastroscopy may either be that the picture is impaired by a large quantity of blood (44%) or because the lesion is not found (56%) (1). Based on suspicion and local expertise a choice can then be made to continue with a repeat gastroscopy, balloon-assisted enteroscopy, capsule enteroscopy, angiography or technetium-labelled erythrocyte scintigraphy (2). Aside from this, there is little to be gained from radiological diagnostics (CT, MRI etc.).

Studies from hospital material show that Dieulafoy's lesion is the cause of between 1% and 5.8% of acute gastrointestinal bleedings (4, 5). It is also assumed that the condition is underdiagnosed. Bleeding is often intermittent and can thus be difficult to detect. It presents mainly as haematemesis and/or melena,

and in a minority of cases there may be fresh rectal bleeding (haematochezia). The defect most frequently occurs in the stomach sac, and usually on the lesser curvature within 6 cm of the junction of the oesophagus and stomach sac (1, 4). One third of the lesions are outside the stomach sac (1, 6). Although the duodenum is the second most frequent location, a focus on the descending part of the duodenum is very unusual (7). However, the lesion can occur in any area of the intestine and has also been described as occurring outside the intestine (1).

The patient group with Dieulafoy's lesion closely resembles the group with peptic ulcer. In one study the average age of patients with Dieulafoy's lesion was 67, and there was a preponderance of men (1, 4). In up to 90% of the cases there are comorbid diseases such as ischaemic heart disease, renal failure, diabetes, hypertension and liver failure (6). Stomach pains with Dieulafoy's lesion are, however, unusual (2), and no clear causal association with the use of NSAID drugs, acetylsalicylic acid or warfarin has been found (1, 4).

The main differential diagnoses for upper gastrointestinal bleeding are mentioned above. When an endoscopic diagnosis of Dieulafoy's lesion is made, a bleeding peptic ulcer has in practice already been ruled out. Bleeding close to the papilla must be distinguished from bleeding emanating from the biliary tree (haemobilia), when blood will leak from the papilla. The latter type of bleeding is less usual, often resulting from trauma, and may be accompanied by stomach pain and jaundice (8).

Surgery was previously the only curative treatment for this condition and historical material describes a mortality rate of 80% (1). Endoscopic treatment has now become the preferred treatment modality, and progress in the treatment has resulted in a fall in mortality to 8.6% (1). Available endoscopic treatment alternatives can either be thermal therapy (electrocoagulation, argon plasma coagulation etc.), local injection therapy (epinephrine and sclerotherapy) or mechanical therapy (band ligation and haemoclipping). Injection and argon plasma coagulation

ation were used in combination in our patient. Endoscopic haemostasis is described as occurring in over 90 % of the cases (4). Mechanical therapy is considered to be most effective (1). Use of at least two treatment methods has proven to reduce the rate of rebleeding (1). In one study, rebleeding occurred in seven of 39 patients within three days of successful primary haemostasis (9). In one review article the rate of rebleeding is considered to be between 9 % and 40 % (1). A repeat endoscopic treatment is preferred for rebleeding.

Endovascular detection and treatment of bleeding lesions is an alternative to endoscopic treatment (6). Surgical treatment is reserved for the 5 % of cases which fail to respond to endoscopic and/or endovascular treatment (1). Surgical treatment consists of either ligation or wedge resection of the lesion. Encouraging progress has been described involving laparoscopic treatment and methods combining perioperative endoscopy and laparoscopy, or laparoscopy following prior endoscopic tattooing of the lesion (1).

Bleeding from Dieulafoy's lesion will often present diagnostic and therapeutic

challenges. In the vast majority of cases, gastrointestinal endoscopy will provide the correct diagnosis and effective treatment. A study with a 28-month follow-up period has shown that the prognosis for those discharged from hospital following endoscopic haemostasis is excellent. (5). Mortality is primarily associated with comorbid conditions (4, 5).

**Vikas Kumar Sarna (born 1980)**

is a doctor in specialty training in internal medicine and gastroenterology at the department of internal medicine, Diakonhjemmet Hospital.

The author has completed the ICMJE form and reports no conflicts of interest.

**Njaal Stray (born 1944)**

is a specialist in gastroenterology. He is Departmental Senior Consultant at the department of internal medicine, Diakonhjemmet Hospital.

The author has completed the ICMJE form and reports no conflicts of interest.

**References**

1. Baxter M, Aly EH. Dieulafoy's lesion: current trends in diagnosis and management. *Ann R Coll Surg Engl* 2010; 92: 548–54.
2. Jaspersen D. Dieulafoy's disease controlled by Doppler ultrasound endoscopic treatment. *Gut* 1993; 34: 857–8.
3. Hyun CB. Periampullary Dieulafoy's lesion. *Hosp Physician* 2005; 41: 23–7.
4. Walmsley RS, Lee YT, Sung JJ. Dieulafoy's lesion: a case series study. *World J Gastroenterol* 2005; 11: 3574–7.
5. Baettig B, Haeckel W, Lammer F et al. Dieulafoy's disease: endoscopic treatment and follow up. *Gut* 1993; 34: 1418–21.
6. Alshumrani G, Almuaikel M. Angiographic findings and endovascular embolization in Dieulafoy disease: a case report and literature review. *Diagn Interv Radiol* 2006; 12: 151–4.
7. Rana SS, Bhasin DK, Gupta R et al. Periampullary Dieulafoy's lesion: an unusual cause of gastrointestinal bleeding. *JOP* 2010; 11: 266–9.
8. Chin MW, Enns R. Hemobilia. *Curr Gastroenterol Rep* 2010; 12: 121–9.
9. Lim W, Kim TO, Park SB et al. Endoscopic treatment of dieulafoy lesions and risk factors for rebleeding. *Korean J Intern Med* 2009; 24: 318–22.

*Received 12 October 2011, first revision submitted 28 February 2012, approved 14 April 2012. Medical editor: Jon Amund Kyte.*