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DIEULAFOY'S LESION: CASE REPORT

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SUMMARY

A sixty-year old male patient was referred to the author as a case of massive rectal bleeding after haemorrhoidectomy. He underwent urgent total colonoscopy and bleeding Dieulafoy's lesion was identified as a source of haemorrhage. Injection with dilute adrenalin in and around the bleeding lesion was carried out with prompt haemostasis and no recurrence. High index of suspicion and early therapeutic endoscopic intervention is extremely useful in this rare but important cause of massive gastrointestinal bleeding.

INTRODUCTION

Dieulafoy's lesion is essentially a tortuous, submucosal artery which protrudes through a small mucosal defect and may spontaneously rupture leading to haemorrhage. Awareness of this condition and early endoscopy is the mainstay of diagnosis. Injection with dilute adrenaline in and around the bleeding vessel is safe and effective with good long term results. This is a case report of a 60 year old male who was referred to the author as a case of massive rectal bleeding after a haemorrhoidectomy. Dieulafoy's lesion was identified and injected with good results.

CASE REPORT

A total of 855 upper and lower gastrointestinal endoscopies have been carried out by the author between January, 1995 and 30th October 1999. Only one case of Dieulafoy's lesion was identified in a 60 years old male patient. This patient was earlier treated for haemorrhoids when he had presented with rectal bleeding. Ten days later, he was re-admitted with massive rectal bleeding which was continuous, frank red with clots and not associated with defaecation. He was on warfarrin for recurrent deep venous thrombosis diagnosed five months earlier and of which the cause was not known and the INR was 3.54. His haemoglobin on admission was 9.7 gm/dl which decreased to 7.6 gm/dl despite two units of blood transfusion. He required a total of four units of blood to keep his Hb at 10 gm/dl. He underwent a total colonoscopy with a fiber-optic colonoscope approximately 12 hours after his rectal bleed which revealed a Dieulafoy's lesion 13 cms from anal verge with major stigmata of recent haemorrhage (pulsating red clot attached to the mucosal defect). There was no other source of bleeding identified upto the caecum and this fact together with the stigmata of recent haemorrhage convinced the author that the Dieulafoys lesion was the only possible cause of his rectal bleeding. It was injected with dilute adrenaline with good results and has not had a recurrence of bleeding despite continuing his warfarrin.

DISCUSSION

Dieulafoy's lesion is an uncommon but important cause of acute, recurrent and sometimes massive upper or

lower gastrointestinal haemorrhage. Bleeding occurs from a pinpoint, non ulcerated, arterial lesion, usually from gastric fundus (75 - 95%) but can also occur from distal oesophagus, small intestine, colon or rectum(1,2). The incidence of the lesion may vary from 0.5% to 14%(2).

The first case was described by Gallard in 1884 followed by Dieulafoy, a French surgeon, who described three cases in 1898(3). Dieulafoy's lesion is a difficult lesion to identify when it is not bleeding as the overlying mucosa may look normal. It has been given other names like cirroid aneurysm, submucosal arterial malformation and calibre persistent artery.

In case of massive upper gastrointestinal bleeding no cause may be identified in four to nine of cases; one to two per cent of these unidentified sources of bleeding could be a Dieulafoy's lesion(4). There is a male preponderance, M:F = 2:1 and majority of the patients are in the fifth decade of their lives. A Dieulafoy's lesion could be overlooked as concomitant lesions such as ulcers, varices or haemorrhoids may be wrongly considered responsible for the haemorrhage.

The pathogenesis of Dieulafoy's lesion was initially thought to be an aneurysm in combination with atherosclerosis in one of the vessels within the walls of stomach or intestines. A congenital or acquired vascular malformation has also been suggested as a cause. The lesion consists of a large, tortuous submucosal artery which protrudes through a 2-5mm mucosal defect. The mechanical pressure from a large, pulsating artery may erode the thin, overlying mucosa leading to spontaneous rupture and massive G1 bleeding(5).

There has been no association mentioned between Dieulafoys lesion and DVT in the literature so far. The most common presenting symptom is recurrent, often massive gastrointestinal haemorrhage characterised by haematemesis and melaena in 51% of patients, while 28% may present with haematemesis alone and 18% presenting with melaena alone. Patients with lesions distal to the jejunum present with massive rectal bleeding. There is no history of dyspepsia, anorexia, NSAID or alcohol abuse(6).

Dieulafoy's lesion is diagnosed at endoscopy as a

minute mucosal defect with a large submucosal artery either bleeding actively or with a clot adherent to the defect.

Dieulafoy's lesion is difficult to identify and high index of suspicion is required to make a diagnosis. It can be identified in 92.3% to 96.4% of cases if endoscopy is performed within the first two hours of bleeding(2). The endoscopic appearance varies from normal mucosa to a 2 to 5 mm mucosal defect with a protruding vessel or with a clot. About half of the lesions are identified during the initial endoscopic examination while 33% require more than one endoscopy for confident identification. The rest are identified either intraoperatively or by angiography (1,7).

Therapeutic endoscopy is the initial treatment of choice. In the pre-endoscopy era, mortality rates approached 80% but with the development of effective endoscopic haemostatic methods, the mortality rate is expected to be minimal as long as the cause is recognised early(1,6). The modalities used include injection sclerotherapy, laser photocoagulation, epinephrine injection, haemoclipping and banding. Endoscopic therapy is successful in 85% of cases in achieving permanent haemostasis, ten per cent require a repeat endoscopic therapy while five per cent may require surgical intervention(8). Surgical exploration with intra-operative endoscopy can achieve excellent results combining the advantages of endoscopic visualisation with open surgical ligation of the bleeding vessel(8).

In summary Dieulafoy's lesion should be considered in cases of acute, recurrent, massive gastrointestinal

bleeding. Awareness of the condition and experience in endoscopy are the mainstay of diagnosis. Therapeutic endoscopy is the first line of treatment. It is safe and effective with good long-term results.

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REFERENCES

1. Katz, P.O. and Salas, L. Less frequent causes of upper gastrointestinal bleeding. *Gastroent. Clin. N. Amer.* 1993; **22**:875-89.
2. Baettig, B., Haecki, W., Lammer, F. and Jost R. Dieulafoy's disease: endoscopic treatment and follow-up. *Gut.* 1993; **34**: 1418-21.
3. Veldhuyzen van Zanten, S.J.O., Bartelsman, J.F.W.M. and Schipper, M.E.I. Recurrent massive haematemesis from Dieulafoy vascular malformations. *Gut.* 198: 213-22.
4. Durham, J.D., Kumpe, D.A., Rothbart, L.J. and van Stiegmann G. Dieulafoy disease: arteriographic finding and treatment. *Radiology.* 1990; **174**:937-41.
5. Miko, T.L. and Thomazy, V.A. The caliber persistent artery of the stomach: a unifying approach to gastric aneurysm, Dieulafoy's lesion and submucosal arterial malformation. *Hum. Path.* 1988; **19**:914-21.
6. Reily, H.F. and Al-Kawas, F.H. Dieulafoy lesion: Diagnosis and management. *Dig. Dis. Sci.* 1991; **36**:1702-7.
7. Helliwell, M. and Irving, J.D. Haemorrhage from gastric artery aneurysms. *Brit. Med. J.* 1981; **282**:460-1.
8. Balserrak, J.C. and Neal, D. Intraoperative endoscopy as an adjunct to surgical ligation of multiple arteriovenous malformations. *Surg. Lap. Endo.* 1996; **6**:68-70.