Painless Abdominal Apoplexy

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Spontaneous rupture of retroperitoneal vessels is rare, but when it occurs it is almost invariably accompanied by pain, ileus, and increasing shock (Kennedy Browne and Glashan, 1965).

In the following case an apparently normal retroperitoneal artery ruptured, producing severe shock but no pain whatsoever.

CASE REPORT

The patient, a man aged 62, was admitted to St. Thomas's Hospital on 19 February 1965. Apart from trigeminal neuralgia for seven years, he had been well until the day of admission, though eight years previously he had undergone a Polya partial gastrectomy for duodenal ulceration.

Six hours before admission he collapsed at home and was taken to the Maida Vale Hospital, where he was found to be shocked and was transferred to St. Thomas's Hospital. He did not remember any warning symptoms before his collapse, but stated that for the previous week his stools had been loose, though defaecation occurred only once daily. On admission he had no pain whatsoever, though when questioned he admitted to minimal abdominal discomfort and had a constant urge to defaecate.

He was shocked, with hypotension, systolic arterial blood pressure 60 mm. Hg, peripheral vasoconstriction, pallor, and excessive sweating. The oral temperature was 99° F. (37.2° C.), and the pulse rate 92. No abnormalities were found in the heart, lungs, or nervous system. His tongue was moist but coated. The abdomen was soft and easily palpated, no mass being felt. There was neither abdominal tenderness nor distension, and the bowel sounds were active without being abnormal. Rectal examination revealed no abnormality. While in the casualty department he passed a loose offensive stool, which, though showing numerous Gram-positive and negative bacilli, and Gram-positive cocci in the smear, subsequently grew no pathogenic bacteria. He then vomited, and examination of both vomitus and stool for occult blood was negative.

The haemoglobin was 13.2 g./100 ml. (90%), and the plasma sodium and potassium were 125 and 3.8 mEq/l. respectively. An electrocardiogram showed no evidence of myocardial infarction.

In the absence of evidence of gastrointestinal bleeding, the loose offensive stool suggested enteritis. After taking specimens for culture, treatment with ampicillin was begun and an intravenous infusion of dextran (Dextravan) was set up. The blood pressure rose to 90/76 and the pulse fell to 80 after only 500 ml. of this infusate, and the infusion was continued with saline. The blood pressure soon reached 120/90, and remained at this level.

Six hours later he developed minimal left-sided abdominal discomfort, and the signs of free fluid were now detectable within the abdomen. The haemoglobin had fallen to 8.6 g./100 ml. (59%). The white blood cell count was 12,000/cu. mm., 80% polymorphonuclear cells, and the erythrocyte sedimentation rate was 67 mm. in one hour (Westergren). An erect radiograph of the abdomen did not show any fluid levels.

Laparotomy was performed through a right paramedian incision. More than 1 litre of blood was evacuated from the peritoneal cavity, and the whole of the transverse mesocolon was engorged with blood. On the undersurface of the mesocolon, close to the main stem of the middle colic artery, a haematoma had burst through the peritoneum. This was evacuated and a ruptured vessel was seen to be bleeding profusely. Both ends were ligated and a biopsy specimen was taken. The viability of the transverse colon remained unimpaired throughout and the abdomen was closed with drainage. Skin and muscle biopsy specimens were also taken.

His postoperative course was uneventful, but the bowel actions remained frequent throughout his stay in hospital and he passed two to four loose stools a day. Five weeks after operation the faecal fat excretion was found to be somewhat raised at 8.7 g./24 hours. At that time the haemoglobin was 10.6 g./100 ml. (73%), serum calcium 8.9 mg./100 ml., and plasma potassium 4.3 mEq/l. His bowel action appeared to be slowly returning to normal and he was discharged from hospital on 31 March.

During the next 10 months he gained 37 lb. (16.8 kg.) in weight and his bowel action returned to normal. Eleven months after

operation he was readmitted for estimation of his faecal fat excretion, which was then found to be normal (4.5 g./24 hours). No clinical abnormalities were found at this time. The arterial blood pressure was 170/100 and blood analysis showed haemoglobin 13.2 g./100 ml. (90%); the white blood cell count was 6,800/cu. mm., with a normal differential count, and his E.S.R. was 1 mm. in one hour. The plasma electrolytes and the blood urea were normal.

Histological examination of the arterial biopsy specimen showed a normal artery. No evidence of a generalized disease such as polyarteritis nodosa could be found in the muscle biopsy.

DISCUSSION

Kennedy Browne and Glashan (1965) reviewed the world literature on abdominal apoplexy, confirming it to be a very rare condition. They quoted 47 cases and added three of their own. Among the 31 patients in whom the source of the bleeding was found, only three examples of spontaneous rupture of the middle colic artery and its branches were described. They also list the presenting symptoms of these cases, and in 42 of the 47 where these were known epigastric or abdominal pain was the major symptom. Shock, dyspnoea, gastric upset, and precordial pain were recorded.

Of the three previous cases of middle colic vessel rupture, two presented with abdominal pain. The third patient had no pain and died before the cause of her shock was recognized (Lafferty and Pearson, 1940).

Our patient presented in a state of clinical shock and his main symptoms were diarrhoea and vomiting. At no time did he experience abdominal pain. Laparotomy was not performed until 12 hours after his admission, when free fluid was detected within the abdomen and it was found that the haemoglobin level had fallen.

He also had trigeminal neuralgia and was receiving 200 mg. t.d.s. of carbamazepine (Tegretol). During the week before his admission the trigeminal neuralgia had become worse, necessitating injections of methadone. The latter drug had last been given six hours before he collapsed, and its analgesic action does not usually persist after four hours. It seems unlikely, therefore, that it was responsible for the absence of abdominal pain. It is more difficult to assess the role of carbamazepine in this respect. This drug is still under trial as an anticonvulsant, but seems to be a specific analgesic for trigeminal neuralgia. The mechanism of the latter action is unknown, but it is possible that carbamazepine may have helped to minimize the abdominal symptoms.

The ruptured vessel showed no evidence of atheroma or other disease. This finding agrees with those of Kennedy Browne and Glashan that in most cases the burst vessel is normal. No evidence of polyarteritis nodosa was found in the biopsy specimens taken from this patient, and indeed his subsequent good health makes it most unlikely that there was any such systemic disease.

Faecal fat excretion was slightly raised in the convalescent period, whereas 10 months later it was normal. The significance of these findings is obscured by the fact that the patient had undergone partial gastrectomy, and it is uncertain whether the steatorrhoea was related to his abdominal apoplexy.

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