Uterine restoration following fibroid expulsion after uterine artery embolisation using gelfoam

Sir,

We have been using uterine artery embolisation routinely at our centre for the last two years to treat selected symptomatic patients with fibroids. We report a case of a 35-year-old woman who presented with menorrhagia and dysmenorrhoea with a uterus measuring 5.9 cm in diameter, as seen on transabdominal ultrasound and MR imaging. The patient underwent bilateral uterine artery embolisation using a single 5 F Uterine Artery Catheter (Cook, Bloomington, IN, USA) from the right transfemoral route using standard technique. Instead of using polyvinyl alcohol particles we used gel foam particles as detailed in the article by Katsumori et al. Embolisation of both uterine arteries was effected to the point of near occlusion of the uterine arteries and the uterine vascular bed with reflux of contrast into the arch segment and the descending parts of the uterine arteries. Post-embolisation crampy abdominal pain was effectively controlled using intravenous infusion of a combination of pentazocine and midazolam which continued for two days following the procedure. Following the procedure, the patient’s symptoms of menorrhagia and dysmenorrhoea improved in the first menstrual cycle itself. Three months following the procedure, the patient reported painless evacuation of a fleshy mass per vaginum on the first day of her menstrual period. Ultrasound revealed a soft abdomen, with hepatosplenomegaly and no evidence of any mass or fibroid. Blood examinations revealed haemoglobin of 11 g/dL, total leucocyte count of 9.9 x 10^3/L, and platelet count of 234 x 10^3/L. A soft mass was palpable in the region of the uterus, which on further interrogation was identified as a sized uterus with no evidence of fibroid. A midline incision was made and a soft mass was excised with cautery and sutured. Ultrasound revealed no evidence of any residual mass. The patient made an uncomplicated recovery and was discharged on the 5th postoperative day with no recurrence of symptoms. The patient has remained asymptomatic since this episode for the last one year.

Uterine artery embolisation has been tried as an alternative to surgery and has proved successful in treating symptomatic fibroids. Permanent embolisation particles (such as polyvinyl alcohol) are usually injected into uterine arteries to obtain fibroid shrinkage. Results of fibroid embolisation have been very encouraging. In the study by Klein et al, 92% patients were satisfied with the reduction of bleeding, and 78% were satisfied with the reduction in pressure symptoms. The mean decrease in uterine volume was 36%, and the mean decrease in the size of the dominant fibroid was 49%. Most of the shrinkage of the fibroid occurs within a 6-month period with further reduction occurring between 6-12 months. A few studies have reported expulsion of the fibroid following embolisation, as happened in our case. There has, however, been only one report in English literature so far of complete uterine restoration following expulsion of the fibroid, which was reported following the use of polyvinyl alcohol particles, with complete cessation of symptoms thereafter. Our case became totally asymptomatic and had painless expulsion of the fibroid three months following embolisation and is unique because it followed embolisation using gel foam particles, which are very economical and yet highly effective embolisation materials as recently reported.

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References

Persistent hypotension and splenic rupture in a patient with Plasmodium vivax and falciparum co-infection

Sir,

A 28-year-old man was admitted with fever, abdominal pain, vomiting and loose stools of three days’ duration. He was from a region non-endemic for malaria. There was no history either of previous episodes of malarial infection or of travel to an endemic area. On examination, the pulse rate was 106/min and blood pressure was 100/70 mm of Hg. He was febrile (temperature 40°C), and was pale and icteric. His abdominal examination revealed a soft abdomen, with hepatosplenomegaly and mild left hypochondrial tenderness.

Investigations revealed haemoglobin of 5.7 g/dL, total leucocyte count of 4.9 x 10^3/L and a platelet count of 23 x 10^3/L. Thin blood smear revealed Plasmodium vivax rings, schizonts and gametocytes. Gametocyte and ring stages of Plasmodium...
falciparum were also seen. The parasitic index for *Plasmodium falciparum* and vivax (combined parasitaemia) was 4%. Total bilirubin was 5.1 mg/dl and direct bilirubin was 0.5 mg/dl. The aspartate aminotransferase, alanine aminotransferase and alkaline phosphatase were 130, 31 and 43 IU/L respectively. The serum creatinine was 1.2 mg/dl. He was started on intravenous infusion of quinine at a dose of 10 mg/kg/dose, every eight hours and transfused one unit of blood. Since the post- transfusion haemoglobin was only 6.1 gm/dl, a further unit was transfused, following which the haemoglobin increased to 7.1 gm/dl.

He continued to have abdominal pain, vomiting and loose stools. In spite of transfusing six litres of intravenous fluids and two units of blood, the systolic blood pressure continued to be between 90 and 100 mm of Hg. An ultrasound examination of the abdomen revealed moderate splenomegaly (17 cm) with a large peri-splenic haematoma, and moderate intraperitoneal haemorrhage. Computerised tomography revealed a mixed density lesion in the perisplenic region with left-sided pleural effusion, confirming the diagnosis of splenic rupture.

He underwent an emergency laparotomy, which revealed hemoperitoneum with over one litre of blood in the peritoneal cavity and friable spleen with capsular tear. Splenectomy was performed. Pathological examination revealed a laceration on the antero-superior surface. The post-operative period was uneventful and he was discharged after 7 days. The repeat thin film examination of blood revealed no parasites. He had a normal inflammatory profile with no signs of anaemia.

Splenectomy was disposed this patient to spontaneous splenic rupture. He continued to have abdominal pain, vomiting and loose stools. In spite of transfusing six litres of intravenous fluids and two units of blood, the systolic blood pressure continued to be between 90 and 100 mm of Hg. An ultrasound examination of the abdomen revealed moderate splenomegaly (17 cm) with a large peri-splenic haematoma, and moderate intraperitoneal haemorrhage. Computerised tomography revealed a mixed density lesion in the perisplenic region with left-sided pleural effusion, confirming the diagnosis of splenic rupture.

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Splenectomy as a cause of death or morbidity has been well described, mostly in individuals residing in non-endemic areas,1 as was the case in our patient. It is more common with *Plasmodium vivax* infection and has been described in the acute phase of the illness. The majority of the patients described in the literature had classic features of splenic rupture, including signs of peritonitis and marked left hypochondrial pain, tenderness and guarding. Our patient had no prominent intra-abdominal signs. The clinical presentation was that of a patient with malaria with unexplained hypotension and severe anaemia unresponsive to transfusions. In countries where physicians encounter malaria frequently, it is important to remember that this life-threatening complication may present with subtle clinical features that may not help to readily localize the pathology to the spleen.

Our patient had a mixed *Plasmodium vivax* and *falciparum* infection. To the best of our knowledge, this is the first report of a *Plasmodium vivax* and *falciparum* mixed infection with splenic rupture. Since there is no data, it is difficult to comment on the relative prognosis of patients with mixed infection as compared to that of patients with isolated *Plasmodium vivax* or *falciparum* infection. The vast majority of cases of spontaneous splenic rupture have been described with *Plasmodium vivax* and the presence of *Plasmodium vivax* as one of the infecting species, rather than the mixed infection, may have predisposed this patient to spontaneous splenic rupture.

Conservative, non-operative management of splenic rupture has received some attention, particularly because of the risk of future episodes of malaria in an asplenic patient.2,3 Trans-catheter embolisation of the splenic artery has been described.4 However, conservative therapy requires rapid availability of unlimited quantities of blood, high quality of clinical monitoring, and sophisticated imaging techniques while splenectomy is a relatively rapid and effective way of handling a life-threatening complication. Hence, operative management of splenic rupture is likely to remain the treatment in many situations.5 It is crucial for clinicians to remember the possibility of spontaneous splenic rupture because early diagnosis coupled with a relatively simple surgery can lead to dramatic recovery, and the failure to do so, to high mortality.

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