Introduction
Spontaneous splenic haemorrhage and rupture (SSHR) is a rare entity usually associated with infectious, haematologic, neoplastic or connective tissue diseases. In these cases, the spleen is usually enlarged, and rupture is attributed to splenic involvement by the primary disease [1]. Spontaneous splenic rupture (SSR) during thrombolytic or anticoagulant treatment has also been reported frequently [2], but only two cases due to ticlopidine and one case due to salicyclate have been reported. We report the case of a 54-year-old man with haemorrhagic shock due to spontaneous splenic haemorrhage and rupture following dual antiplatelet (aspirin and clopidogrel) therapy. He was successfully treated with selective angioembolization of the bleeding branch of the splenic artery.

Case history
A 54-year-old man was admitted to the Emergency Department because of sudden onset of severe left hypochondrial pain of 1-day duration. He denied history of any gastrointestinal or urological symptoms. There was no history of recent trauma. His past medical history involved coronary angioplasty for triple vessel disease 14 days ago. He was on dual antiplatelet agents [Tab. Ecosprin (acetylsalicylic acid) 150 mg once daily and Tab. Plavix (clopidogrel) 75 mg twice daily]. He was known to have diabetes and hypertension and was on treatment with metformin (500 mg) twice a day and metoprolol (25 mg) once a day. There was a history of substantial alcohol consumption (175 ml/day) and smoking (10 cigarettes per day) for the last 25 years. There was a history of blunt abdominal trauma treated conservatively 5 years ago.

On examination, his pulse rate was 110/min and blood pressure was 110/80 mmHg. Abdominal examination revealed tenderness and guarding in the left hypochondrium. Investigations revealed normal counts and bleeding parameters. An urgent ultrasonography of the abdomen revealed haemoperitoneum with a large intrasplenic heterogeneous area (7.3 × 3.4 cm), with a subcapsular haematoma and perisplenic collection compressing the spleen. There was flow noted in the splenic artery (Figs. 1 and 2).

He was shifted to the high dependency unit and was managed with analgesics, intravenous fluids and strict bed rest. A computed tomography (CT) scan of the abdomen revealed haemoperitoneum with a large intrasplenic heterogeneous area (7.3 × 3.4 cm), with a subcapsular haematoma and perisplenic collection compressing the spleen. There was flow noted in the splenic artery (Figs. 1 and 2).

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He was shifted to the high dependency unit and was managed with analgesics, intravenous fluids and strict bed rest. A computed tomography (CT) scan of the abdomen was arranged, but during this he became haemodynamically unstable, with a blood pressure of 90/60 mmHg and a pulse rate of 110/min. His haemoglobin dropped to 10 g% (more than 33% fall). He underwent 1 U of blood transfusion to keep his haemoglobin above 10 mg/dl in view of his cardiac state.

Keywords:
antiplatelet therapy, endovascular management, haemorrhagic shock, spontaneous splenic haemorrhage

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comorbidities. Once resuscitated, an emergency CT scan was carried out, which confirmed active extravasation of contrast from one of the intrasplenic arterial branch.

Options available at this stage were emergency surgery and angiographic embolization. In view of the recent myocardial infarct, it was decided to embolize the bleed. After obtaining informed consent from the patient, Digital Subtraction Angiography through the right transfemoral route using 4-Fr sheath and 4-Fr cobra glide catheter (Fig. 3a and b) showed active bleeding from the posterior division of the splenic artery, which was selectively catheterized using a microcatheter (Progreat, Somerset, NJ, USA). The bleeder was successfully embolized using gel foam particles, preserving the rest of the splenic vasculature. Recovery was uneventful and haemoglobin levels remained normal. Follow-up ultrasonography after 5 days showed regression of haematoma (7.3 × 2.4 cm) (Fig. 4). On telephonic follow-up after a month, the patient was asymptomatic and was doing well.

**Discussion**

SSR in the absence of trauma is exceedingly rare and is usually associated with an underlying pathological condition, mostly neoplastic [6]. Anticoagulants and thrombolytic agents have been cited as potential causes of SSR in the literature. To our knowledge, SSR has been reported in only two cases due to ticlopidine [3] and in one case due to salicylate (aspirin) [4], treated with splenectomy.

In the presented case, splenic haematoma and rupture might have been prompted by the double antiplatelet effect of ecosprin and clopidogrel that the patient was recently started on after coronary stenting. These drugs have antiplatelet effects and act by inhibiting the production of thromboxane A2 in platelets, producing an inhibitory effect on platelet aggregation. In our case,

**Figure 1**

Ultrasonographic (USG) Doppler of the spleen showing a subcapsular haematoma compressing the spleen with flow in the splenic vasculature.

**Figure 2**

Computed tomography (CT) scan of the abdomen: (a) arterial phase axial image showing active extravasation of contrast from the posterior branch of the splenic artery (arrow); (b) venous phase coronal reconstruction showing subcapsular haematoma and haemoperitoneum.

**Figure 3**

Digital subtraction angiography: (a) Pre-embolization selective splenic angiogram showing active focal extravasation of contrast from one of the branch of splenic artery (arrow) in the interpolar region; (b) postembolization selective splenic angiogram showing complete exclusion of the bleeding vessel from the circulation with preservation of the remaining splenic parenchyma.

**Figure 4**

Ultrasonographic (USG) Doppler of the spleen showing regressing subcapsular haematoma with preserved flow in the splenic vessels.
we hypothesize that the use of ecosprin and clopidogrel has possibly created a haemorrhaging substrate. Despite knowledge on ecosprin and clopidogrel, its association and role in the pathogenesis of SSR has rarely been described before.

There are no guidelines on management of SSR. Data on risk factors, outcome, morbidity and mortality as regards SSR are limited. In haemodynamically stable patients, a conservative approach as advocated for traumatic splenic injuries is probably safe [7–9]. Transfusion and restriction of physical activity are generally required. Although splenectomy has been advocated in the past as the definitive therapy [2], the risk of morbidity and mortality after splenectomy is high [10–12]. In our patient, haemodynamic instability, falling haemoglobin, need for transfusion and active extravasation on contrast-enhanced CT scan were eventually regarded as indications for emergency angioembolization. Selective transcatheter embolization of the splenic artery branch, which permits preservation of the spleen, has become an alternative to surgery for obtaining splenic haemostasis even in haemodynamically unstable patients.

**Conclusion**

This case report highlights the potential rare complication of ecosprin and clopidogrel in causing splenic haemorrhage and rupture. Initial observation of haemodynamically stable patients with SSHR secondary to antplatelet is adequate, but the threshold for operation should be low. Although splenectomy has been advocated in the past as the definitive therapy, selective transarterial embolization of the splenic artery is a safe and effective treatment option.

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**Conflicts of interest**

There are no conflicts of interest.

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