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VASCULAR AND INTERVENTIONAL RADIOLOGY

Endovascular treatment of spontaneous extraperitoneal haemorrhage: immediate and long-term clinical efficiency

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Abstract
Purpose This study aimed to evaluate the effectiveness of transcatheter embolisation in the treatment of spontaneous extraperitoneal haemorrhage (SEH).
Materials and methods We retrospectively evaluated the technical and clinical success in terms of immediate and long-term mortality in a series of patients who underwent endovascular treatment of SEH from January 2005 to December 2010. A statistical comparison of pre- and postoperative transfusion requirements was performed by using the Student's *t* test, with statistical significance set at $p < 0.005$.
Results In the period considered, 30 patients (16 women and 14 men; mean age, 73.3 years ±15.6) with SEH underwent endovascular treatment. Technical success was obtained in all cases (100 %), and a statistically significant reduction in blood transfusion requirements was observed (mean preoperative requirement: 7.5 U/day ±3; mean postoperative requirement 2.8 U/day) ($p < 0.005$). We observed a postoperative mortality of 10 % (3/30 patients) and mortality at 6 and 12 months was 14.8 % (4/27 patients) and 26 % (6/23 patients), respectively.
Conclusion According to our experience and to the literature, transcatheter arterial embolisation represents the treatment of choice in patients with SEH, as it ensures complete therapeutic success.

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Introduction

Spontaneous extraperitoneal haemorrhage (SEH) is a particular clinical entity which manifests itself in the absence of any injury or specific underlying pathology [1]. It is usually associated with anticoagulant therapy, coagulopathy and endstage renal disease [2–4]. Its incidence has been for years considered very low, but the considerable increase in patients receiving anticoagulation therapy for various diseases [5–9] (atrial fibrillation, valvular heart disease, prophylaxis of deep vein thrombosis/pulmonary thrombo-embolism in addition to the development of increasingly effective anticoagulant, antiplatelet and antithrombotic agents) has made this complication highly relevant [10–12], with the annual incidence of major bleeding in patients on oral anticoagulant therapy currently estimated to be 0.2–3 % [13, 14].

SEH treatment consists in suspending anticoagulation therapy with correction of coagulation and restoration of blood volume through fluid replacement and blood transfusions [3, 4].

In the case of haemorrhage refractory to transfusions and of arterial bleeding, transcatheter embolisation represents the most valuable therapeutic option because it is able to stop the bleeding with less intraprocedural risk as compared to open surgery [3, 15].

Few studies have evaluated the efficacy of endovascular treatment of SEH and most of them are limited by small patients series [3, 16–21]. The objective of this study was to analyse our experience with transcatheter embolisation

in the treatment of SEH to evaluate its effectiveness in terms of immediate and late clinical success, and to compare our data with those reported in the literature.

Materials and methods

Thirty patients, who underwent endovascular treatment for SEH between January 2005 and December 2010 were retrospectively included in the study. Patients with a history of trauma, major surgery or underlying disease related to bleeding were excluded. The diagnosis of SEH was established, in accordance with the literature [1], on the basis of the finding of active bleeding in the absence of a causal event (traumatic, iatrogenic or pathological). Table 1 lists the risk factors: all patients were receiving anticoagulant/antiplatelet therapy.

The indication for endovascular treatment was the presence of persistent anaemia after conservative therapy (fluid replacement, transfusion of 5–15 U of packed cells, correction of risk factors and anticoagulants).

In 28/30 cases (93 %), angiography with contrast-enhanced computed tomography (CT) was performed with a 64- and 16-slice multidetector-row CT scanner (GE Medical Systems, Milwaukee, WI) to identify the bleeding site and the target vessel. The study protocol consisted of axial scans at the site of suspected haemorrhage and scans before and after the administration of contrast medium, with post-processing (Table 2). On baseline CT, the bleeding site was identified as a hyperdense mass attributable to the presence of fresh blood with attenuation values ranging from 60 to 80 HU; after contrast administration, active bleeding was displayed as a focal area of high density in the arterial phase.

In 2/30 cases (6.6 %), the CT study was not performed because of haemodynamic instability (systolic blood pressure <90 mmHg), and the patients underwent digital subtraction angiography (DSA). DSA was performed in all patients in the angiography room (Integris V5000 Philips, Eindhoven, The Netherlands), with approach through the common femoral artery (5F introducer Terumo Corp., Tokyo, Japan) and local anaesthesia (10 ml of lidocaine 2 %). In cases, where the CT study clearly indicated the bleeding vessel, the treatment involved selective catheterisation of the vessels by means of angiographic catheters (Cobra and Vertebral, Terumo Corp., Tokyo, Japan); where the CT scan was not conclusive, angiography with a pigtail catheter (Terumo Corp, Tokyo, Japan) followed by selective catheterisation of the injured vessel was performed.

In the presence of bleeding from small vessels superselective catheterisation was achieved using 0.025 in. a Progreat microcatheter (Terumo Corp., Tokyo, Japan) and its 0.021 in. micro-guidewire which allowed embolisation with microcoils (Boston Scientific, Natick, MA, USA) and microparticles (contour 150–250, 250–355, 355–500 μm, Boston Scientific, Natick, MA, USA). When the active bleeding vessels could not be reached, we proceeded to embolise the main afferent vessel with coils of larger calibre (Balt Extrusion, Montmorency, France).

Table 1 Risk factors

No. of patients	Risk factors	Comorbidities
14	Anticoagulant therapy (heparin)	Deep vein thrombosis, coronary artery disease, cardiac valve replacement
12	Anticoagulant therapy (warfarin)	Atrial fibrillation, cardiac valve replacement
4	Anticoagulant therapy (fondaparinux)	Deep vein thrombosis, coronary artery disease, unstable angina

The treatment algorithm was as follows: in the case of a bleeding vessel calibre of <1.5 mm (calculated on the basis of the calibre of the 5F catheter used in the diagnostic phase) we employed microparticle embolisation, whereas in the case of a calibre larger than 1.5 mm we always used coils, with preliminary use of microparticle injection before coil release in the presence of multiple haemorrhagic foci. After angiography to evaluate the success of the treatment [22], we proceeded with haemostasis by manual compression in 18 cases and in 12 cases by Proglide Perclose system (Boston Scientific, Natick, MA, USA).

We retrospectively reviewed the technical success, mortality in the short and medium term and the incidence of re-bleedings; pre-and post-procedural transfusion requirements were statistically compared using Student's t test, considering $p < 0.005$ to be significant. Student's t test was also used to compare the interval between clinical suspicion and treatment and the severity of anaemia in patients who died and survived, considering $p < 0.005$ to be significant.

Results

From January 2005 to December 2010, 30 patients (16 women and 14 men; mean age, 73.3 years ±15.6) with SEH underwent endovascular treatment with transcatheter embolisation. Two of them were emergency procedures and the remaining 28 were urgent procedures.

A preliminary CT scan was performed in 28/30 patients (93 %): in all cases CT was conclusive as it allowed identification of the haematoma with active contrast leakage, with clear visualisation of the bleeding vessels (Fig. 1).

Table 2 Computed tomography (CT) scan protocol

Variable	64-slice CT		
Scan type	Helical	Contrast concentration (mgI/ml)	370–400
Kv	120	Contrast volume (ml)	120
MAs	300	Contrast flow rate (ml/s)	3–4
Rotation time (s)	0.6	Hydrosaline solution volume (ml)	40
Collimation (mm)	Arterial phase 0.625/0.75	Hydrosaline solution flow (ml/s)	3–4
	Venous phase 1.2/1.25		
Pitch	0.984	Delay (bolus tracking)	Arterial flow: bolus tracking +12 s portal flow: 70 s delayed phase: 3 min
Reconstruction interval (mm)	1.25		
Slice thickness (mm)	2.5		
Reconstruction filter	Standard		
Scanning direction	Cranio-caudal		
Scanning range	Bleeding zone		

DSA showed a single source of bleeding in 12 cases (40 %), two bleeding vessels in 11 cases (36.6 %), and multiple haemorrhagic foci in the remaining 7 cases (23.4 %). In four patients, we performed complete embolisation of the posterior trunk of the hypogastric artery.

Pre-procedural haemoglobin values ranged from 3.6 to 8.2 g/dl (mean, 5.6 g/dl ±1.7) and haematocrit values between 15–26 % (average, 21.4 % ±6.2).

The time elapsed between the clinical and laboratory suspicion of active bleeding and the embolisation procedure was highly variable and dependent on the severity of the bleeding and the outcome of any conservative approach. The mean interval was 8 h, with range from 30 min in two haemodynamically unstable patients to 36 h in patients with progressive anaemia. The time elapsed between CT angiography and the embolisation procedure was instead solely dependent on the possible time needed to transfer the patient from another hospital, and performing and reading the study: demonstration of any active arterial bleeding on CT was promptly followed by endovascular treatment.

Technical success was obtained in all cases (100 %) (Fig. 2), with a mean procedure time of 52 min (range 24′–78′) and a mean fluoroscopy time of 30 min (range 14′–86′).

A statistically significant reduction in daily transfusion requirements was observed, from a mean pre-treatment value of 7.5 U/day ±3 (range 5–15 U) to a mean post-treatment value of 2.8 U/day (range 1–10 U) ($p < 0.005$).

There were 3/30 post-procedural deaths (<30 days) (10 %): in one case, a patient arrived in the angiography suite in haemodynamic shock (BP < 90 mmHg, massive bleeding continued for some time), whereas the remaining two patients, after optimal embolisation, developed massive bleeding at other extraperitoneal sites (abdominal wall and pre-sacral region, respectively) within 48 h after treatment associated with cardiocirculatory failure, which precluded additional embolisation.

There were no significant differences between clinical outcome (death/survival) and time interval from clinical suspicion (average time to procedure in deceased patients was 7 h 52′ vs. 8 h 7′ in survivors) or between clinical outcome and severity of anaemia (mean Hb of deceased patients was 5.3 g/dl vs. mean 5.6 g/dl of survivors).

None of the patients treated was subjected to subsequent decompressive laparotomy.

Overall mortality at 6 and 12 months was 14.8 % (4/27 patients) and 26 % (6/23 patients), respectively, due in all cases to reasons not directly related to the haemorrhage (myocardial infarction, cancer, stroke, sepsis and acute respiratory distress).

Discussion

Extraperitoneal bleeding in most cases is associated with a traumatic event or iatrogenic injury [23–25], but it can also be observed in the case of ruptured aneurysm of the abdominal aorta or in the course of neoplastic or

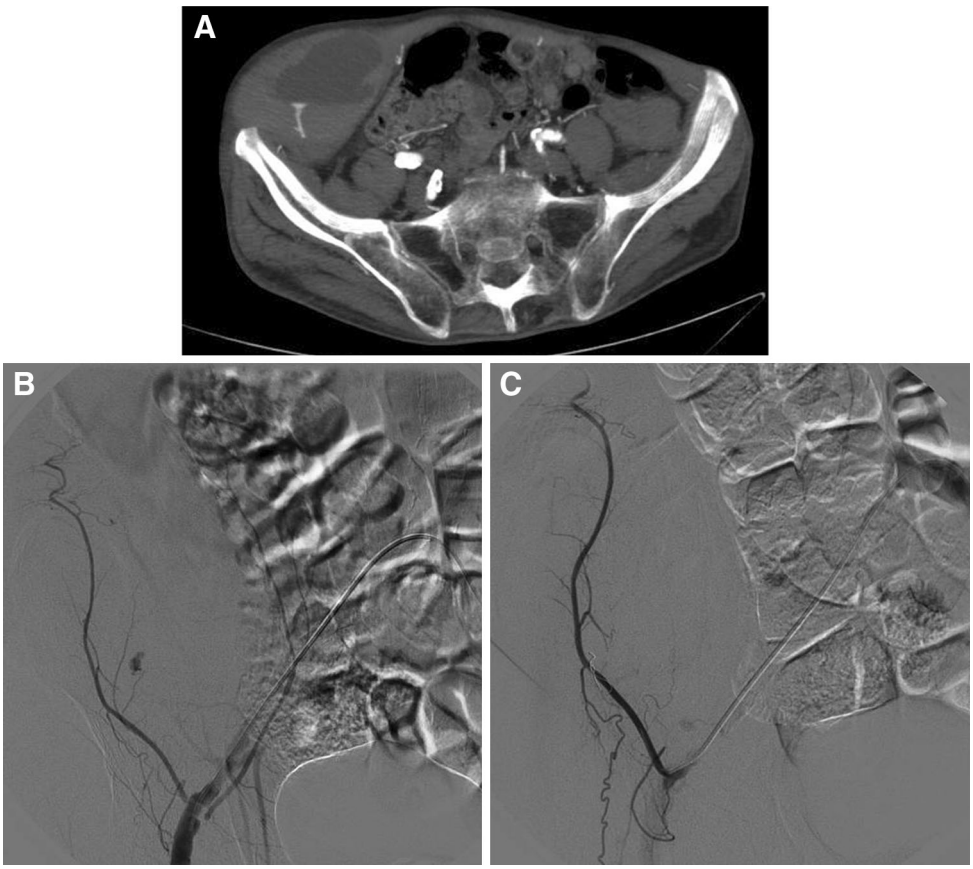

Fig. 1 **a–c** Embolisation of spontaneous extraperitoneal haemorrhage. Preliminary contrast-enhanced CT scan shows the presence of a large haematoma in the anterolateral abdominal wall with active bleeding from the superficial circumflex artery (**a**), successfully treated by transcatheter embolisation with particles and coils (**b**, **c**)

inflammatory diseases; especially, of the kidneys or adrenal glands [26]. A relatively uncommon form of extraperitoneal haemorrhage is spontaneous, not associated with trauma, surgery or underlying diseases [1]. This form is mostly associated with changes of the coagulation state due to bleeding disorders or anticoagulant therapy [27]. In recent years, the incidence of this condition has greatly increased because of anticoagulant therapy and related risks [10]: in our experience, we have observed a progressive increase in the incidence of SEH, with doubling of the cases observed from four cases in 2005 to eight cases in 2010.

Endovascular arterial embolisation is nowadays an established therapeutic option in the management of many haemorrhagic conditions [28–32]. There are numerous requests for embolisation of spontaneously bleeding vessels in patients on oral anticoagulants/heparin: we therefore considered it important to present our experience in terms of clinical and therapeutic management and results.

Not much is known about SEH pathophysiology: supposed risk factors are atherosclerosis of small vessels, minor trauma, occult vascular disease of the kidneys and adrenal glands and immune-mediated microangiopathy induced by heparin [33].

The diagnosis of SEH is essentially based on clinical and laboratory findings; CT, however, plays a key role in identifying the site of bleeding and the blood vessels responsible [20]. In our experience, CT identified the vessels to be embolised, allowing proper treatment planning with maximum intensity projection multiplanar reconstructions. In agreement with the literature [34], in the case of negative CT, DSA has to be considered the first option for a correct diagnostic and therapeutic interpretation.

The first-line treatment of SEH is conservative: suspension, when possible, of anticoagulant therapy, identification and correction of coagulation disorders, fluid infusion and blood transfusion [3, 4].

Endovascular treatment with transcatheter embolisation has become the first choice in SEH management because surgery is limited by the difficulty identifying and treating bleeding vessels the context of a massive haematoma and the risk that surgical manoeuvres may increase bleeding or re-start a tamponaded bleed [30].

The indication for angiography in emergency/urgent settings is established in the presence of persistent anaemia despite adequate transfusion therapy that can be identified in >4 U of packed red blood cells in 24 h or >6 U of packed red blood cells in 48 h [28] and on the basis of the considerable experience gained with the treatment of post-traumatic haemorrhage [37–39].

The diagnostic and therapeutic effectiveness of DSA in the treatment of SEH has been documented by recent

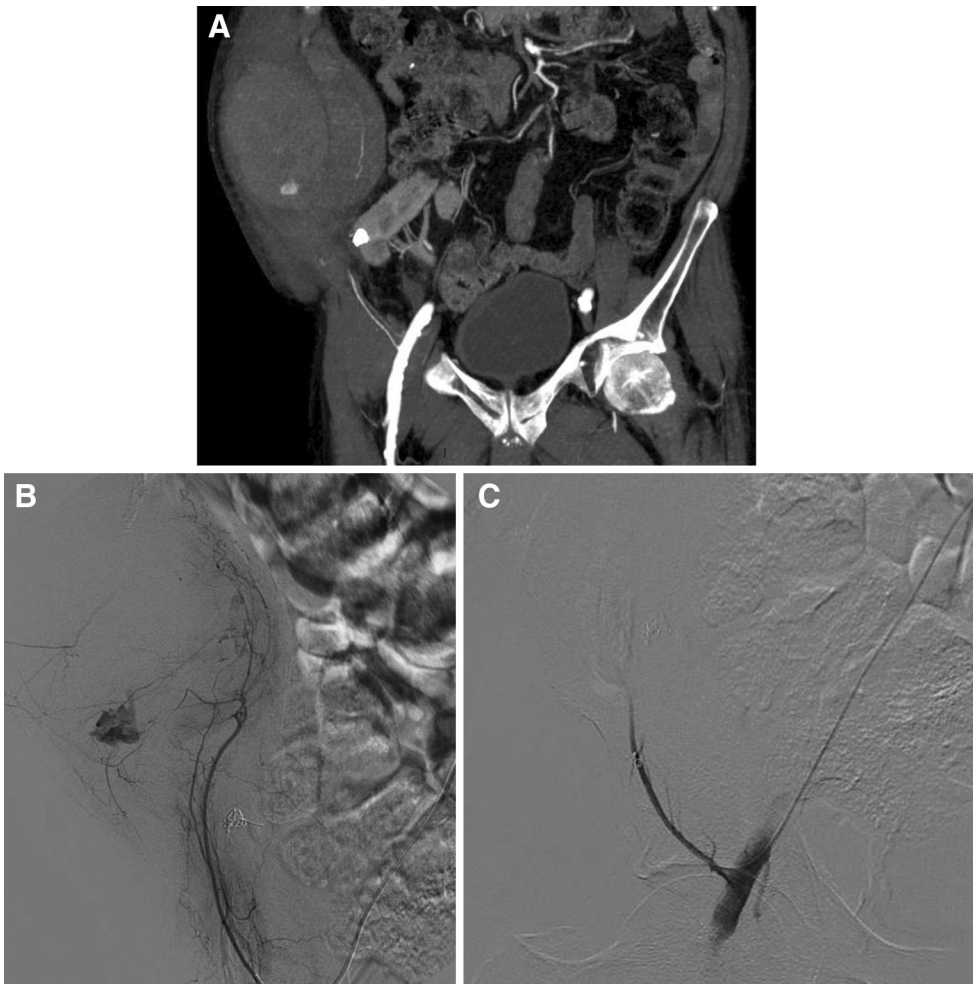

Fig. 2 **a–c** Embolisation of spontaneous extra-peritoneal haemorrhage. The coronal multiplanar reconstruction (MPR) shows active bleeding from the superficial circumflex iliac artery, as confirmed by angiographic examination (**a**, **b**). The endovascular approach with coils and particles allowed appropriate treatment (**c**)

studies [3, 16–21, 27, 33, 40]. In our experience, DSA allowed identification of all cases of bleeding, even in those three cases where CT failed to identify the bleeding vessel. This finding is in agreement with the reports by Zissin et al. [19] and Farrelly et al. [40] and demonstrates that initial CT followed by DSA is indeed the correct diagnostic pathway. DSA is indicated in the case of negative CT examination if required by the patient's clinical condition; in haemodynamically unstable patients, DSA should be performed as the first-choice examination as the time factor is essential in this setting [40].

Treatment modalities depend on the operator's confidence with different materials: in our experience we favoured the use of coils and microparticles as we believe these materials to be more easily handled. Other reports, however, have widely described the use of absorbable gel (Gelfoam, Pharmacia and Upjohn, Kalamazoo, Michigan) with optimal results [40]. Our treatment planning involves micro-catheterisation of the most distal portion of the bleeding vessel and the release of microcoils and particles until complete cessation of contrast leakage. In cases of multiple bleeding vessels and difficult micro-catheterisation, proximal embolisation with larger calibre coils is performed.

In our experience, transcatheter embolisation achieved complete (100 %) technical success. This finding is in agreement with all published studies [16–21], and demonstrates the effectiveness of this therapeutic approach in the management of spontaneous bleeding. The treatment proved effective even in patients who arrived in the angiography suite in haemodynamic shock, and in these patients prompt treatment can be lifesaving. The therapeutic efficacy of DSA was also demonstrated by the statistically significant reduction of transfusion requirements: this result is also consistent with the literature [27] and demonstrates that the technical success of the treatment (cessation of active contrast leakage at final angiography) is associated with clinical success.

Concordance of all published studies on the effectiveness of arterial embolisation in the management of SEH allows us to consider this approach the first-choice treatment option.

Despite a technical success of 100 %, we observed a postoperative mortality of 10 % (3/30 patients). One of

these patients had arrived with substantially compromised cardiovascular condition (haemodynamic shock) and died at 24 h due to multiorgan failure, despite correct embolisation of the bleeding vessels. The other two patients died at 48 h due to massive bleeding from new, previously unaffected sites as the patients failed to reach the angiography suite in time for further treatment. This observation leads us to believe, in agreement with Sharafuddin et al. [33], that spontaneous haemorrhage can be related to a widespread small-vessel disease in coagulated patients which, associated with microtrauma, can produce massive haemorrhagic manifestations.

Mortality at 6 and 12 months was 14.8 % (4/27 patients) and 26 % (6/23 patients), respectively, and not related to haemorrhage or embolic treatment in all cases. This result is consistent with the literature data, documenting a high rate of mortality post-embolisation: Pathi et al. [20] reported that 3/4 patients with spontaneous haemorrhage successfully treated endovascularly, died within 30 days of treatment for reasons not related to embolisation or its complications; Rimola et al. [17] observed that, compared with a complete technical success of embolisation in 12 patients with SEH, three patients died within 8 days and four patients within 30 days of treatment, again for reasons not related to haemorrhage. Finally, in a recently published study, Farrelly et al. [40], in a total of 25 patients with SEH successfully treated with embolisation, observed one postoperative death due to massive bleeding from other sites not previously treated and a 48 % mortality rate at 12 months due to co-morbid conditions. These results indicate that even though the treatment is associated with a high technical success rate and a low postoperative mortality, SEH occurs in patients with heavily compromised general condition and is, therefore, associated with a poor prognosis [40].

In conclusion, on the basis of our experience and the literature data, endovascular treatment of SEH represents the treatment of choice, as it can provide complete therapeutic success. However, adequate and close clinical and therapeutic follow-up is needed to reduce long-term mortality, especially of those patients with heavily compromised clinical condition.

Conflict of interest Giuseppe Guzzardi, Rita Fossaceca, Paolo Cerini, Marco De Bonis, Emanuele Malatesta, Ignazio Divenuto, Mariangela Lombardi, Alessandro Carriero declare no conflict of interest.

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