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CASE REPORT
Coronary heart disease

Spontaneous retroperitoneal haemorrhage post-coronary angioplasty: a case report

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Background Spontaneous retroperitoneal haemorrhage (SRH) is a rare cause of retroperitoneal haemorrhage in patients who are on anticoagulants or antiplatelet agents or both.

Case summary We report here a rare and catastrophic complication of use of anticoagulants and antiplatelet drugs in a case undergoing coronary angioplasty. The patient had multiple coronary risk factors and developed acute myocardial infarction with pulmonary oedema and hypotension during hospitalization for treatment of lower respiratory tract infection and diabetic ketoacidosis. He underwent successful angioplasty of the culprit vessel but later developed hypotension attributable to retroperitoneal haemorrhage. No bleeding site was identified despite extensive evaluation of the aorta and iliac vessels.

Discussion A diagnosis of SRH is considered when a patient on anticoagulants or antiplatelet drugs develops retroperitoneal haemorrhage without any specific identifiable site of bleeding in the retroperitoneum. Diffuse vasculopathy and atherosclerosis or vasculitis of the small vessels in the retroperitoneum may result in rupture of the most friable vessels and result in bleeding. Intense cough, forceful vomiting or sneezing may also be responsible for traumatizing the vessels and resulting in bleeding. Most cases recover with conservative management but some may benefit from interventional occlusion of the leak or surgical decompression in cases of abdominal compartment syndrome.

Keywords Spontaneous retroperitoneal haemorrhage • Post-angioplasty • Anticoagulants • Antiplatelet drugs • Case report

Learning points

- Unexplained drop in haemoglobin and blood pressure–post-percutaneous coronary intervention must prompt the physician to search for the haemorrhagic complications like spontaneous retroperitoneal haemorrhage.
- Co-administration of anticoagulants and antiplatelet agents may result in spontaneous diffuse bleeding from retroperitoneal vascular beds.
- Non-contrast-computed tomography abdomen delineates retroperitoneal haemorrhage well.

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Introduction

Retroperitoneal haemorrhage is a serious complication of coronary interventions that occurs most commonly due to vascular trauma during the manipulation of hardware. Spontaneous retroperitoneal haemorrhage (SRH) is a rare cause of retroperitoneal haemorrhage that has been described in a few cases who are on anticoagulants or antiplatelet agents or both.[1] It is hypothesized that vasculopathy or unrecognized trauma to small vessels in the retroperitoneum on the background of anticoagulation or antiplatelet agents may result in bleeding in the retroperitoneum.[2,3]

Timeline

Day 1	Admission with fever and breathlessness, diagnosed as septicaemia with diabetic ketoacidosis, paroxysmal atrial fibrillation with fast ventricular rate
Day 2	Managed with antibiotics, insulin, and antipyretics
Day 3	Severe chest pain and breathlessness and hypotension.
9 a.m.	Managed as acute pulmonary oedema and acute inferior myocardial infarction with junctional rhythm. Endotracheal intubation done.
11 a.m.	Shifted to cath lab for coronary angiography and revascularization
1:45 a.m.	Shifted back to coronary care unit
2:30 p.m.	Inotropes tapered off, cardiac rhythm stable
4 p.m.	Hypotension noted, blood pressure 80–90/60 mmHg, given fluid challenge and low dose inotropes started. No electrocardiogram changes. ABG revealed drop in haemoglobin and a rise in lactates.
5 p.m.	Dose of inotropes increased, bedside ultrasound done to rule out significant groin/or abdominal haematoma. Blood transfusions started.
10:30 p.m.	Computed tomography scan of the abdomen and aortic angiogram revealed retroperitoneal haemorrhage. Surgical intervention ruled out.
Day 4	Multiple blood transfusion and colloids given to manage hypovolaemic shock.
Day 5 9 a.m.	Declared dead

Case presentation

A 66-year-old man presented to the department of internal medicine with history of fever, cough, and difficulty in breathing and exertional chest pain. He had past history of hypertension, diabetes (diabetic nephropathy, estimated glomerular filtration rate 45 mL/min), and smoking. On admission, he was febrile and breathless. The chest had bilateral rhonchi and basal crepts. His blood sugar was 465 mg/dL and the ketone levels were high in urine. Arterial blood gas estimation showed metabolic acidosis. Other blood tests are summarized in *Table 1*. Chest X-ray showed increased bronchovascular markings in both the lower lobes (Supplementary material online, *Figure S1*). Computed tomography (CT) scan chest showed bilateral pneumonic consolidation in the lower lobes (Supplementary material online, *Figure S2*). He was managed with insulin, antibiotics, intravenous fluids, and the ketoacidosis was corrected. Reverse transcription–polymerase chain reaction for COVID-19 was negative.

On Day 3 of admission, he had severe chest pain and breathlessness with hypotension and the electrocardiogram (EKG) showed junctional rhythm with ST-elevation in inferior and lateral leads (*Figure 1*). He was noted to be in acute left ventricular failure and required mechanical ventilation to maintain oxygen levels. Bedside echocardiogram showed that the patient had moderate to severe mitral regurgitation and severe left ventricular systolic dysfunction. He was given ticagrelor 180 mg and aspirin 325 mg orally and started on inotropes and diuretics and shifted to cardiac cath lab for primary angioplasty. The procedure was performed through the right femoral route (6F artery and 7F vein accessed). Additional 5F arterial sheath was introduced through the left side in preparation for need of emergent left ventricular support device. Coronary angiogram revealed a chronic total occlusion of right coronary artery. There was 30–40% stenosis in distal left main and 80% stenosis of osteoproximal left circumflex artery (*Video 1*). The left anterior descending artery was free of significant disease. Percutaneous coronary intervention was done and a stent was implanted from left main to left circumflex artery with good result (*Video 2*). ST-elevation settled in the electrocardiogram (*Figure 2*). A total of 7000 IU of unfractionated heparin was given to maintain an activated coagulation time between 250 and 300 s during the procedure. The procedure was complicated by sustained ventricular tachycardia and paroxysms of atrial fibrillation with fast ventricular rate requiring multiple DC shocks and intravenous cordarone (Supplementary material online, *Figure S3*). The patient improved within a few hours and haemodynamics and arrhythmia settled down. Inotropes were discontinued.

Later in the evening, the patient started having hypotension (blood pressure 80–90/60 mmHg and heart rate -110/min, sinus rhythm). The EKG was unchanged. There was a small groin haematoma and no tenderness was noted in the flanks. Intravenous fluids and colloids were given and when the haemodynamics didn't improve, inotropes were added. A bedside ultrasound showed a small haematoma in the groin but no blood was seen in the abdomen sufficient to explain the hypotension. Blood gases showed rising lactate levels and a 4.6 gm/dL fall in haemoglobin (11.7–7.1 g/dL). Six units of blood, four units of fresh frozen plasma were given over next 24 h. The abdominal girth increased gradually pointing towards retroperitoneal haemorrhage. After stabilization, CT scan of the abdomen was performed which revealed a massive retroperitoneal bleed into the left psoas and the left iliacus muscle (*Figure 3*). A CT aortogram was also performed which failed to show any bleeder. A peripheral angiogram was also performed of the left iliac arteries and no bleeder was identified (*Video 3*). Antiplatelet drugs were discontinued. Laboratory results showed that the platelet count and prothrombin time were normal (196 000 cells/mm^3 and 14.2 s, respectively). The activated partial thromboplastin time was marginally above normal (58.3 s; control

Table 1 Summary of biochemical and haematological parameters

Parameter	Day 1	Day 2	Day 3	Day 4	Day 5	Reference value
HB (g/dL)	12.1	11.7	7.1[a]	13.0[b]	7.8	13–17 (g/dL)
TLC (cells/cumm)	27 520	25 850	34 900	32 290	30 880	4000–10 000 (cells/cumm)
Platelet count (cells/cumm)	576 000	49 300	400 000	272 000	196 000	150 000–410 000 (cells/cumm)
PCV (%)	35.7	29.4	30.1	37.6	24.2	40–50%
Serum urea (mg/dL)	79	87	87	96	129	10–45 (mg/dL)
Serum creatinine (mg/dL)	2.6	2.5	1.9	2.0	3.4	0.2–1.2 (mg/dL)
Sodium (mEq/L)	127	133	139	147	155	135–148 (mEq/L)
Potassium (mEq/L)	6.9	4.7	4.3	3.6	5.3	3.5–5.3 (mEq/L)
INR (s)				1.4	1.4	0.93–1.46 (s)
PT (s)				16.8	16.7	11.5–14.6 (s)
APTT (s)				62.2	58.3	26–40 (s) (control: 33.2 s)
Procalcitonin (ng/mL)			20.17			0.5–2.0 ng/mL
CRP (mg/dL)	19.27					0–0.5(mg/dL)
D-Dimer (ng/mL)	14200					0.0–200 (ng/mL)
Ferritin (ng/mL)	1412					20–250 (ng/mL)
Interleukin-6 (pg/mL)	130.4					0–6.99 (pg/mL)

[a]Evening sample few hours after bleeding.
[b]Post blood transfusion.

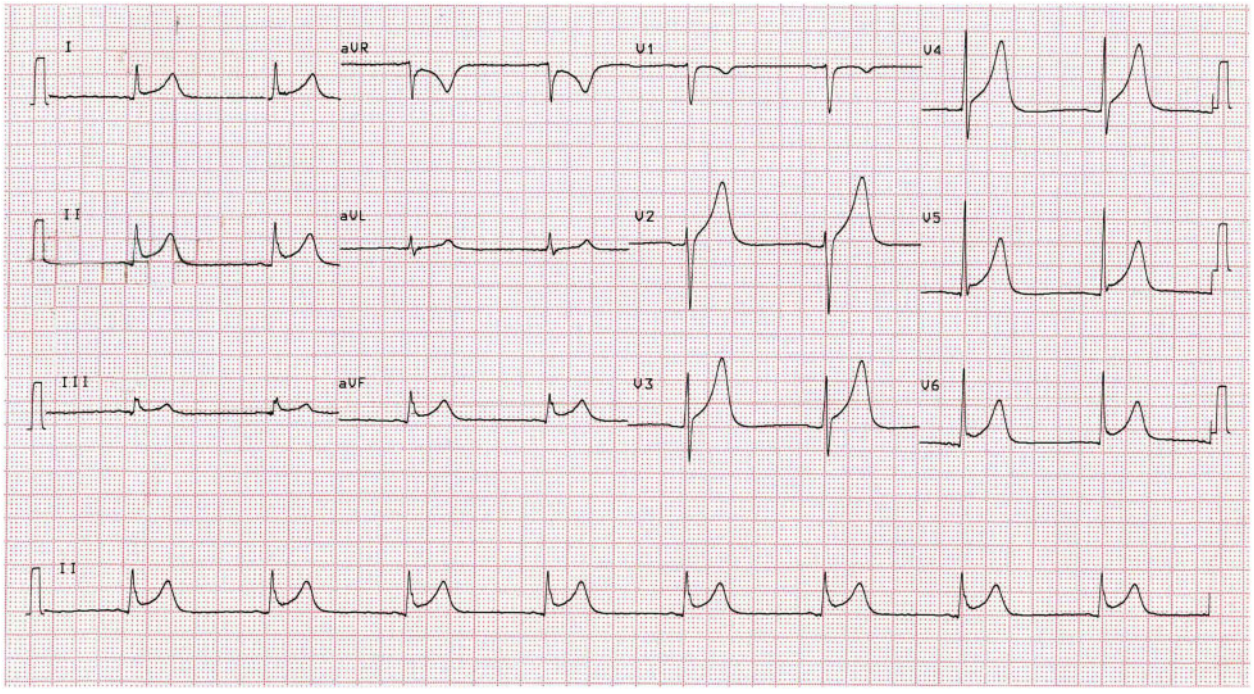


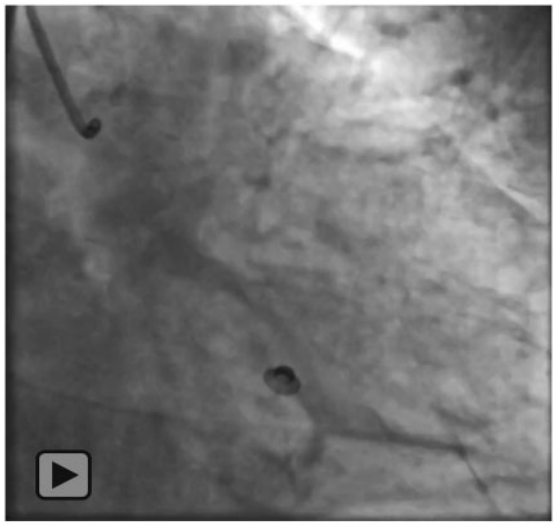
Figure 1 Junctional rhythm with ST-elevation in inferior and lateral leads.

34.2 s). The surgeons ruled out any surgical intervention because there was no specific site of bleeding. We continued to replace blood and fresh frozen plasma. Despite best of efforts, the bleeding could not be controlled and the patient died.

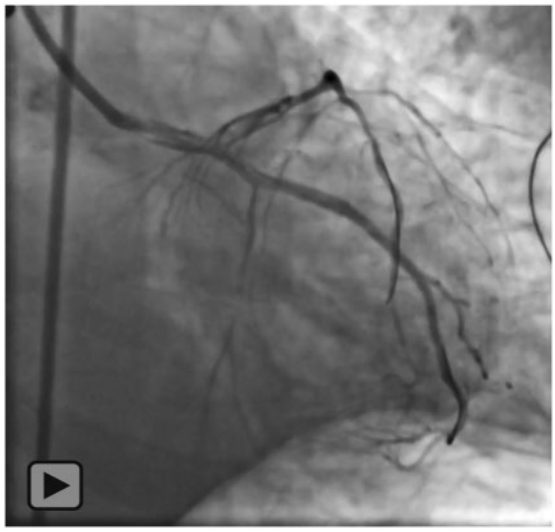
Discussion

SRH is a rare cause of retroperitoneal haemorrhage. Only a few cases have been reported in the literature and in most, the patients were on anticoagulants or antiplatelet drugs.[1] Diffuse vasculopathy and

atherosclerosis or vasculitis of the small vessels in the retroperitoneum may result in rupture of the most friable vessels and result in diffuse bleeding. Intense cough, forceful vomiting or sneezing may also be responsible for traumatizing the vessels and resulting in bleeding.[2,3] Most of the cases respond to conservative treatment with blood products (fresh frozen plasma, whole blood, and platelets) and volume resuscitation. Very few, with a major identifiable source of leak, may benefit from interventional coil embolization of the culprit vessel or those with abdominal compartment syndrome may need surgical decompression.[4,5] In our case, no specific bleeder was identifiable. The massive haemorrhage noted on the CT scan in the psoas and iliacus muscle was totally inexplicable by the intervention



Video 1 Angiogram showing 30-40% stenosis in distal left main and 80% stenosis of osteoproximal left circumflex artery.



Video 2 Angiogram showing the result after stenting of the distal left main to the left circumflex artery.

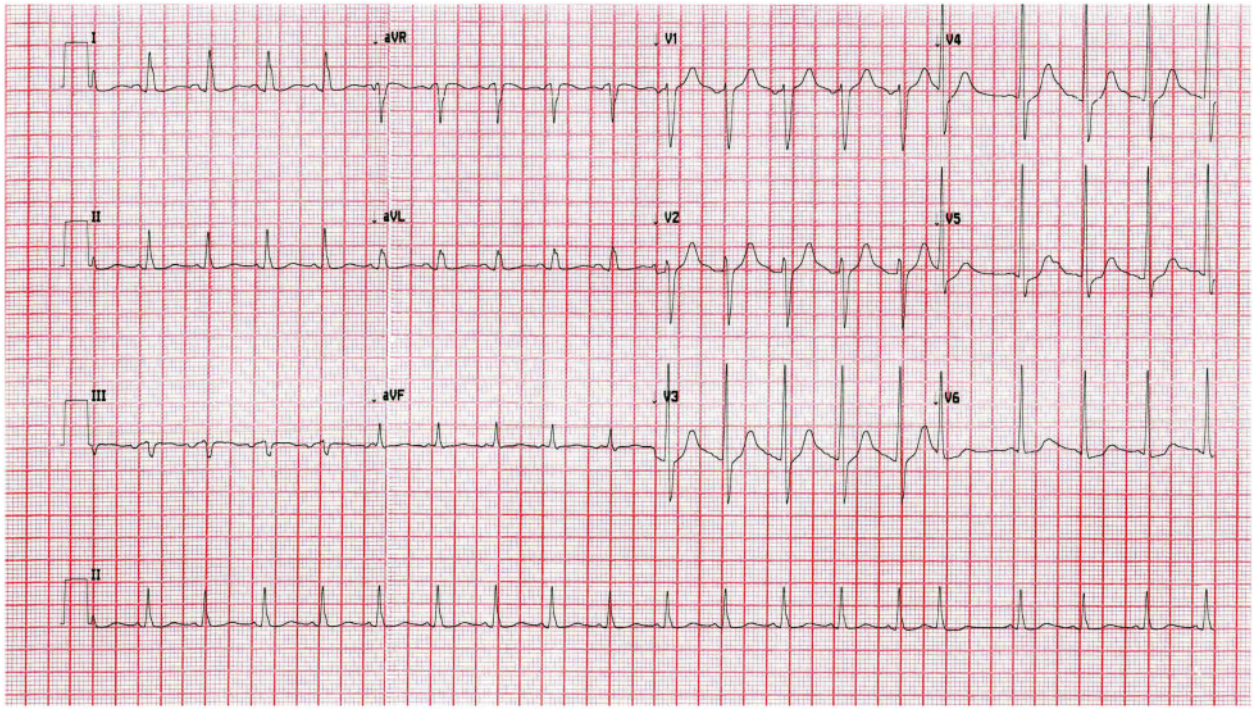


Figure 2 Post-intervention electrocardiogram: ST-segments settled.

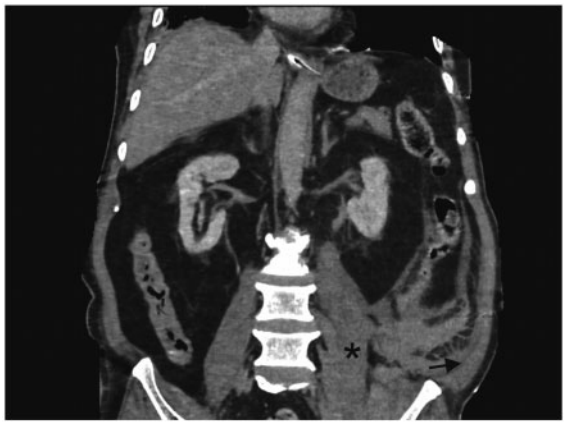
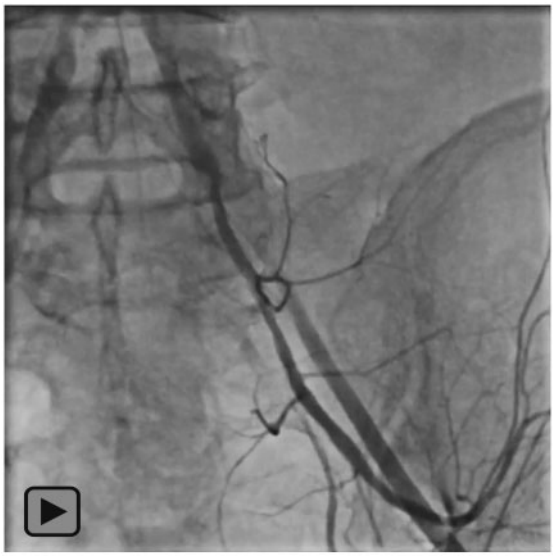


Figure 3 Contrast-enhanced computed tomography (coronal section). Asterisk indicates bulky left psoas muscle with haematoma extending into the left iliacus muscle (arrow).



Video 3 A peripheral angiogram of the left iliac arteries showing no extravasation of contrast anywhere.

performed. However, since the patient had sepsis and diabetic nephropathy, qualitative platelet dysfunction aggravated by administration of anticoagulants and antiplatelet agents may have triggered the spontaneous haemorrhage. Additionally, microvascular trauma could have been caused by the jerky motion of the torso during cardioversion in our case.

Conclusions

SRH is a rare and catastrophic complication of anticoagulant and antiplatelet drugs of unclear aetiology. The risk of SRH increases in patients of old age, anaemia, sepsis, nephropathy, or hepatic disease. A high index of suspicion is needed to diagnose retroperitoneal haemorrhage post-revascularization. Non-contrast CT scan abdomen is the best modality to diagnose SRH.

Lead author biography

Dr Anshul Kumar Jain is an interventional cardiologist working in New Delhi, India. His major areas of interest include coronary interventions and prevention of coronary artery disease in young Indians.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient's next of kin in line with COPE guidance.

Conflict of interest: none declared.

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