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CASE REPORT

Massive, Spontaneous Ruptured Renal Subcapsular Haematoma Following Thrombolytic Therapy in Acute ST-Elevation Myocardial Infarction

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ABSTRACT

Thrombolytic therapy remains widely used in majority of developing countries, where delivery of primary percutaneous coronary intervention (PCI) remains a challenge. Unfortunately, complications following such therapy remains prominent, predominantly bleeding-related problems. We present a rare case of massive renal subcapsular haemorrhage and hematoma following thrombolytic therapy. A 61-year old gentleman presented following an episode of chest pain due to acute ST-elevation myocardial infarction. Due to potential delays in obtaining PCI, the patient was counselled for thrombolysis using streptokinase which he had consented to. Unfortunately, within 36 hours of admission, he developed abdominal pain, haematuria, hypotension and altered mental status, associated with acute drops in haemoglobin levels. Following initial resuscitation efforts, a Computed Tomography scan of the abdomen was performed revealing a massive renal subcapsular hematoma, likely secondary to previous thrombolysis. Renal subcapsular hematoma can either be spontaneous or iatrogenic, the latter often due to coexisting renal-based neoplasm or vasculitidies. Iatrogenic causes include trauma, following renal biopsies or anticoagulation therapy amongst a few others. Iatrogenic renal subcapsular haemorrhage and hematoma formation are rare following thrombolysis. Our literature search revealed only one other similar case, although this was following administration of recombinant Tissue Plasminogen Activator in a case of acute ischaemic cerebrovascular accident. This case highlights the complexity in management, following the findings in terms of need for cessation of dual antiplatelet therapy and timing for PCI and stent selection.

KEYWORDS: Renal subcapsular haematoma, thrombolytic therapy, ECG changes, myocardial infarction.

INTRODUCTION

Thrombolytic therapy is indicated in cases of Acute ST-Elevation Myocardial Infarction (STEMI) when primary percutaneous coronary intervention (PCI) cannot be delivered within a recommended time frame [1,2]. Its use is increasingly rare in developed countries with well-established STEMI networks [3]. However, developing countries, including Malaysia, still rely on thrombolytics as a means of urgent reperfusion. We report a rare case of massive subcapsular haemorrhage and hematoma formation following thrombolytic therapy and how we had subsequently managed this complex case.

CASE PRESENTATION

A 61-year old gentleman presented to the emergency department following sudden onset of chest heaviness an hour prior. He suffered from hypertension and dyslipidaemia and was a chronic smoker of 30 pack years. An electrocardiogram revealed ST-segment elevation in leads II, III and aVF, with reciprocal ST-segment depression in lead I, aVL and V3 to V6, consistent with acute, inferior ST-elevation Myocardial Infarction, of Kilip 1 classification. The patient was counselled for reperfusion therapy in the form of thrombolysis, for which he consented to. Unfortunately, at the time, primary percutaneous intervention could not be offered due to lack of access within proximity of the hospital.

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A total of 1.5 Megaunits of intravenous infusion streptokinase was administered over 1 hour, with no immediate complications. He was then admitted to the coronary care unit for closer monitoring with subsequently reduction in ST-segment elevation by more than 50% with improvement in symptoms, signaling successful reperfusion.

The patient, however, started complaining of right hypogastric pain, 20 hours from the thrombolysis, which was associated with frank haematuria. There was no other evidence of bleeding elsewhere. The patient denied any trauma prior to presenting to hospital, and throughout his hospital stay. It was initially assumed that the patient was suffering from gastric irritation and haematuria following dual antiplatelet (DAPT) use, which were immediately ceased. Unfortunately, 16 hours later, the patient became drowsy (Glasgow Coma Scale of 11 – Eye 3, Verbal 3 and Motor 5) and hypotensive with a blood pressure of 98/65 mmHg. Urgent blood investigations revealed a significant drop in haemoglobin concentration (10.5 g/dL to 7.6 g/dL) with worsening urea and creatinine levels (Table 1).

Table 1 Blood Investigations Performed in the First 48 Hours of Admission

Duration from Admission	Admission	20 Hours	36 Hours	48 Hours
Haemoglobin (g/dL)	11.9	10.5	7.6	4.6
White Cell Count (x10^{9}L)	23.7	25.3	30.6	15.3
Platelet (x10^{9}L)	274	208	194	197
Prothrombin Time (s)	15.9	N/P	N/P	13.1
INR	1.42			1.16
Activated Partial Thromboplastin Time (s)	27			24.5
Sodium (mmol/L)	N/P	137	134	148
Pottasium (mmol/L)		3.8	3.7	3.7
Urea (mmol/L)		9.8	29.0	16.7
Creatinine (mmol/L)		123	322	154

N/P = Not Performed

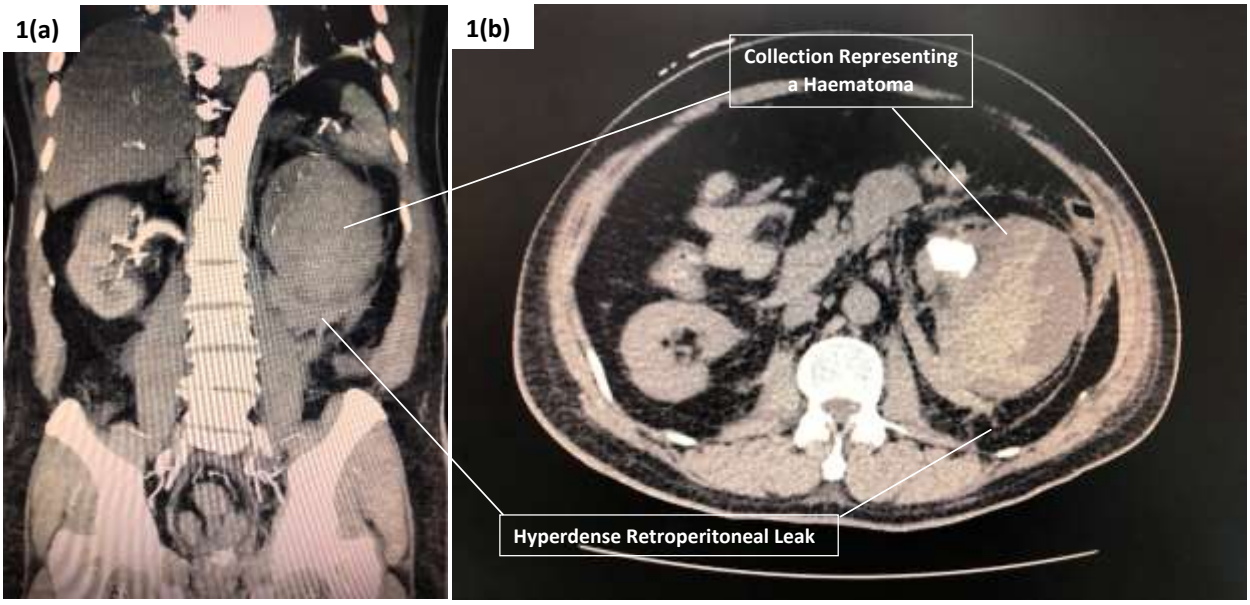

Figure 1 Computed Tomography Imaging of the Abdomen on (a) coronal & (b) transverse planes, revealing a well-defined heterogenous hypodense collection with hyperdense layering of different attenuation within, at the left renal subcapsular measuring approximately 10.4 x 6.8 x 12.7cm, with hyperdense contents leaking out into the left posterior pararenal space and retroperitoneum. Findings are consistent with a renal subcapsular hematoma with evidence of retroperitoneal leak.

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Packed red cell transfusion was administered and DAPT was ceased. Both an urgent Computed Tomography (CT) scan of the head and abdomen was performed in view of the history. The former was unremarkable for any acute intracranial bleeds. The latter, however, revealed a massive right sided ruptured renal subcapsular haematoma, with evidence of leak into the retroperitoneal space. There was no evidence of concomitant masses to suggest pre-existing neoplasm (Figure 1).

An urgent urology consult was obtained following this, but due to the recent cardiac event, surgical intervention was deemed too high a risk at the time. Supportive management in intensive care was recommended with further reversal of coagulopathies and blood loss as required. Unfortunately, due to continuous blood loss, the patient succumbed to the complication within 48 hours of admission.

DISCUSSION

Our national NCVD database showed 69.2% of STEMI patients receiving thrombolytics as an initial reperfusion therapy, more so (81%) in non-PCI centres with very few (<0.1%) being transferred to PCI-capable centre for primary PCI [4]. The shift towards recommending primary PCI over thrombolysis in majority of guidelines stems from both the efficacy of the former and the reduced efficacy (when administered late) in the latter [5-7]. Furthermore, significant complications were associated with thrombolysis the commonest being intracranial haemorrhages [8,9].

Renal subcapsular hematoma can either be spontaneous or iatrogenic, the latter often due to coexisting renal-based neoplasm or vasculitidies [10]. Iatrogenic causes include trauma, following renal biopsies or anticoagulation therapy amongst a few others. Iatrogenic renal subcapsular haemorrhage and hematoma formation are rare following thrombolysis. Our literature search revealed only one other similar case, although this was following administration of recombinant Tissue Plasminogen Activator in a case of acute ischaemic cerebrovascular accident [11]. In the context of percutaneous intervention, there have been reports of renal hematoma following percutaneous transluminal stent placement in the renal artery (via direct perforation) and percutaneous coronary intervention via femoral-access (via plaque dislodgement at the aorto-renal junction, following catheter or guidewire passage, lacerating the aorta and dissecting into the perirenal space) [10,12,13].

Clinical presentation includes that of Lenk's triad (acute flank pain, tenderness, and internal bleeding), which our patient suffered from within hours of streptokinase administration [14]. Differentials to consider include other causes of acute abdomen, including a perforated viscus, retroperitoneal haemorrhage or a dissecting aneurysm, all of which would benefit from CT imaging of the abdomen as performed in our patient.

This case posed a challenge as the decision to subsequently perform a cardiac angiography and percutaneous intervention was delayed in view of active bleeding. Traditionally, patient deemed at risk of bleeding complications would be considered for bare-metal stent (BMS) deployment over DES, to reduce prolonged DAPT commitment. However, following recent evidence from the LEADERS-FREE and a recently presented and soon to be published LEADERS-FREE II trials, BioFreedom™ Polymer-Free DES have shown superiority in terms of bleeding and thrombotic risks, over BMS and which would have proceeded if the patient had survived the acute deterioration instead [15].

CONCLUSION

Although bleeding is a common complication following therapy for acute coronary syndrome, renal subcapsular haemorrhage remains relatively rare. Early recognition, clinically and radiographically, may be lifesaving and helps guide subsequent steps in management including selection of stent for deployment, as seen in our patient.

Conflict of Interest

Authors declare none

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