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Case Report

Two cases of atraumatic adrenal hemorrhage: A review of active management, conservative management, and challenges faced ☆,☆☆

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ABSTRACT

Adrenal hemorrhage (AH) is an uncommon and potentially disastrous affliction that carries an accepted mortality risk of 15%. Variable symptomatology can cause a diagnostic dilemma and may be missed. We present 2 cases of right-sided AH; both cases were initially presumed to be renal colic. Case 1 was an 86-year-old gentleman, presenting with right flank pain found to have a right-sided atraumatic AH. He presented with hemorrhagic shock, requiring angioembolization of the bleeding vessel. Case 2 was a 62-year-old gentleman who presented with right flank pain and was found to have a right-sided atraumatic AH. He was hemodynamically stable and successfully managed conservatively. Adrenal hemorrhage is a potentially fatal affliction that may be missed. CT scans are the recommended imaging modality during an acute presentation due to wider availability and fast assessment. We demonstrate a hemodynamically stable patient managed with a 'watch and wait' approach and an unstable patient managed with resuscitation followed by urgent angioembolization.

Background

The adrenal glands lay overlooked since Galen's description of accessory renal tissue, until Roman Anatomist; Bartholomeus Eustachius (1520-1574) proverbially brought its existence back to the medical forum. The works of Addison (1849) and Brown-Séquard (1856) cemented the vital roles that adrenals play in the sustenance of life. Less than a decade later Canton (1863) recorded apoplexy of the supra-renal capsule [1].

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Adrenal hemorrhage (AH) has an overall accepted mortality rate of 15% [2]. Prior to the widespread use of CT scans, AH would almost exclusively be found at autopsy with an incidence of 0.14% to 1.8%, with some studies finding incidence as high as 25% [2,3]. This leads us to believe AH is a potentially fatal disease that can be missed and is not routinely suspected.

The rich arterial supply to the adrenal gland increases its proclivity to bleeding; supplied superiorly by branches of the inferior phrenic artery, medially by an adrenal branch of the abdominal aorta, and inferiorly by branches of the renal artery. From these 3 main arteries, up to 60 smaller branches can supply each gland [3].

The high volume of arterial inflow into the cortex and medulla with relatively few venous channels creates arterial congestion, a 'vascular dam' of sorts. Therefore, increases in venous pressure, due to trauma (80% of AH cases), sepsis, or vasoconstriction during shock are theorized to overload the capillary network [4–6]. A 25-year study by Vella et al. identified 141 patients with AH, 56 cases were due to a complication of sepsis, 28 cases were detected with an adrenal incidentaloma, 16 cases were spontaneous AH with bleeding into the abdominal cavity, and 3 cases linked with anticoagulant usage.

Presenting symptomatology is vague; AH may present asymptomatically or with generalized abdominal pain, loin pain, or flank pain. Non-specific symptoms such as lethargy and weakness add to the diagnostic dilemma.

We describe 2 cases of a unilateral right-sided, atraumatic adrenal hemorrhage (AH), both were initially presumed cases of renal colic. Our first case represented with catastrophic hemorrhagic shock in the setting of rivaroxaban use and the other case as suspected adrenal malignancy.

Case report 1

An 86-year-old male presented to his primary career with right flank pain radiating to their right groin with worsening exercise tolerance from 1 kilometer to 250 meters over 2 weeks. His other comorbidities included a previous splenic infarction of unknown etiology, stable angina, atrial fibrillation requiring rivaroxaban for the last 3 years, and the excision of a suspected melanoma.

His GP had arranged a CT Renal (non-contrast-enhanced CT) as an outpatient with the presumed diagnosis of renal colic (Figs. 1A and C). This identified 2 suprarenal soft tissue masses (12 and 5 cm) on his right side, with a likely diagnosis of metastases or lymphoma.

He was urgently referred to the emergency department where a 5 x 5 cm patch of right flank bruising was identified on clinical examination. His heart rate (HR) was 78-110 beats/min (in atrial fibrillation) and had an elevated blood pressure of 172/70 mmHg. His abdomen was soft and nontender. His hemoglobin (Hb) was 104 g/L (Ref 130-180), white cell count was normal at 9 × 10^9/L (Ref 4.0-11.0), C-reactive protein (CRP) was elevated at 105 mg/L (Ref <1). Coagulation profile, kidney function, liver function test (LFTs), and lactate level were unremarkable. His serum sodium was low at 128 mmol/L (Ref. 135-147), serum potassium was normal at 4.3 mmol/L (Ref. 3.5-4.5), and normal serum glucose of 7.7 mmol/L (Ref. 4.0-7.8 mmol/L).

The CT of his abdomen revealed bleeding into his right suprarenal gland with an associated large-sized right retroperitoneal hematoma (120 × 90 × 83 mm) (Figs. 1B and D). Multidisciplinary consultation with hematology, general surgery, and interventional radiology culminated in a 'watch and wait' management plan. His anticoagulants were withheld and did not require steroid support. The patient remained hemodynamically and biochemically stable over the next 5 days before his discharge home with a plan for an outpatient CT adrenal scan, serum, and urine biochemical evaluation of adrenal function.

(A) 24-hour urine collection; Metanephrines, Normetanephrine, Total Vanillylmandelic acid (VMA); Catecholamines, total Cortisol, urinary aldosterone
(B) Serum; Catecholamines, fractionated Metanephrines, Dehydroepiandrosterone sulfate (DHEA), Renin, a Dexamethasone suppression test (DMST), serial blood glucose monitoring, synthetic ACTH testing

On discharge, the patient was advised to withhold his anticoagulation till his hematology outpatient appointment. With regards to these outpatient tests, he was only able to complete the DMST portion; a baseline serum cortisol of 657 nmol/L (Ref. 145-619), the morning after a 1mg Dexamethasone dose revealed a serum cortisol as; 0850 h 617 nmol/L (Ref <50).

Ten days after discharge and 31 hours following his DMST, he was readmitted with worsening right flank pain, abdominal distension, and hemodynamic instability. The patient was found to have self-commenced rivaroxaban following discharge. The Hb level was 85 g/L (Ref 130-180), WCC and CRP were elevated at 22.6 × 10^9/L (Ref 4.0-11.0) and 139 mg/L respectively (Ref <1). The platelet level was normal at 345 × 10^9/L (Ref 150-450). International normalized ratio (INR) was elevated at 2.7 (Ref <1.3), the estimated glomerular filtration rate was 70 mL/min/1.73 m^2 (Ref >60) and Cr was 86 micromol/L (Ref 60-110). Serum sodium was 127 mmol/L (Ref 135-147), potassium 4.5 mmol/L (Ref 3.5-4.5) His respiratory rate (RR) was 32 breaths/min, HR and BP were 120 beats/min and 65/36 mmHg respectively. He responded favorably to intravenous fluids, 1 unit of packed red blood cells, Prothrombinex administered at a dose of 30 units/kg and commenced on 4 mcg/min of adrenaline.

He underwent urgent angioembolization to control an active bleed from the inferior adrenal artery seen on CT angiography (Fig. 2). Histoacryl glue embolization of the right inferior adrenal artery was successful and no other bleeding vessels were found (Fig. 3). His adrenaline infusion was subsequently weaned off.

The patient did not suffer from any further episodes of adrenal bleeding, and his hemoglobin level remained stable at 100 g/L post-treatment. He underwent 25 days of rehabilitation postembolization before being discharged home. Unfortunately, the patient succumbed to a stroke 8 months after his adrenal hemorrhage, following the cessation of anticoagulants.

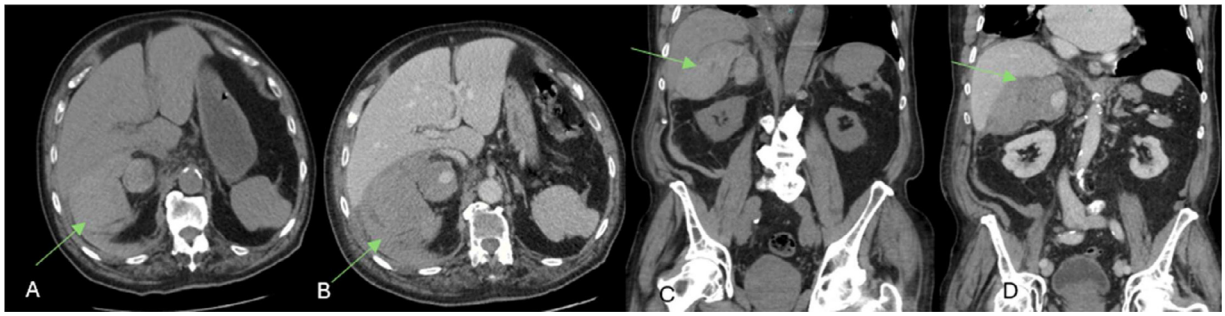

Fig. 1 – Abdominal and pelvic computed tomography (CT) scan of an 86-year-old gentleman in Case 1 (A) green arrow pointing towards the adrenal hemorrhage. The maximal size of the abnormality was 120 x 90 x 83 transverse, craniocaudal, and anteroposterior measurements respectively. (A) axial view of primary career organized CT KUB (Kidney, Ureter, and Bladder); Mean density of ovoid lesion; 59.7 Hounsfield Units (HU). (B) axial view of repeat CT AP with contrast enhancement, organized in Emergency. Ovoid density was identified again; Mean density 59.9 HU, with an area of hyperdensity on the medial side of ovoid mass; 116 HU. (C) coronal view of primary career organized CT KUB, HU as in (A). (D) coronal view of repeat CT AP with contrast enhancement organized in Emergency. HU as in (B).

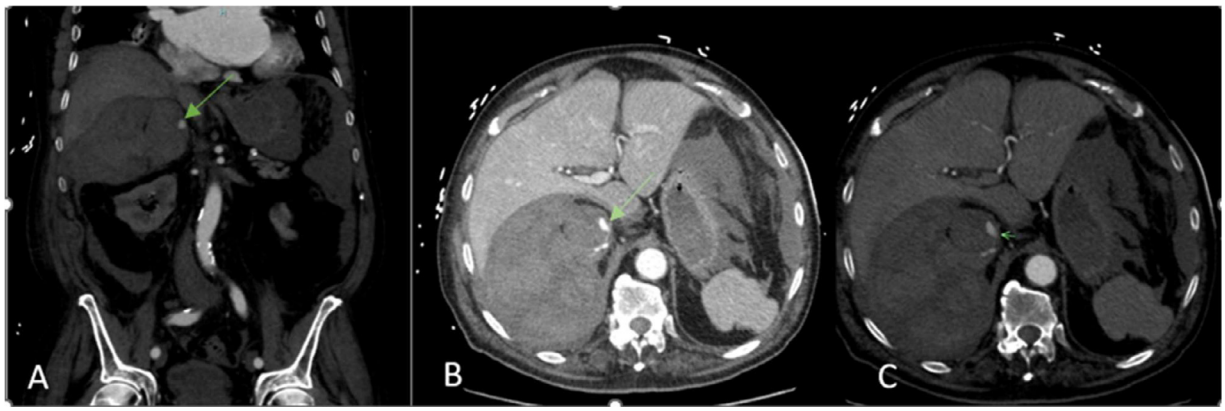

Fig. 2 – Abdominal and pelvic CT of the 86-year-old gentleman on representation (Case 1) showing. (A) CT Angiogram of the Abdomen, identifying focal hyperattenuation in the right adrenal hematoma consistent with active bleeding. (Green arrow) The mean density of ovoid mass is 52.7 HU with medial hyperdense lesion; the mean density is 180 HU. (B) Contrast-enhanced CT of the Abdomen in the late arterial phase. Hyperdense focus is consistent with active bleeding. (Green arrow) The mean density of ovoid mass is 52 HU and medial hyperdensity is 205 HU. (C) CT Angiogram of Abdomen with arterial phase, Hyperattenuating focus consistent with active bleed. (Green arrow) The mean density of ovoid mass is 51.3 HU and medial hyperdensity 162 HU.

Case report 2

A 63-year-old male presented to the emergency department with a 1-day history of acute onset right-sided flank pain, nausea, vomiting, and diaphoresis. His comorbidities included hypertension, hypercholesterolemia, type 2 diabetes mellitus, and a previous laparoscopic cholecystectomy. Abdominal examination revealed a soft but distended abdomen with evidence of bruising and tenderness on the right flank. The HR and BP were 84 beats/min and 180/90 mmHg respectively. His Hb was 121 g/L (Ref 130-180), WCC 12 × 10^9/L (Ref 4.0-11.0), platelets were normal at 402 × 10^9/L (Ref 150-450), sodium and potassium 143 mmol/L (Ref. 135-147) and 3.6 mmol/L (Ref 3.5-4.5) respectively. Estimated GFR > 90 mL/min/1.73 m^2 (Ref > 60), Cr 80 micromol/L (Ref 60-110), CRP 16 mg/L (Ref <1). His LFTs, lipase, serum glucose, and troponin levels were unremarkable.

A Renal CT organized in the emergency department for presumed renal colic (Figs. 4A and B) showed a 110 x 105 x 105 mm sized centrally hypodense and peripherally hyperenhancing ovoid mass (642 cc) in the right upper retroperitoneum suggestive of a stable right adrenal hematoma. This hematoma was indenting the subcapsular margin of his liver; segment VI, pushing on the upper pole of the right kidney. This pathology was unexpected and therefore required immediate re-evaluation with a multiphase CT of his abdomen. This confirmed a right adrenal mass without further evidence of active bleeding (Figs. 4C and D). He was admitted to the ward for conservative management.

Two days later he developed a new tachycardia with a HR of 120 beats/min with BP of 133/77 mmHg. Hb dropped to 90

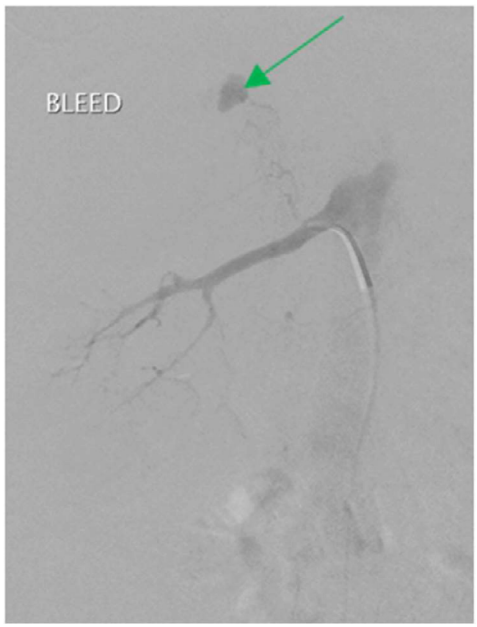

Fig. 3 – Interventional Angioembolization showing contrast extravasation from the right inferior adrenal artery following cannulation of the right renal artery.

g/L (Reg 130-180), and CRP increased to 218 mg/L (Ref <1), but the rest of his full blood examination, kidney and liver function and electrolytes were all normal. CT angiogram of the abdomen showed a stable size of the large 112 x 117 x 105 mm hemorrhagic right adrenal mass with no active contrast extravasation or soft tissue enhancement to suggest an active adrenal bleed. It showed a new small right-sided pleural effusion with associated basal atelectasis. He received 2 units of packed red blood cell transfusion and had continued conservative management with pain control. The patient did not experience any more episodes of tachycardia and his hemoglobin level remained stable at 106 g/L before discharge on day 5 of his admission.

Outpatient follow-up showed a normal urinary VMAs, metanephrines, normetanephrines, cortisol, and normal serum cortisol, catecholamines, metanephrines, normetanephrines; DHEA, Serum Renin: Urinary Aldosterone ratio, DMST, and ACTH. A repeat adrenal CT 6 months following discharge, showed a decreased size of the right adrenal hematoma, now measuring 91 × 83 × 84 mm (Fig. 5). Due to the large size of the hematoma the decision was made to surveil its resolution further.

A repeat CT scan after another 6 months has been organized to ensure further resolution of the hematoma. Following this scan, a multidisciplinary discussion will take place considering the patient's preference for an elective adrenal biopsy. We presume this will find a nonfunctioning right adrenal adenoma.

Discussion

Abdominal, loin, and flank pain, although vague, have been well-documented clinical symptoms of AH [3,7]. Whereas nausea, vomiting, dizziness, and neurological manifestations appear less often and when coupled with signs of hypotension (usually the result of exsanguination), tachycardia, and pyrexia; can be mistaken for sepsis. Hypertension may be due to a release of cortisol and catecholamines secondary to an expanding hematoma [2,3,8,9]. Both cases initially presented with an elevated blood pressure of 172/70 and 180/90 in cases 1 & 2 respectively.

Computed tomography (CT) has been shown to be the most effective way in the quick and early evaluation of AH [2,3].

In CT scans, an acute-subacute hemorrhage may show an ovoid mass with a radiological density of 50-90 Hounsfield units. Subsequent unenhanced CT scans may then show interval resolution (assuming no further bleeding) and a reduction in its attenuation [2,10,11]. Perirenal stranding, retroperitoneal hemorrhage, and crural thickening are important related findings. In the setting of AH; blood will appear to be both hyperdense and heterogenous in appearance [7].

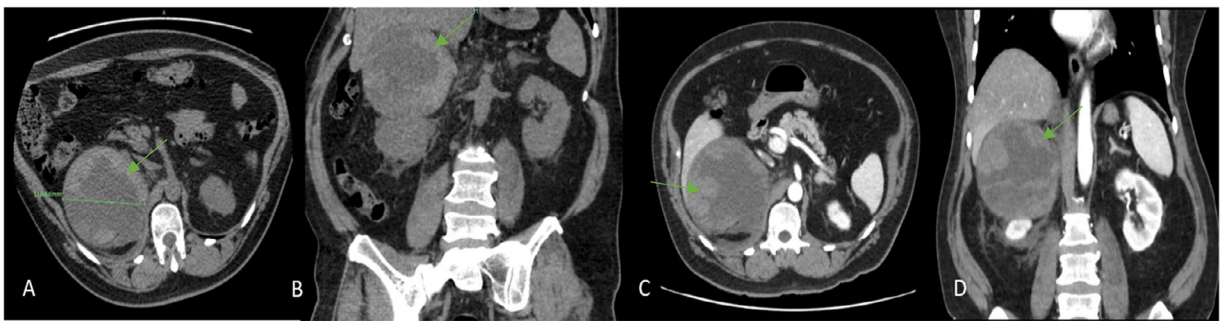

Fig. 4 – Abdominal & Pelvic Computed Tomography (CT) scan of a 63-year-old gentleman in Case 2. A green arrow pointing towards the adrenal hemorrhage. The maximal size of the abnormality was 110 x 105 x 105 transverse, craniocaudal, and anteroposterior measurements respectively. A) CT KUB axial organized in Emergency, identifying an ovoid lesion (green arrow) with a mean density of 27.2HU B) CT KUB sagittal organized in Emergency, density as in Fig. 1A C) CT AP contrast-enhanced; Multiphase Axial organized in Emergency with a mean density of 31 HU. D) CT AP contrast-enhanced, Multiphase Sagittal organized in Emergency. Density as in Fig. 1C.

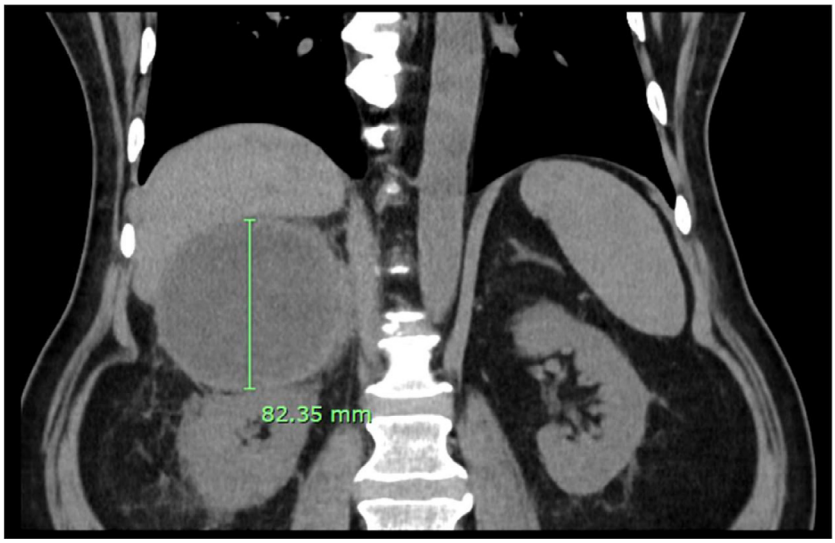

Fig. 5 – Repeat CT AP contrast-enhanced scan, organized as an outpatient, six months following discharge. Demonstrating a decrease in size of the hematoma; 91 x 82.35 x 84 mm transverse, craniocaudal, and anteroposterior measurements respectively with a mean density of 21HU.

Ultrasound with or without contrast enhancement may be useful in children, however, becomes technically more difficult as the patient ages [2,3,10]. Magnetic resonance imaging is the most sensitive and specific modality. It will highlight the presence of methemoglobin via the high intensity of a T1-weighted image [2,10] and can help identify solid components suggestive of an underlying tumor or subacute versus chronic hemorrhage [7]. The lack of ionizing radiation adds to the usefulness in young and pregnant populations however, the availability of MRI is dependent on department resources and may not be appropriate in an unstable patient due to the time taken to complete a scan.

Catecholamine release versus hemodynamic instability due to hemorrhagic shock may cause a significant challenge to the treating surgeon and anesthetist.

In an AH secondary to a hypersecretory adrenal tumor, biochemical imbalances may occur and pose a further challenge to management [12]. Hyperaldosteronism (Conn's) can result in renal sodium and water retention with concurrent potassium excretion causing; hypertension resistive to pharmacologic therapy and prolonged action of neuromuscular blocking agents due to hypokalemia and associated metabolic alkalosis [12,13]. Patients suffering from hypercortisolemia may need blood pressure, blood glucose, and hypokalemia optimized; adrenal enzyme inhibitors such as ketoconazole and mitotane have been suggested [14–16].

Adrenal insufficiency (AI) or Addison's disease can manifest with hyponatremia, hyperkalemia, and hypoglycemia when 90% of the adrenal gland is destroyed [3,5,6]. In the setting of unilateral AH this is thankfully a rare occurrence due to a normal functioning contralateral adrenal gland, even in a bilateral AH, AI affects 16.7% to 50% of patients [3,6]. An acute Addisonian Crisis (AC) may present similarly to hemorrhagic shock along with biochemical findings of AI [17]. In the acute setting in the emergency department initial serum hyponatremia, hyperkalemia, serum cortisol, and hypotension not responding to fluid resuscitation may point toward an AC. A short synachten (corticotropin) test is considered a "gold standard" diagnostic tool for AI [3,18,19].

However it must be said, that diagnostic testing in the acute bleed will yield unsatisfactory results and must not delay IV fluids/blood transfusion and 100 mg hydrocortisone or dexamethasone equivalent if AC is suspected [3,18].

Management of adrenal hemorrhages has changed over the years, with the availability of CT, exploratory laparotomy has fallen out of favor [3,6,20]. Treatment is based on the hemodynamic stability of the patient. Both traumatic and atraumatic causes of AH without evidence of active bleeding can be treated conservatively combined with serial blood work, testing for adrenal function, treatment of hypofunction with corticosteroids, blood transfusions, and nutritional support [3].

Angioembolization has been shown to effectively treat adrenal hemorrhage in the acute setting with successful cessation of bleeding when the location of the bleeding can be identified; with hemodynamic stability in 94.1% of patients, with subsequent clinical improvement in 82.3% of patients during their admission [21].

A treatment algorithm has been suggested by Elhassan *et al*. 2023 whereby an assessment of the acute abdomen with a timely CT is the first port of call. A unilateral AH that is clinically stable and shows no signs of adrenal insufficiency, can be monitored without glucocorticoid replacement. Whereas a unilateral AH who is clinically unstable and has ongoing hemorrhage should be considered for emergency laparotomy or angioembolization. In Case 1 we opted for the latter [22].

If the decision is for emergency laparotomy, preparation should be made for fluctuations in hemodynamic stability. During the removal of hyperfunctioning adrenal tumors, glucocorticoid replacement in the form of 100-200 mg hydrocortisone should be administered intravenously at the time of adrenal vein ligation [23].

Emergency laparotomy carries in-hospital mortality of 7.1% (2.3%-13.3%) at 30 days [24] and should be avoided if possible. Interval adrenalectomy has been suggested once the patient is adequately optimized; when inflammation and hematoma have resolved to allow for easier dissection [7]. A study by A Ali, G Singh, SP Balasubramanian 2018, had a median range of 7-11 weeks for interval adrenalectomy [25]. In case 2, due to the size of the hematoma, a longer period of monitoring was opted for.

Conclusion

Adrenal hemorrhage is an uncommon condition and can be misdiagnosed. Clinical manifestations may be vague and laboratory values may remain within normal limits. Both of our cases were presumed renal colic in the first instance.

Case 1 was initially managed conservatively however represented in severe hemorrhagic shock due to continued anticoagulant use, his re-bleed required prothrombinex to reverse anticoagulation, vasopressor support, and angioembolization of the bleeding vessel. Outpatient adrenal function testing was incomplete as he unfortunately passed away due to thromboembolic disease likely secondary to anticoagulant cessation.

Case 2 was managed conservatively on the ward with analgesia, blood transfusion, and close monitoring. He demonstrated some resolution on a 6-month follow-up CT and unremarkable adrenal function evaluation as an outpatient.

In the setting of the acute abdomen, stabilization with intravenous access and fluid therapy, blood products, and vasopressors in the hemodynamically unstable patient is paramount and should be the first step in treatment. Anticoagulants should be ceased immediately and reversal agents such as prothrombinex should be considered after consultation with Hematological services.

As CT scans have become more widespread and readily available, rapid evaluation in the emergent setting should be prioritized. Contrast-enhanced CT scans, particularly in the arterial phase may help the clinician identify an active bleed. MRI scans, whilst sensitive and specific, are time-consuming and availability depends on department resources.

If contrast extravasation is visualized, CT-Angiography with a view to angio-embolize the offending vessel should be prioritized, and if unsuccessful; emergency laparotomy can be considered. We have demonstrated that a hemodynamically stable patient can be monitored with serial imaging as an outpatient.

Serial biochemical testing for adrenal failure is suggested as good practice, however, there is no accepted practice as to how frequent or length of time this should be done. We hope adrenal hemorrhage literature will continue to expand and provide robust treatment algorithms for clinicians.

Patient consent

We at Western Health, Melbourne, Australia, confirm that written informed consent has been obtained for publication from the patient(s) and/or their legal representative(s)/guardian(s).

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