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Spontaneous renal subcapsular hematoma; a case report and reviewing management options

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Abstract: Spontaneous subcapsular renal hematoma is a rare complication with potentially fatal effects in clinical practice. We discuss a case of a patient who arrived at the emergency room with abrupt onset flank discomfort and hematuria. The damaged kidney was effectively embolized by interventional radiology, and the patient quickly recovered. Follow-up tests revealed that the hematoma had shrunk in size. We believe that early arterial embolization should be explored in the care of patients with renal bleeding because it may enhance outcomes.

Keywords: Flank pain; Hematoma; Hematuria; Kidney Diseases

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1. Introduction

The kidney's subcapsular region is a possible location where fluid might collect, compressing the renal parenchyma. Kidney trauma is the most common cause of subcapsular renal hematomas. Renal hematoma is an uncommon emergency presentation. An unidentified intraparenchymal renal hemorrhage in a patient on anticoagulation or without a history of trauma is termed a spontaneous renal hemorrhage (RH) or hematoma (1). The most prevalent causes of spontaneous RH include vasculitides (polyarteritis nodosa), concealed vascular renal tumors (renal cell carcinoma or angiomyolipoma) and vascular abnormalities.

Uncontrolled hypertension, infection, ruptured hemorrhagic cysts, and big renal stones causing erosion have all been linked to a few occurrences (2). The literature describes a number of different treatment perspectives for RH. Therapeutic suggestions have developed throughout time and frequently reflect the writer's or clinician's expertise and area of practice. In all instances, initial reports from surgical subspecialties advised radical or modified nephrectomy. Multiple case reports, however, show that a careful hospitalization or outpatient therapy trial may be beneficial (3). Pain relief and hydration are among the nonsurgical treatments, as are antibiotics if an infectious etiology is suspected and blood product replacement in individuals with severe anemia (4).

Conservative therapy, on the other hand, comes with the consequences of reduced renal function as compared to other types of therapy. Another approach for controlling active bleeding, particularly in hemodynamically unstable patients, is selective arterial embolization (5). Given the solid data backing its benefits in situations of renal bleeding, whether related to trauma or not, as well as its low risk profile, several writers propose embolization as the ultimate treatment (6). Some writers, however, still see embolization as a temporary therapy and a backup to nephrectomy (4). Another point of dispute is clinical and imaging follow-up in the case of RH. In general, outpatient follow-up and imaging are required till the hemorrhage has dissipated, a cause has been determined, or a final operation has been done (6).

2. Case presentation

A 65-year-old male patient presented to the emergency department (ED) with severe left flank pain for seven hours. The pain is graded at 7 out of 10. The pain radiated to the left lower quadrant and lower back. The patient also complained of malaise, fatigue, and constipation. He denied nausea, vomiting, or fever. The patient has no urine output as he is a known case of end-stage renal disease on dialysis four times per week. Additionally, he has diabetes mellitus, hypertension, chronic anaemia, peripheral vascular disease, secondary hyperparathyroidism of renal origin, hypothyroidism, and obstructive sleep apnea. Medications used include aspirin, bisoprolol, atorvastatin, insulin, and cinacalcet.

The patient's vital signs showed a blood pressure of 101/60 mmHg, a pulse rate of 80 /minute, a respiratory rate of 18 /minute, a temperature of 36.7 °C, and an oxygen saturation of 98% on room air. On physical examination, an obese patient with a pedunculated soft abdomen, multiple bruises, and no tenderness in the left flank on deep palpation. Chest and cardiac examinations are unremarkable. Thus, analgesia was commenced, and a set of blood tests were arranged. Additionally, a non-enhanced abdominopelvic computed tomography (CT) scan was done. The blood tests revealed that the patient had elevated renal function (creatinine level of 665 micromol/l and urea level of 18.80 mmol/l), a white

Figure 1 There is a large left renal subcapsular hyperdensity compressing the renal cortex anteriorly in keeping with subcapsular haematoma. This haematoma has posterior extension to involve the perinephric fat and extends to the retroperitoneal space. Associated fat stranding and thickening of the left Gerota facia. This haematoma measures about 13×10×7 cm.

Figure 2 Large hematoma extending from the left kidney into perirenal, pararenal and retroperitoneal spaces measuring 76×87×117 mm (T×AP×H) associated with hypoattenuation foci within the haematoma mainly arising from the most periphery of the renal parenchyma, thickened left Gerota fascia, and perirenal fat mesentery which is stranded and displacing the left adrenal gland superiorly, minimal free fluid in the left side of the abdomen and left paracolic gutter.

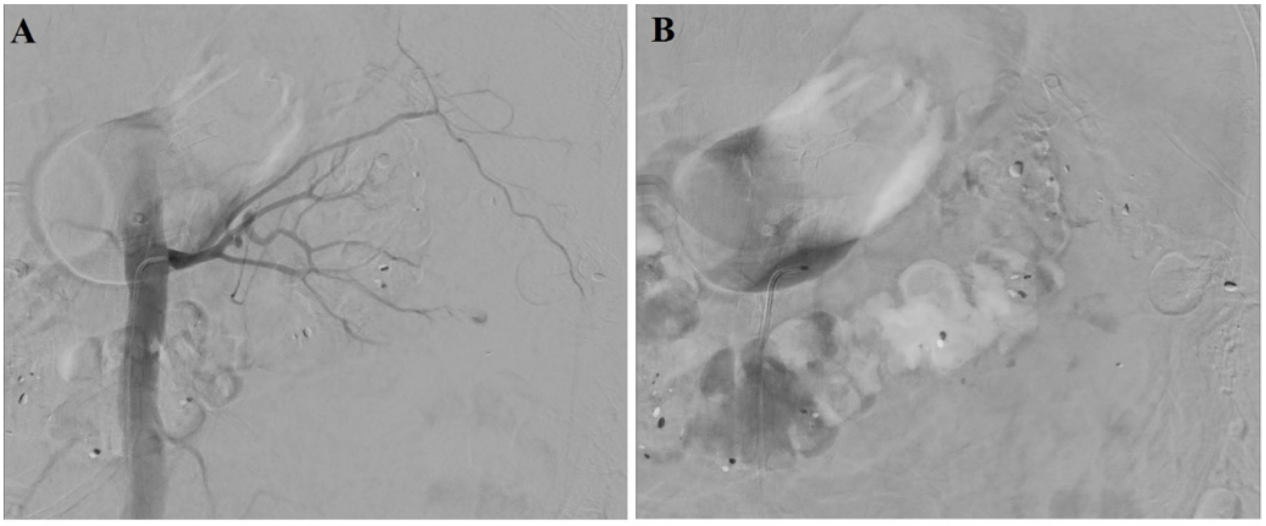

Figure 3 Angiography demonstrated active extravasation from the capsule of the left kidney (from the most all the branches).

blood cell count of 13.4×10^9/L, Hgb level of 82 g/L, a platelet count of 315×10^9/L, and a C-reactive protein level of 43.3 mg/L. The CT showed a large left renal subcapsular hyperdensity compressing the renal cortex anteriorly, in keeping with subcapsular hematoma (Figure 1).

Thus, the urologist was consulted, and an abdominopelvic CT with intravenous (IV) contrast was arranged to exclude active bleeding. The patient was admitted to the hospital for continuous observation, monitoring of Hgb, and further management. The abdominopelvic CT with IV contrast showed large left renal and perirenal hematoma with evidence of extravasation from the interlobar renal arteries of

the lower pole mainly (Figure 2).

The patient's Hgb dropped to 69 g/L at night. Consequently, angiography was performed, which demonstrated active extravasation from the capsule of the left kidney from most of all the branches (Figure 3). Therefore, the decision was made to embolise the entire left kidney using a combination of coils and PVA particles (Figure 4). Additionally, the patient received two packed RBCs. The next day, the patient underwent her regular hemodialysis session and her repeated Hgb came to 82 g/L. On the 3rd day, the patient was reported to be pain free, with no nausea or vomiting, no fever, no hematuria, and tolerated orally. He had his Hgb level at 83 g/L and was thus discharged to follow up with the clinic.

The patient missed his appointment with the clinic. Two weeks later, the patient came to the ED complaining of progressively increasing left flank and back pain for almost one week. He denied fever or hematuria. His vital signs were within the normal limit. His blood tests revealed white blood cells of 16.1×10^9/L, Hgb of 91 g/L, platelets of 505×10^9/L, and C-reactive protein of 223.3 mg/L. The patient's CT scan revealed no active bleeding with regression of sup capsular hematoma size in addition to edema and the fluid surrounding the left kidney (Figure 5). Therefore, the patient was offered admission to rule out infected hematoma, with better IV antibiotic coverage and monitoring of hematoma regression. However, the patient refused and left, against medical advice on oral antibiotics.

Three days later, He came again with similar pain with no change in pain character. His blood tests showed mild improvement of his inflammatory markers (white blood cell of 12.6×10^9/L, Hgb 93 g/L, platelet 464×10^9/L, and C reactive protein of 218.8 mg/L). The patient was discharged to follow with clinic on his prescribed medications. Two weeks later, He came back to ED with same complain of left flank pain and hematuria. His vital signs were within normal limit and his blood tests showed a white blood cell of 6.6 $x10^9$/L, Hgb 94 g/L, platelet 322 $x10^9$/L, and C reactive protein of 104.4 mg/L). The patient's CT scan revealed regression of sup capsular hematoma size. His urine tests revealed features of urinary tract infection. The patient was discharged to follow with urology clinic. In the urology clinic, the patient complained of chronic mild to moderate bilateral lower abdominal pain and flank pain associated with dysuria and hematuria. An US was arranged by the urologist to follow the size of hematoma and to exclude any other pathology or free fluid (Figure 6).

The patient was reassured that the disease progress as expected with no active bleeding and he should comply with his medications with regular clinic follow up.

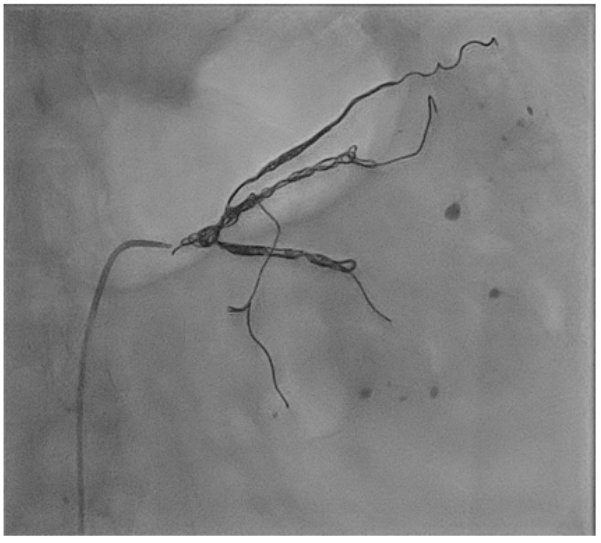

Figure 4 Embolisation the entire left kidney using combination of coils and PVA particles (500-700 um).

3. Discussion

RH is an uncommon disease with potentially fatal implications and widely differing treatment options. RH patients typically report having Lenk's triad of flank discomfort, soreness, and "symptoms of blood loss," such as nonspecific exhaustion, dizziness, tachycardia, and hypotension, relying on how much blood has been lost. RH can, however, be mistaken for a number of acute abdominal diseases. As a result, RH is frequently identified by chance during a contrast-enhanced abdominal CT scan or an ultrasound. However, for conclusive diagnosis and preprocedural planning, abdominal CT angiography is the preferable imaging modality. Hematuria and anemia are both common laboratory results (7). According to the little research on the subject, many patients can be managed conservatively with pain treatment and regular clinical monitoring. Given the potentially fulminant nature of untreated internal bleeding, harsher interventions are frequently required. The patient, in our instance, had left flank discomfort, slight anemia from his baseline, and stable vital signs. Imaging, on the other hand, revealed vigorous extravasation. This might be used to justify more aggressive intervention in the early stages. This contradictory thinking is replicated in the literature, with several writers proposing pain management and antibiotics as early therapeutic choices, as well as immediate embolization or partial nephrectomy (7).

When there is clinical or imaging evidence of current bleeding, we advocate a lower threshold for immediate embolization. Patients who have an active hemorrhage at the time of presentation are more likely to have ongoing or repeated bleeding, resulting in increased pain from dilation of the renal capsule or surrounding structures, hemorrhagic shock complications, or hypertension associated with Page phenomena. When compared to nephrectomy, selective renal artery embolization is a far less intrusive surgery that has a minimal risk on the patient and this leads in dramatically better kidney function than conservative treatment (5). As a result, it appears to be the most sensible option for treatment in this situation. Treatment delays can lengthen the patient's hospitalization, increasing the risk of hospital-acquired ill-

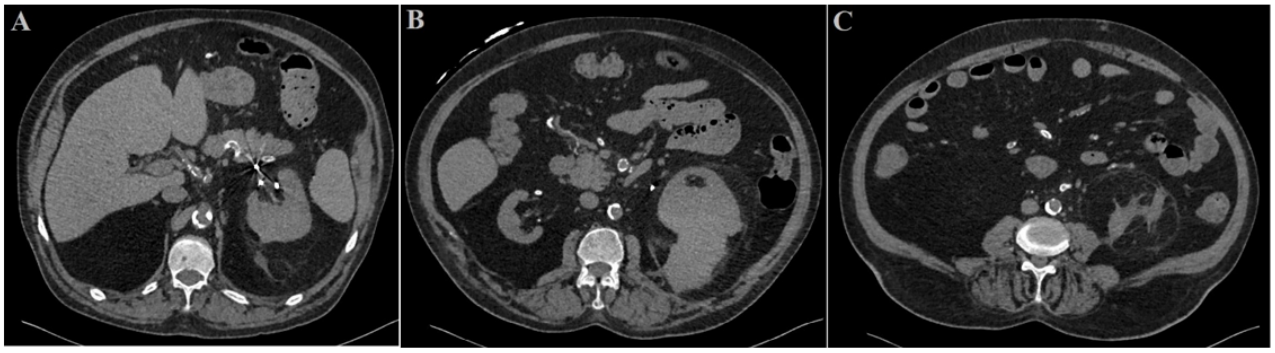

Figure 5 Large left kidney subcapsular haematoma which appear mildly less than before measuring 9×6 cm versus 11×7 cm. No obvious new bleeding. The degree of the oedema , stranding and fluid surrounding the left kidney is also appear less than before.

Figure 6 Transverse and sagittal view of the left kidney showed the subcapsular hematoma with its dimension and volume.

nesses while also adding to the financial load affecting the patient and the whole healthcare system. This is particularly true if the patient requires another admission. As opposed to readmission to the hospital or analogous laparoscopic or open procedures, arterial embolization techniques are frequently less expensive.

According to some writers, the first therapy of RH must be determined exclusively on the basis of clinical manifestations. Although physiological parameters are important, imaging is required to diagnose RH, and active extravasation proof on CT scan should also drive first therapy. Angiogram with embolization is indicated after proper stabilization in case the patient's vital signs are unstable or there is ongoing extravasation on radiography (8). If vital signs remain stable and imaging shows no substantial extravasation, conservative care with short-term hospitalization followed by continuous clinical monitoring as an outpatient may be acceptable. Selective artery embolization should be done if continuing bleeding is visible clinically (deteriorating vital signs or progressive pain), by radiology follow-up (ongoing extravasation or hematoma enlargement) or by serial hemoglobin/hematocrit labs.

If the underlying reason cannot be determined during the first workup, outpatient follow-up is also necessary. If no cause is discovered during early monitoring, the physician may decide that intermittent imaging of the patient is necessary. When cases of vasculitis like polyarteritis nodosa or others are discovered, more therapy can be required depending on the severity of the problem. Embolization can be used to treat high-flow vascular abnormalities. If a tumor is discovered, the following stages of treatment are determined by the size of the tumor. Biopsy and tumor ablation may be used to treat every renal lesion smaller than 4 cm in size and affecting only the kidney. Larger tumors, as well as those that have spread metastatically or lymphatically, may necessitate a partial or total nephrectomy (9, 10). In our patient, the patient had multiple risk factors like hypertension, peripheral vascular disease and he is on antiplatelet therapy.

4. Conclusion

Spontaneous renal hemorrhage is a rare but potentially fatal condition described as ongoing or past kidney bleeding without anticoagulation or triggering trauma. On acceptable baseline therapy and follow-up guidelines, the literature is divided, with suggestions ranging from simple therapeutic care to complete nephrectomy. While conservative care may be the best option for clinically stable patients, lowering the embolization threshold may have improved our patient's results and must be addressed in any RH case involving continuous extravasation.

5. Declarations

5.1. Acknowledgment

None.

5.2. Authors' contribution

All the authors met the standards of authorship based on the recommendations of the International Committee of Medical Journal Editors.

5.3. Conflict of Interest

There is no conflict of interest to disclose.

5.4. Funding

There is no funding or support to declare.

5.5. Consent for publication

Patient's consent for publishing the case with no identifiable personal information was obtained.

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