Signature: © Pol J Radiol, 2016; 81: 572-577
DOI: 10.12659/PJR.899710

Polish Journal of Radiology
www.PolRadiol.com
CASE REPORT

Received: 2016.05.24
Accepted: 2016.06.03
Published: 2016.11.28

Authors' Contribution:
A Study Design
B Data Collection
C Statistical Analysis
D Data Interpretation
E Manuscript Preparation
F Literature Search
G Funds Collection

Renal Artery Injury Secondary to Blunt Abdominal Trauma – Two Case Reports

Zahoor Ahmed[ADEF], Syed Nabir[BCEF], Mohamed Nadeem Ahmed[BCDF], Shatha Al Hilli[BCDF], Vajjala Ravikumar[BCDF], Umais Zaid Momin[CDEF]

Department of Radiology, Hamad General Hospital, Doha, Qatar

Author's address: Zahoor Ahmed, Department of Radiology, Hamad General Hospital, Doha, Qatar, e-mail: zahoory3k@gmail.com

Summary

Background: Blunt abdominal trauma is routinely encountered in the Emergency Department. It is one of the main causes of morbidity and mortality amongst the population below the age of 35 years worldwide. Renal artery injury secondary to blunt abdominal trauma however, is a rare occurrence. Here, we present two such cases, encountered in the emergency department sustaining polytrauma following motor vehicle accidents.

Case Report: We hereby report two interesting cases of renal artery injury sustained in polytrauma patients. In these two cases we revealed almost the entire spectrum of findings that one would expect in renal arterial injuries.

Conclusions: Traumatic renal artery occlusion is a rare occurrence with devastating consequences if missed on imaging. Emergency radiologists need to be aware of the CT findings so as to accurately identify renal artery injury. This case report stresses the need for immediate CT assessment of polytrauma patients with suspected renal injury, leading to timely diagnosis and urgent surgical or endovascular intervention.

MeSH Keywords: **Abdominal Injuries • Multiple Trauma • Renal Artery**

PDF file: http://www.polradiol.com/abstract/index/idArt/899710

Background

Injury is the leading cause of death and disability worldwide and Qatar is no different. The most common injury-related causes of death and disability, regardless of age or gender, in decreasing order are: motor vehicle accidents (MVA), self-inflicted injuries, and inter-personal violence [1]. As per the International Road Federation, Qatar has one of the highest road traffic death rates in the region, with 19 deaths per 100 000 population [2].

Patients involved in road traffic accidents frequently sustain polytrauma. Polytrauma is defined as two or more injuries to physical regions or organ systems, one of which may be life-threatening, resulting in physical, cognitive, psychological, or psychosocial impairments and functional disability [3]. Renal artery injury is a rare and serious complication of polytrauma. Fewer than a thousand cases have been reported in the literature since first described by Von Recklinghausen in 1861 [4]. It is often encountered alongside with other intra-abdominal visceral injuries. We hereby report two interesting cases of renal artery injury sustained in polytrauma patients. In these two cases we encountered almost the entire spectrum of findings that one would expect in renal arterial injuries.

Management depends upon the grade of injury, hemodynamic status, renal function, and, importantly, the availability of treatment options. Surgical revascularization or stent placement is preferred in patients with a bilateral injury or a solitary kidney [5]. Non-operative management is currently the accepted therapeutic option in most patients with traumatic collusion of the main renal artery. The results of surgical revascularization have been poor thus far, with long-term preservation of kidney function in fewer than 25% of patients [6].

Case Report

Case 1

Our first case is of an 18-year-old male brought to the emergency department, after a motor vehicle collision – unrestrained car driver.

On arrival, the patient was desaturated and hypotensive. His blood pressure at that time was recorded as 70/40 mmHg. Initial evaluation revealed a GCS of E1M4V1 and low air entry to his left hemithorax, apart from bruises on the scalp, chest and abdomen. Laboratory investigations found a hemoglobin level of 13.1 g/dL, WBC 1.9×10^3 μL and platelets 40×10^3 μL. His blood urea nitrogen was 5.2 mmol/L, serum creatinine 95 μmol/L. He was immediately intubated and aggressively resuscitated with fluids and inotropes. As the patient's hemodynamics started to improve, he was referred to the clinical imaging department for an emergency whole-body CT scan.

Contrast-enhanced CT scan of the abdomen demonstrated abrupt termination of the proximal segment of the left renal artery (Figure 1) with adjacent active contrast extravasation/pooling, suggesting renal arterial laceration. There was non-enhancement of the left renal artery – devascularized left kidney (Figures 1–3). Moderate amount of left perinephric hematoma was also noted. The right renal artery and renal parenchyma demonstrated essentially normal enhancement.

In addition, the patient also had multiple splenic lacerations (Figure 4) and a large perisplenic hematoma. The patient had also sustained pancreatic lacerations (Figure 5) and left diaphragmatic rupture (Figure 6).

CT scan of the brain demonstrated haemorrhagic contusions in the right frontal and temporal lobes. Chest CT demonstrated bilateral lung contusions, multiple rib fractures and bilateral hemopneumothorax.

Following the CT scan, the patient underwent emergency exploratory laparotomy with left diaphragmatic repair, stomach repair, splenectomy, left nephrectomy, and small and large bowel serosal repair. He was then shifted to the trauma ICU. A second-look laparotomy with partial abdominal closure was performed two days later. The final abdominal closure was done after further 7 days. Serial hemoglobin levels revealed him to be hemodynamically stable throughout his hospitalization. Serum creatinine was within normal limits. He was finally discharged from hospital in stable condition.

Case 2

Our second case is of a 21-year-old male patient brought to the emergency department following a motor vehicle accident – pedestrian struck by a truck.

The patient was in a critical condition, with a blood pressure of 90/60 mmHg, GCS of E1M1V1 and low oxygen saturation of 71%. Physical examination revealed injuries to the scalp, face, chest and abdomen. There was bilateral

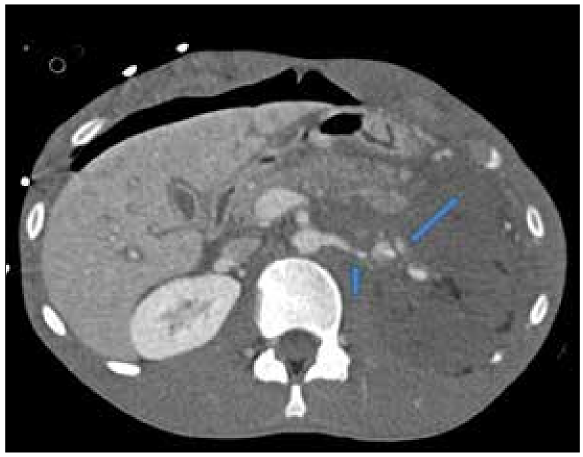

Figure 1. Contrast-enhanced CT scan of the abdomen demonstrating abrupt termination of the left renal artery (short arrow), with adjacent contrast blush/extravasation (long arrow), suggesting renal arterial laceration. Devascularized left kidney is also noted. The right parenchyma is normally enhanced.

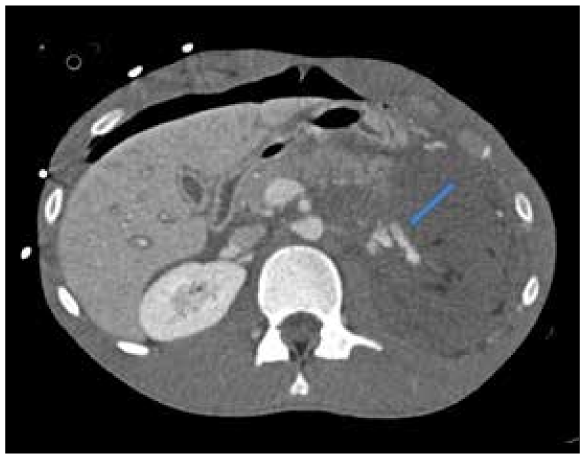

Figure 2. Pooling of extravasated contrast (arrow) at the left renal hilum with devascularized left kidney.

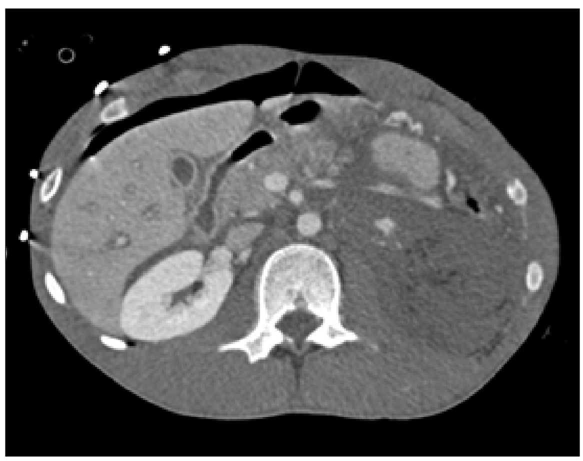

Figure 3. Devascularized left kidney with pooling of extravasated contrast at the left renal hilum.

Figure 4. Multiple splenic lacerations (arrows) with perisplenic hematoma.

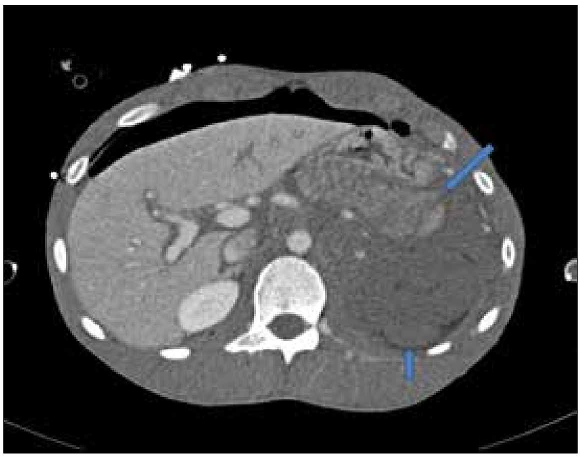

Figure 5. Pancreatic lacerations (long arrow) with peripancreatic hematoma. Non-enhancing left kidney (short arrow) is also noted at this level.

decreased air entry in the chest. Pupils were constricted and non-reactive.

Initial laboratory investigations revealed hemoglobin of 4.9 g/dL, hematocrit of 16.5%, platelet count of 104×10^3 μL, blood urea nitrogen of 3.6 mmol/L and serum creatinine of 91 μmol/L.

After aggressive resuscitation and stabilization, he was referred to the Clinical imaging department for CT scan.

CT scan of the abdomen revealed abrupt truncation of the proximal segment of the left renal artery (Figure 7), with a surrounding hematoma. No active extravasation of contrast was appreciated at that time. There was reconstitution of flow in the distal segment of the left renal artery (Figure 8), thereby suggesting partial thrombosis/occlusion of the artery. This was further corroborated by relative hypoperfusion of the left kidney, with patchy enhancing areas. In addition, there was a right renal laceration (Figure 9) with an associated right perinephric hematoma. However, the right renal artery was intact. Small lacerations in segment 7 of the liver were also identified.

Figure 6. Coronal reformatted contrast-enhanced CT image. Abnormal high location of the spleen in the left hemithorax, with left-sided hemothorax, indicating left diaphragmatic rupture. Devascularized left kidney and normally-enhancing right kidney are also visualized.

Figure 7. Contrast-enhanced CT of the abdomen demonstrating abrupt termination of the left renal artery (long arrow) and decreased perfusion of the left kidney (short arrow).

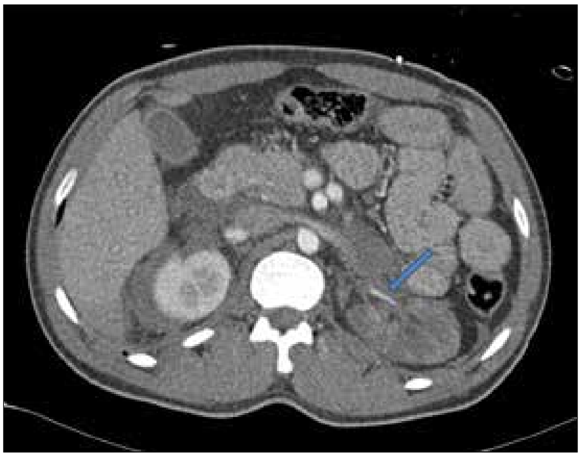

Figure 8. Relative hypoperfusion of the left kidney. There is reconstitution of flow in the distal segment of the left renal artery (arrow), suggesting partial thrombosis/obstruction. Note the right perinephric and left perihilar hematoma, without apparent active contrast extravasation.

Figure 9. Right renal parenchymal laceration (arrow) with perinephric hematoma.

CT scan of the chest demonstrated bilateral lung contusions, hemopneumothorax, multiple rib fractures, fractures of the right clavicle and left scapula. CT brain revealed multiple facial fractures, without apparent intra cranial hemorrhage.

Immediately following CT the patient was shifted to the intervention suite to assess the extent of renal artery injury and for possible endovascular revascularization. Digital subtraction angiogram revealed total occlusion of the left renal artery (Figure 10) with extravasation of contrast (Figure 11). The guide wire could not be advanced beyond the point of occlusion, at the mid segment of the renal artery. Revascularization was not achieved due to prolonged ischemia.

The patient was shifted to the trauma ICU and then to the recovery room, where the patient was recuperating at the time of writing this report.

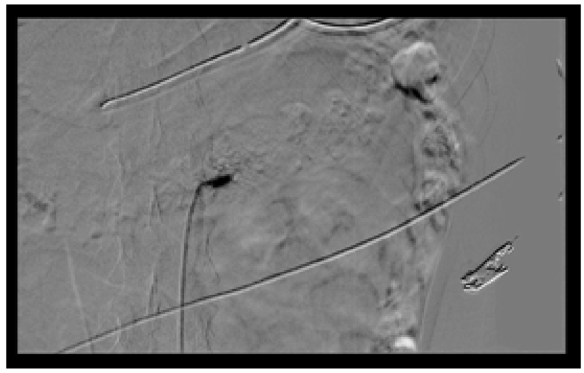

Figure 10. Selective left renal digital subtraction angiogram demonstrating abrupt cut-off at the junction of the proximal/mid segment of the left renal artery.

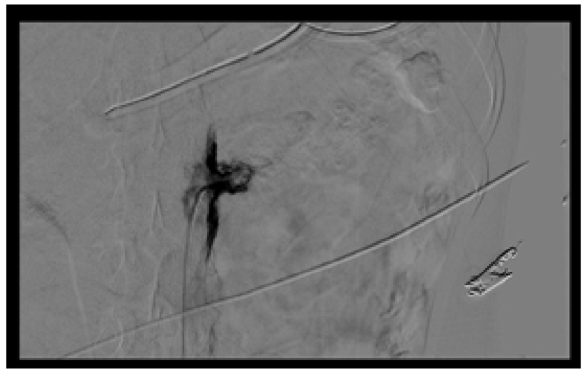

Figure 11. Selective left renal digital subtraction angiogram showing extravasation of contrast from the left renal artery. There is non-opacification of the distal renal arterial segment.

Discussion

Depending on the severity of trauma, renal injuries are traditionally classified into five grades [7,8]. This classification system recognizes the progressive nature of the parenchymal and vascular damage according to the increasing level of trauma. The incidence of renal artery injury from blunt trauma ranges between 0.05%, as reported by Demetriades' group in the National Trauma Data Bank [9], and 0.08% as reported by Bruce et al. [10] The left kidney is reported to be slightly more prone to injury.

Children are more vulnerable to renal artery injury in blunt traumas. This is attributed to the relatively larger kidneys in the pediatric population, their higher mobility, and a relative lack of surrounding protective tissues [15].

According to literature, the relative rate of injury is 1.4:1 to 2:1 when comparing left to right sides. Possible explanations for this include the following: firstly, the right renal artery is shielded by the inferior vena cava and duodenum and stabilized in place by the liver and duodenum. In contrast, the left renal artery is shorter and more acutely angled than the right one and is hence more prone to stretch injuries [11–13]. This finding was also observed in both our cases, where although the patients sustained multi-organ injuries, the right renal artery remained intact. The first case had left renal laceration with contrast extravasation, while the second case had partial left renal thrombosis/occlusion.

Renovascular injuries are associated with multiple non-renal organ injuries and with a high mortality rate of 19% to 44% [14]. In both the cases described above, non-renal injuries were observed. Both the cases had a significant chest injury. In the first case, splenic, pancreatic, and diaphragmatic injuries were appreciated, while in the second case there were liver and contralateral renal lacerations.

Renal vascular injury secondary to trauma can be of two types – arterial spasm and arterial occlusion. Arterial spasm is considered to be secondary to contusion [16]. This condition should not be confused with traumatic occlusion of the renal artery due to thrombosis or intimal flap formation, which can cause devascularization of the kidney. Additional attention should be paid to distinguish between these two conditions because of the different therapeutic options available [17,18]. Devascularization of the entire kidney due to vascular laceration or renal artery thrombosis is the most severe form of renal injury (grade 5). If the kidney is devascularized due to an isolated intimal injury to the renal artery that results in thrombosis, extensive retroperitoneal hemorrhage and hematuria may be absent [19].

Renovascular injuries usually occur about 2 cm from the origin of the main renal artery but may involve branch renal vessels, as was seen in one patient in this series [20]. In both our patients, the main renal artery was involved at the junction of the proximal and mid segments.

The typical CT findings of traumatic renal infarction include abnormal enhancement of the affected kidney, retrograde opacification of the renal vein from the inferior vena cava, and abrupt truncation/irregularity of the renal arterial lumen at the point of occlusion with contrast extravasation. Abnormal renal enhancement may range from irregular linear hypodensities to delayed or diminished enhancement of the affected kidney. This "asymmetric nephrogram" is more prominent as the degree of injury increases, reflecting the severity of renal parenchymal ischemia [21]. There is non-opacification of the renal pelvicalyceal system, secondary to ischemic nonfunctioning renal parenchyma. The 'cortical rim nephrogram sign' represents enhancement of the peripheral outer cortex due to perfusion from capsular collateral blood flow [11]. The cortical rim nephrogram sign of a devascularized kidney may be absent in the acute setting [19].

Renal artery spasm secondary to contusion can cause non-enhancement of one or both kidneys. Hence, it is imperative to distinguish between these two conditions based on clinical, laboratory and imaging findings, as the management differs. In our case 2, an immediate renal angiogram was performed that confirmed occlusion of the left renal artery with some contrast puddling at the site of injury. Sometimes, severe renal artery spasm and traumatic occlusion of the renal artery may occur simultaneously, making the correct interpretation dicey at times.

The most critical factor in preserving renal function is the reestablishment of blood flow at the earliest. Spirnak and Resnick [22] defined the optimal time frame for revascularization as 6–12 hours from injury. However, ischemic changes start to appear around 60–120 minutes after injury, thus it is believed that the optimal time frame is likely to be much shorter, between 3 and 4 hours, as stated by Cass [11] and Flye et al. [23].

As in our case, renal vascular injuries rarely occur in isolation. Cass et al. reported an average of 3.7 associated injuries per patient, with 85% of patients requiring laparotomy for their intraabdominal injuries [11].

Kidney salvage rates have been low after surgical revascularization of blunt injuries, with high rates of recurrent thrombosis [6,9,24]. These discouraging results have persuaded most surgeons to avoid operative renal artery revascularization when there is a functioning contralateral kidney [14,26]. Furthermore, since the majority of these patients have other life-threatening injuries, their management takes a higher priority. These situations have resulted in a shift towards a more conservative approach to this kind of injury. Recently, there have been encouraging reports about percutaneous revascularization by endovascular stenting in stable patients with unilateral renal artery occlusion [25–28].

Conclusions

Traumatic renal artery occlusion is a rare occurrence with devastating consequences if missed on imaging. Emergency radiologists need to be aware of the CT findings so as to accurately identify renal artery injury. This case report stresses the need for immediate CT assessment of polytrauma patients with suspected renal injury, leading to timely diagnosis and urgent surgical or endovascular intervention.

References:

1. Al Sheikhly AS: Pattern of trauma in the districts of Doha/Qatar: Causes and suggestions. E3 Journal of Medical Research, 2012; 1(2). 025–028
2. Mock C et al: Strengthening care for the injured: Success stories and lessons learned from around the world. World Health Organization. *http://whqlibdoc.who.int/publications/2010/9789241563963_eng.pdf*
3. Polytrauma/TBI system of care. US Department of veteran affairs. *http://www.polytrauma.va.gov/news-and-resources/terminology-and-definitions.asp*
4. Von Recklinghausen F: Haemorrhagische Niereninfarkte. Arch Path Anat Physiol, 1861; 20: 205–7 [in German]
5. Fraser JD, Aguayo P, Ostlie DJ, St Peter SD: Review of the evidence on the management of blunt renal trauma in pediatric patients. Pediatr Surg Int, 2009; 25: 125–32
6. Haas CA, Dinchman KH, Nasrallah PF, Spirnak JP: Traumatic renal artery occlusion: A 15-year review. J Trauma, 1998; 45: 557–61
7. Moore EE, Cogbill TH, Malangoni MA et al: Organ injury scaling. Surg Clin North Am, 1995; 75(2): 293–303
8. Tinkoff G, Esposito TJ, Reed J et al: American Association for the Surgery of Trauma Organ Injury Scale I: spleen, liver, and kidney, validation based on the National Trauma Data Bank. J Am Coll Surg, 2008; 207: 646–55

9. Sangthong B, Demetriades D, Martin M et al: Management and hospital outcomes of blunt renal artery injuries: Analysis of 517 patients from the.National Trauma Data Bank. J Am Coll Surg, 2006; 203: 612–17
10. Bruce LM, Croce MA, Santaniello JM et al: Blunt renal artery injury incidence, diagnosis, and management. Am Surg, 2001; 67: 550–56
11. Cass AS: Renovascular injury from external trauma. Urol Clin North Am, 1989; 16: 213–20
12. Lee JT, White RA: Endovascular management of blunt traumatic renal artery dissection. J Endovasc Ther, 2002; 9: 354–58
13. Carroll PR, McAninch JW, Klosterman P, Greenblatt M: Renovascular trauma: Risk assessment, surgical management, and outcome. J Trauma, 1990; 30: 547–52, discussion 553–54
14. Santucci RA, Wessells H, Bartsch G et al: Evaluation and management of renal injuries: Consensus statement of the renal trauma subcommittee. BJU Int, 2004; 93: 937
15. Brown SL, Elder JS, Spirnak JP: Are pediatric patients more susceptible to major renal artery injury from blunt trauma? A comparative study. J Urol, 1998; 160: 138–40
16. Dinchman KH, Spirnak JP: Traumatic renal artery thrombosis: evaluation and treatment. Semin Urol, 1995; 13(1): 90–93
17. Rogers CG, Knight V, MacUra KJ et al: High-grade renal injuries in children – is conservative management possible? Urology, 2004; 64(3): 574–79
18. Henderson CG, Sedberry-Ross S, Pickard R et al: Management of high grade renal trauma: 20-year experience at a pediatric level I trauma center. J Urol, 2007; 178(1): 246–50
19. Harris C, Zwirewich CV, Lyburn ID et al: CT findings in blunt renal trauma. Radiographics, 2001; 21: S201–14
20. Barlow B, Gandi R: Renal artery thrombosis following blunt trauma. J Trauma, 1980; 20: 614–17
21. Dobrilovic N, Bennett S, Smith C et al: Traumatic renal artery dissection identified with dynamic helical computed tomography. J Vasc Surg, 2001; 34: 562–64
22. Spirnak JP, Resnick MI: Revascularization of traumatic thrombosis of the renal artery. Surg Gynecol Obstet, 1987; 164: 22–26
23. Flye MW, Anderson RW, Fish JC, Silver D: Successful surgical treatment of anuria caused by renal artery occlusion. Ann Surg, 1982; 195: 346–53
24. Elliott SP, Olweny EO, McAninch JW. Renal arterial injuries: A single center analysis of management strategies and outcomes. J Urol, 2007; 178: 2451–55
25. Lee SH, Lee HC, Oh SJ et al: Percutaneous intervention of spontaneous renal artery dissection complicated with renal infarction: A case report and literature review. Catheter Cardiovasc Interv, 2003; 60: 335–38
26. Dowling JM, Lube MW, Smith CP, Andriole J: Traumatic renal artery occlusion in a patient with a solitary kidney: case report of treatment with endovascular stent and review of the literature. Am Surg, 2007; 73: 351–53
27. Flugsrud GB, Brekke M, Roise O: Endovascular stent in the acute treatment of blunt renal arterial injury. J Trauma, 2005; 59: 243–45
28. Whigham J, Bodenhamer JR, Miller JK: Use of the Palmaz stent in primary treatment of renal artery intimal injury secondary to blunt trauma. J Vasc Interv Radiol, 1995; 6: 175–78