Bilateral traumatic renal artery dissection and thrombosis complicated by hypertension and renal failure

Mat Nor K1,2, Azad Hassan AR3, Gobal SV4, Abdullah BJJ*, 1,2

1 University of Malaya Research Imaging Centre, Faculty of Medicine, University of Malaya, Kuala Lumpur, Malaysia
2 Department of Biomedical Imaging, Faculty of Medicine, University of Malaya, Kuala Lumpur, Malaysia
3 Department of Surgery, Faculty of Medicine, University of Malaya, Kuala Lumpur, Malaysia
4 Department of Anesthesia, Faculty of Medicine, University of Malaya, Kuala Lumpur, Malaysia

Received 2 October 2012; received in revised form 1 April 2013; accepted 27 April 2013

ABSTRACT

We present a case of bilateral renal artery dissection (BRAD) following serious motor vehicle accident. This was complicated by arterial hypertension and acute renal failure due to a delay in diagnosis. We present the computed tomography (CT) finding as well as the therapeutic options and discuss the role of the radiologists in communicating important findings. © 2013 Biomedical Imaging and Intervention Journal. All rights reserved.

Keywords: bilateral renal artery dissection, arterial hypertension, acute renal failure, computed tomography.

INTRODUCTION

Renal artery thrombosis is an uncommon complication following blunt abdominal trauma. Bilateral renal artery dissection following a blunt force trauma is extremely rare with less than 20 cases reported in the literature. Early diagnosis is essential to salvage renal function and prevent lifelong dialysis/renal transplant and hypertension. Contrast enhanced is currently the modality of choice to evaluate renal vascular injuries especially in patients with trauma. Despite this many unilateral lesions remain undetected, in part because renal artery occlusions are often associated with serious other injuries. Treatment options vary depending on the severity of the patient’s clinical condition but endovascular procedures are now the preferred treatment to salvage renal function.

CASE REPORT

A 36-year-old man was brought to the emergency department after a serious motor vehicle accident. The patient sustained multiple fractures of left radial head, left proximal ulna, both lower ribs and right hip (with associated dislocation). A Foley’s catheter was inserted to monitor urine output and haematuria was noted. The patient’s remained stable except for acute onset hypertension (230/105 mmHg). The initial blood investigations were essentially normal (urea level of 4.7 mmol/L) though the creatinine was slightly elevated (133 µmol/L). Because of mechanism of injury associated abdominal pain and multiple rib fractures intra-abdominal solid injuries were suspected. A contrast enhanced CT thorax, abdomen and pelvis was performed. This revealed lung contusion, Grade II liver injury and Grade III splenic injury. There was no renal parenchymal enhancement or excretion with only the proximal renal arteries visible (Grade III renal injury) (Figure 1a and 1b). There was no associated para-aortic or perivascular haematoma. No capsular haematoma or parenchymal
contusion was seen either. The renal artery occlusions are consistent with bilateral renal arterial dissection with secondary thrombosis. No other vascular injuries were noted. The blood investigations at admission were essentially normal (Hemoglobin was 151.0 g/L while his urea was 4.7 mmol/L) except the creatinine was at the upper limit of normal (133 μmol/L).

Despite the best efforts to communicate the results to the attending urologists, they were unconvinced, as a bilateral renal artery injury is extremely rare. The patient was then transferred to intensive care unit (ICU) and electively intubated. Patient rapidly progressed to acute renal failure with minimal urine output where the urea had risen to 11.4 mmol/L while the creatinine was 422 μmol/L. An urgent abdominal aortogram and selective renal angiogram showed dissection with thrombosis of both renal arteries approximately 1.0 to 1.5 cm from the origins (Figure 2). A diagnosis of bilateral renal artery dissection was made. Recanalisation was attempted for both arteries but this failed. The urea and creatinine climbed to 46.0 mmol/L and 801 μmol/L respectively over the next 10 days of admission. He was started on haemodialysis in ICU.

**Figure 1** (a) Axial reconstruct contrast enhanced computed tomography scan of the abdomen at level of the right renal artery. Only proximal R renal artery opacified with abrupt contrast cut off of the right renal artery 1.0 cm from its origin (white arrow) (pedicle transection). Left kidney shows non-opacification. No extravasations of contrast or hemoperitonium seen to suggest any other vascular injury. (b) At level of the L renal artery similar appearance where there is sudden cut off of contrast seen 1.5 cm from the origin of left renal artery (white arrow) (left pedicle transection). There is perfusion of lower pole R kidney (bold black arrow) most likely from capsular vessels.

**Figure 2** (a) Flush aortogram shows normal abdominal aorta and its branches (splenic artery (arrowhead); common hepatic artery (thin arrow); SMA (bold arrow) apart from opacification of proximal portions of both renal arteries (asterisks). Selective renal arteriogram using a Cobra catheter of right (b) Showing occlusion at its origin (bold black arrow). There is a sub-intimal dissection as narrow irregular track of contrast (thin arrow). Right renal capsular vessels seen (arrow head). Left renal (c) shows similar occlusion at its origin (bold black arrow) with renal capsular vessels seen (arrow head).
He was transferred to the urology ward for regular dialysis. His blood pressure remained persistently high for approximately 3 weeks. His blood pressure was stabilized with oral Amlodipine and Prazosine. During the follow-up visit after two months, he was already on maintenance dose of oral metoprolol 150 mg TDS to maintain his blood pressure. His hemoglobin had dropped to 9.7 g/ml while the urea and creatinine hovered at 12 mmol/L and 400–500 µmol/L range at the time of discharge.

DISCUSSION

Renal artery thrombosis or rupture and renal parenchymal laceration or haematoma can lead to severe morbidity and mortality. Therefore, an extensive evaluation of renal trauma and understanding the mechanism of renal injuries at the initial trauma assessment will lead to appropriate treatment [1]. Blunt renal vascular injuries following abdominal trauma are rare and are reported in only 3–4% of patients [2–3]. Bilateral blunt renal artery injuries are extremely uncommon with less than 20 reported in the literature. The first case of bilateral traumatic renal artery thrombosis was reported in 1965 [4], even though the first case of traumatic renal artery thrombosis was reported almost a century earlier [5].

Majority of the renal injuries (> 90%) represent parenchymal lesion or segmental vessels injuries and most of the renal injuries result from blunt force trauma (motor vehicle accident or falls from height). Rapid deceleration, such as by high fall, high speed motor vehicle collisions, and pedestrians struck by motor vehicles, are the typical manner of injury [6]. According to Kawashima et al. study [7], 2.7% of patients sustained renal pedicle injury (including tearing and occlusion of both renal arteries and veins) with unilateral occlusions in 1.7% and there were no bilateral occlusion. In a study conducted by van der Wal et al [8], the overall mortality for patients who presented with haematuria was 44% and all the patients who sustained renal vascular injuries have associated vascular or organ injuries.

Complete thrombosis of the renal arterial system is usually the result of severe deceleration and is associated with multiple intra-abdominal injuries. Kawashima et al [7] have revealed that in most cases, unilateral renal artery thrombosis occurs and usually involves the left kidney. This is presumably due to its relatively long left renal pedicle, thus allowing the kidney more mobility. In addition, the left kidney also forms an acute angle with the abdominal aorta compared to the right renal pedicle.

Three sequential mechanisms have been proposed for renal thrombosis; stretch lesion (elastic components of adventitia and media could withstand the stretch mechanism but not a non-elastic intima) leading to intimal tear or intimal fracture followed by sub-intimal dissection subsequently leading to arterial thrombosis [9]. Additionally, contusion of the renal artery against the vertebral column and retroperitoneal haematoma (vascular compression) may lead to thrombosis.

The initial presentation can be non-specific with symptoms even suggestive of renal colic. In the absence of retroperitoneal haematoma, the immediate signs and symptoms of this disorder are mild and the associated injuries are frequently severe, correct diagnosis may be delayed for a considerable period of time. Clinical manifestations of BRAD include progressive renovascular hypertension, changes in renal function, and symptoms of kidney infarction, which were all noted in our patient. Because of its rarity and the non-specific symptoms, the diagnosis is often not considered until additional imaging studies are performed which was again demonstrated in our case.

CT imaging with intravenous contrast is the preferred modality to evaluate renal injuries in stable patients; specific attention should be concentrated on comparison of the immediate images with the delayed nephrogram images.

By using a multiphase renal protocol, the scan can delineate precise anatomic detail of renal injuries while selective arteriogram or venogram could provide details of vascular injuries that could be present. Vessel avulsion and thrombosis are evident on CT, but an arterial dissection that has to yet cause high-grade stenosis or thrombosis may be difficult to detect, so that angiography may be necessary [10]. Sudden termination of contrast enhancement within the affected artery (renal artery cut off sign) with or without contrast extravasation raises high suspicion of vascular injury such as main or segmental renal artery thrombosis or occlusion. Absence of renal parenchymal enhancement on CT nephrogram and/or contrast medium excretion (CT pyelogram) occurs when there is main renal artery occlusion or complete renal pedicle transection [7, 11, 12]. CT often shows non-enhancement of the kidneys with truncation of the renal arterial vessels though capsular supply will result some patchy enhancement. There is no excretion in the collecting system.

Further CT scan can identify the entire spectrum of renal injuries such as renal contusion, subcapsular haematoma (small or large), laceration (incomplete or complete), ureteropelvic junction disruption, shattered kidney, renal vein or artery thrombosis and avulsion of the kidneys. Angiography can precisely demonstrate the extent and nature of the vascular involvement while identifying potential treatment options. Angiography also serves as a baseline study to be used in comparison with follow-up examinations in case where successful recanalisation has been performed.

Evaluation and management of renal injuries have improved over several decades and large cohort studies were done (prospective and retrospective) giving rise to different categories and staging of renal injuries. The American Association for the Surgery of Trauma (AAST) (Accessible online at http://www.aast.org/injury/injury.html) in their extensive studies has developed an organs injuries-severity scale and for renal injuries. Kawashima et al has described the severity of renal injuries by its radiological appearance [7].
The prognosis of renal artery occlusion after blunt abdominal trauma is generally grave, both concerning renal function and survival from all the associated injuries (Hass 1998, Clark 981). Early identification of this injury is crucial since the warm ischemic time for the kidneys has revascularization has been reported up to 12 h after injury, but these cases with prolonged ischemia were attributed to collateral circulation from adrenal or capsular vessels; while still debated, a 4 h warm ischemic time is considered to be the limit [13]. Although early revascularization is predicted to yield the best results, restoring renal perfusion in less than 4 h does not guarantee a good outcome [14]. Surgical mortality rates are 9–20% due to the concomitant abdominal and thoracic injuries and have long term success rates of only 8.8–30% [14–16].

Endovascular stenting has been utilized over the last 15 years [17]; the National Trauma Data Bank reported that of 517 patients with blunt renal artery injuries, 13 patients (3%) were treated endovascula[18]. Although the published data are based only on individual cases or small case series, and it is not clear that the success rate is improved over open vascular repair, endovascular treatments potentially reduce surgical risks and may become the preferred treatment approach in patients not requiring urgent laparotomy due to other intra-abdominal injuries [19, 20]. Unilateral lesions may be treated conservatively, if the contra-lateral kidney is functioning properly, because revascularization in this situation has been found to yield poor results [16]. Only in cases of severe hypertension, a nephrectomy should be considered.

Effective communication is a critical component of diagnostic imaging. Quality patient care can only be achieved when study results are conveyed in a timely fashion to those responsible for treatment decisions. An effective method of communication should: (a) be tailored to satisfy the need for timeliness, (b) support the role of the interpreting physician as a consultant by encouraging physician communication, and (c) minimize the risk of communication errors [21]. The professional radiological organizations have formulated guidelines to prioritize the safe communication of critical test results in recent patient safety goals [22]. This push to speed the communication of results has spurred the creation of numerous automated critical results reporting systems with one goal: fast, accurate reporting of diagnostic results. However as has been exemplified by this case, even though efforts were made to communicate the findings to the surgical team, there was a failure to act on these findings due to a lack of confidence in the radiologist’s report resulting in significant morbidity. Not infrequently, referring physician as well as the radiologist, become codependants in malpractice claims for delay in diagnosis or treatment resulting from failure of communication.

Radiologists have traditionally remained “behind the curtain” when it comes to patient care, the voice without a face. However, there has recently been a surge of research questioning the radiologists’ traditional position in the care of patients and leaving the relaying of exam results to patients in the hands of clinicians [23]. Direct communication to patients by radiologists may improve patient care in a time when patients are more informed and educated about their healthcare. However in this instance, the patient would not have been able to comprehend the gravity of the situation and thus not provide any assistance in getting the appropriate treatment. Despite this it is known that lawsuits are generally less likely to occur or go to trial if a physician has developed a relationship with a patient. Efforts should be made to establish radiologists’ critical position in the health care system as intermediaries between ordering physicians and patients [23] with reevaluation of the established model of communication for reporting radiologic results.

CONCLUSION

We present a case of bilateral renal artery thrombosis secondary to blunt abdominal trauma secondary to a motor vehicle accident. We highlight the imaging features and the need to act rapidly on these to salvage renal function. Unfortunately there was a failure to act on the findings of the CT resulting in acute renal failure and hypertension. We also discuss the changing role radiologist should be taking in the communicating the imaging findings.

REFERENCES

15. Elliott SP, Ohweny EO and McNamich JW. Renal arterial injuries: