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Pictorial review

Traumatic renal artery dissection: from imaging to management



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ARTICLE INFORMATION

Article history: Received 24 April 2020 Accepted 25 August 2020 Injury to the renal artery following blunt trauma is detected increasingly due to widespread and early use of multidetector computed tomography (CT), but optimal treatment remains controversial as no guidelines are available. This review illustrates the spectrum of imaging findings of traumatic renal artery dissection based on our experience, with the aim of understanding the physiopathology of ischaemic damage to the kidney, and the process of choosing the best therapeutic strategy (conservative, endovascular, surgical). Five main patterns of traumatic renal artery dissection are described: avulsion of renal hilum; dissection of the segmental renal branches; preocclusive main renal artery dissection; renal artery stenosis without flow limitation; thrombogenic renal artery intimal tear. In the polytrauma patient, management depends on various factors (haemodynamic status, associated lesions, time of diagnosis) rather than on the degree of renal artery stenosis. Non-operative management (NOM) is the preferred option in case of non-flow-limiting dissection of the renal artery and angio-embolisation is an important adjunct to NOM in cases of active bleeding. Embolisation of the renal artery stump may be the best option in cases of occlusive dissection, as catheter manipulation carries a high risk of vessel rupture. The therapeutic window for kidney revascularisation in cases of flow-limiting dissection of main renal artery may be variable. Endovascular stenting >4 h after trauma should be performed only if residual flow with preserved parenchymal perfusion is detected at angiography. Antiplatelet therapy administration is recommended in cases of stenting, but conditioned by the bleeding risk of the patient.

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Introduction

Blunt trauma is the most common mechanism of renal injury, responsible for 95% of the cases in European studies, while penetrating trauma is more frequently reported in American studies.¹ Isolated kidney damage is not frequent, as concomitant injuries to other organs are identified in >80% of the patients admitted for polytrauma.² Only 5% of blunt renal trauma injuries have involvement of the renal vasculature³; the renal artery is more susceptible to

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dissection during rapid decelerations due to the mobility of the kidneys, which may cause a stretch lesion, leading to intimal tear or to intramural haematoma. Arterial dissection is a dynamic phenomenon that may evolve with time in various ways, as shown by studies of the pathogenic mechanisms in the carotid district.⁴

Renal artery dissections tend to heal spontaneously or with anticoagulant therapy, but in a subset of patients, the endothelial damage provokes thrombus formation at the level of the intimal flap and distal embolisation with peripheral infarcts. Other cases develop an acute arterial occlusion and kidney ischaemia, due to enlargement of the false lumen or to intramural haematoma. When vascular fibrotic remodelling leads to critical stenosis (>70%), there is progression to chronic kidney hypoperfusion, increasing the risk of delayed renovascular hypertension. If the vessel wall is ruptured, an unstable pseudoaneurysm may form, contained only by the adventitial layer, or active bleeding in the retroperitoneal space. In the case of rupture communicating with the venous vessels or with the urinary collecting system, there is formation of artero-venous or of artero-caliceal fistula, respectively.

Nowadays, multidetector computed tomography (CT) with contrast medium administration is performed routinely in cases of polytrauma and enables renal injuries to be divided into five main categories⁵ according to the American Association for the Surgery of Trauma (AAST)

grading system, which was originally based on the anatomical injury seen at surgery.⁶ The CT imaging criteria of the AAST classification of kidney injury was updated in 2018 to include renal vascular lesions⁷: grade III included arterial pseudoaneurysm, arterovenous fistula, active bleeding contained within Gerota's fascia; grade IV included segmental artery or renal vein injury, infarction as a sequela of vascular thrombosis, active bleeding breaching the perirenal space; grade V included laceration of the main renal artery or vein, kidney devascularisation with active bleeding. Although the 2018 revision facilitates the modern multidisciplinary management of renal trauma, providing better risk stratification,⁸ in the case of renal artery dissection, the therapeutic implications are debated because of the complexity of the patients, variable evolution of this pathology, and lack of large meta-analysis.^{5,9,10}

Over recent decades, the approach to renal injures has shifted towards non-operative management (NOM) due to accumulated knowledge regarding its safety and improved outcomes and involves close clinical observation, bed rest, serial laboratory monitoring. NOM is now applied in >90% of the haemodynamically stable or stabilised patients, even for IV–V AAST grades, once considered at higher risk of nephrectomy or dialysis. NOM failure and subsequent surgery (nephrectomy, renorraphy, vascular repair) occur in only 2–5% of all renal injuries.^{1–3} Successful NOM is reported in approximately 75% of AAST grade IV–V kidney

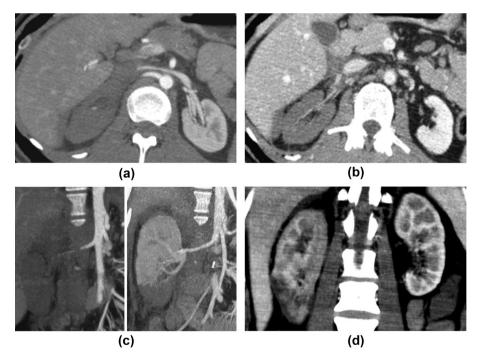


Figure 1 A 29-year-old man who had a skiing accident resulting in right renal pedicle avulsion (AAST grade V) associated with severe head trauma (GCS 5, broken helmet), multiple fractures, right lung contusions with pneumatoceles, and haemothorax, liver grade 2 laceration. (a) Arterial-phase axial CT shows occlusion of the main right renal artery due to dissection and hypoperfusion of the renal parenchyma. (b) Venous-phase axial CT depicts laceration of the right renal artery before (left) and after (right) surgical vascular repair, by means of longitudinal arteriotomy and suture with venous patch, shows good reperfusion of kidney except for the inferior pole. (d) Follow-up arterial CT acquired 10 days after trauma shows infarct of the inferior part is shown in the coronal reconstruction, but good nephrographic effect of the rest of the right kidney, which was only slightly delayed compared to the opposite side.

traumas,¹¹ with higher success rates in grade IV versus grade V injuries (89% versus 52%).¹² Interventional radiology plays a primary role supporting NOM, with proven efficacy of angio-embolisation in cases of active bleeding, pseudoaneurysm, and arterovenous fistula,^{13–15} but also through percutaneous drainage of fluid collections (abscess, urinoma, haematoma),^{3,16,17} and by means of endovascular treatment of renal artery dissections with angioplasty and/ or stenting.^{18–20}

The aim of this review is to focus the attention on the complexity of renal artery dissections through the illustration of the five main imaging patterns encountered in clinical practice by our trauma team, and to discuss how these cases were managed according to the recent literature recommendations.

Imaging patterns

Avulsion of renal hilum

Laceration of both the main renal artery and vein (Fig. 1) usually results in complete devascularisation of the kidney; it is uncommon after blunt renal trauma, but represents a life-threatening emergency. The peri-hilar haematoma is not necessarily large, but helps to distinguish renal pedicle avulsion (AAST grade V) from renal infarction due to vessel

thrombosis (AAST grade IV), which is not associated with active bleeding.⁸ The CT finding of contrast agent extravasation represents a factor that increases the likelihood of embolisation or surgery, whereas the size of the haematoma (considered predictive of NOM failure by some authors²¹) is not included in the AAST criteria. Half of AAST grade V blunt renal injuries, mainly those with "shattered kidney", may be selected for NOM if haemodynamically stable, with a renal salvage rate of 90%.¹¹

Immediate surgical exploration to attempt vascular repair is mandatory in renal pedicle avulsion, although 64% of patients undergo nephrectomy, as pre-hospital prolonged ischaemia usually results in irreversible damage and renal loss.²² Other indications to open repair are penetrating renal trauma, haemodynamic instability, or associated visceral organ damage requiring laparotomy. The patient illustrated in Fig. 1 was immediately brought to the operating room, and kidney revascularisation was achieved by the vascular surgeon approximately 4 h after trauma. The outcome was favourable with preservation of parenchymal viability, except for infarction of the lower pole: the serum creatinine returned to normal during hospitalisation, the renal artery was patent at follow-up Duplex ultrasonography, and the patient remained normotensive.

The kidney is extremely vulnerable to "warm ischaemia" damage, which begins 25–30 minutes after total renal artery

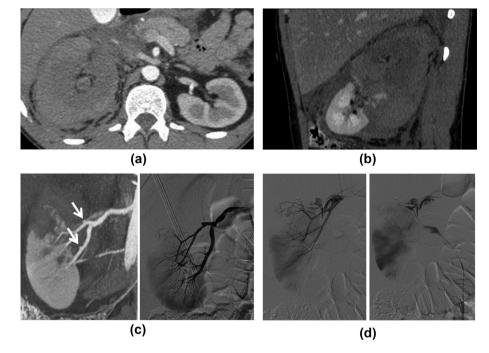


Figure 2 A 33-year-old man, who had fallen from the stairs, presented with right renal trauma (AAST grade IV) and was admitted with severe hypotension responsive to aemodynamic stabilisation. He was treated initially with NOM, fluid resuscitation, and blood transfusions. Arterial phase CT in the axial (a) and sagittal (b) plane, performed 2 days after trauma for persistent blood loss, shows a deep parenchymal laceration in the middle of the right kidney, hypodensity, and swelling of the its superior half due to partial infarction, surrounded by perirenal haematoma, contained within the Gerota fascia. (c) Dissections of segmental branches of the right renal artery are clearly depicted (arrows) on both the coronal MIP reconstruction of the CT angiography (left) and the selective renal arteriography (right) that was subsequently performed to attempt embolisation with microcoils. (d) Vessel perforation and development of artero-caliceal fistula occurred during the microcatheterisation of one segmental artery stump: active extravasation of contrast media is seen in the early phase (left) with sudden opacification of the calico-pyelic system and of the ureter in the late phase (right).

occlusion, according to surgical literature based on crossclamping during partial nephrectomy.²³ The time window for revascularisation before loss of renal function is variable depending on the residual flow, the severity of systemic hypotension, and the collateral circulation through the adrenal and capsular branches. In case of total main renal artery occlusion, successful revascularisation has been reported as long as 12 h after trauma,²⁴ but the ischaemic time limit should probably not exceed 4 h,²⁵ as only 26% of surgical cases with excellent technical repair preserve renal function.²²

Dissection of segmental renal arteries

Partial kidney infarction must raise suspicion for traumatic dissection of the segmental arteries, often due to deep parenchyma lacerations (Fig. 2). Maximum intensity projection (MIP) images in arterial phase CT, reconstructed at 10 mm thickness or more, are the best method to identify the abrupt interruption of the segmental branches due to dissection, which represents a protective mechanism to stop active bleeding.⁵ The patient in Fig. 2 with AAST grade IV renal injury was initially treated with NOM, after stabilisation with blood transfusions according to current guidelines.¹³

The perirenal haematoma was contained by the fascia and stable at control CT 2 days after trauma, without signs of active bleeding, but there was persistent haematuria and progressive decrease of the haematocrit. Angiography was therefore performed in order to embolise using coils the truncated segmental renal arteries, which were considered a potential source of intermittent bleeding.¹⁵ During catheterisation, the patient suddenly became hypotensive, because of dissection reopening and the development of a dangerous artero-calyceal fistula: an occlusion balloon was selectively placed in the main renal artery to achieve temporary haemostasis until emergency nephrectomy was performed. The patient was discharged from hospital in good condition, his serum creatinine was normal, and after 5 years, was diagnosed with essential hypertension, not related to the previous trauma.

This case highlights the risk for the interventional radiologist of dealing with arterial dissections: perforation of the damaged vessel during catheter or guidewire manipulation can precipitate the haemodynamic status and necessitate prompt occlusion with coils, vascular plugs, or



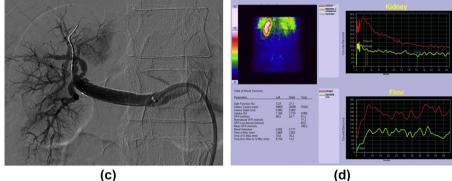


Figure 3 A 40-year-old man with right flank pain after injury with an axe. (a) Coronal multiplanar reconstruction (MPR) of the arterial phase CT demonstrates preocclusive dissection of the main right renal artery due to intramural haematoma and diffuse kidney hypoperfusion, without retroperitoneal haematoma. (b) Selective arteriography shows long critical stenosis, with residual anterograde flow in the main renal artery and patency of the intraparenchymal branches; also collateral circulation through the adrenal artery originating at the beginning of the renal artery is seen. (c) After fenestration of the dissection with a microwire and placement of a self-expandable nitinol stent (Protegè RX carotid stent: 8×40 mm), selective arteriography shows restoration of normal vessel calibre and better opacification of the intraparenchymal branches. (d) Tc-99m DTPA renal scintigraphy, performed 4 weeks later, shows reduced perfusion and concentration of the right kidney in the dynamic curves, but preserved split function (27% versus 73% in the left side).

balloons.^{13,15,18} Antibiotic prophylaxis is recommended in patients with large renal infarcts because perinephric abscesses and infected urinoma or subcapsular haematoma may occur more frequently²; percutaneous drainage of these fluid collections is an important adjunt to NOM, and also to avoid renal compression (Page kidney).¹⁷ Renovascular hypertension is a rare delayed complication, due to kidney ischaemia or compression, to arterial stenosis, or to arteriovenous fistula, which upregulate the renin-angiotensin system³⁷: long-term clinical follow-up must therefore be undertaken, particularly for higher AAST grade renal injuries with vascular compromise.²⁰

Preocclusive main renal artery dissection

Isolated renal artery dissection after minor trauma (Fig. 3) is a rare event, often unrecognised because the nonspecific clinical presentation with acute flank pain and haematuria, which are easily mistaken for renal colic, that leads to delayed diagnosis.²⁷ In the case presented in Fig. 3, contrast-enhanced CT was performed the day after the trauma for persistence of pain: critical long stenosis of the right main renal artery due to dissection with intramural haematoma and hypoperfusion of the kidney was observed (AAST grade V). After vascular surgeon consultation, it was decided to attempt the endovascular approach to save the kidney, as there was residual antegrade flow without thrombosis of the intraparenchymal branches. The time from injury to revascularisation is not considered a limitation in cases of partial renal artery occlusion, considering the variability of dissection phenomenon, with reported good outcomes even days after trauma.²⁸

Renal artery stenting with successful kidney revascularisation was achieved in this case nearly 24 h after trauma. Tc-99m pentetic acid (DTPA) renal scintigraphy is the reference standard to evaluate residual renal function: evaluation of the dynamic curve confirmed preserved right kidney perfusion with split renal function of 27% (above the cut-off value of 25% for renal salvage).²⁹ The patient was discharged normotensive with dual antiplatelet therapy (100 mg acetylsalicylic acid [ASA] + 75 mg clopidogrel) for 1 month and then a single agent (100 mg ASA) for at least 3 months; antiplatelet therapy is essential to prevent stent thrombosis³⁰ and was found to be a predictor of clinical success.²⁸ During follow-up, the nephrologist found hypertension 4 months later, which was controlled pharmacologically with two drugs. CT at 6 months demonstrated regular patency of the stent and symmetric nephrographic effect, but the right kidney was moderately atrophic, with cortical thinning and multiple scars.

Endovascular treatment with stenting is now the preferred option in cases of flow-limiting main renal artery

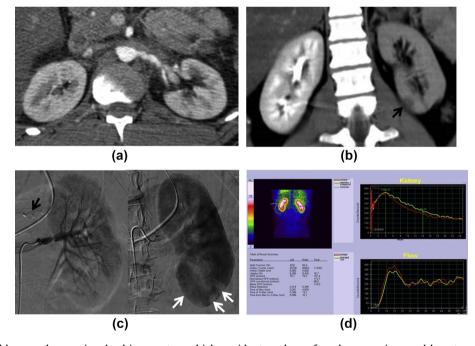


Figure 4 A 27-year-old man who was involved in a motor-vehicle accident, and was found unconscious and hypotensive, sustained left renal artery dissection, cerebral contusion (GCS 8), and spleen laceration with subcapsular haematoma. (a) Arterial phase axial CT shows irregularity of the left main renal artery with stenosis in the distal third, surrounded by peri-hilar fat stranding due to vessel laceration without active extravasation, minimal asymmetry of parenchymal enhancement. (b) Excretory phase coronal 8 mm-thick reconstruction shows delayed nephrographic effect of the left kidney and a wedge-shaped hypodense area at the inferior pole (black arrow), representing a small cortical infarct. (c) Left renal arteriography shows the concentric 60% stenosis without flow limitation in the early phase (right) and some cortical infacts of the inferior pole (white arrows) due to peripheral embolisation in the parenchymal phase (left). The Amplatzer vascular plug positioned in the splenic artery for proximal embolisation is also seen (black arrow). (d) Tc-99m DTPA renal scintigraphy 3 weeks later demonstrated bilateral regular parenchymal uptake and excretion of the radiotracer with symmetric kidney function.





Figure 5 A 58-year-old man, who was involved in a motorcycle accident, sustained thoracic—abdominal trauma with rib fractures and pneumothorax, unstable pelvic ring fracture, splenic (AAST grade I), and left adrenal contusion. (a) Arterial-phase axial CT image shows the intimal flap in the middle left renal artery without subintimal haematoma. (b) Coronal CT 10 mm-thick MIP image shows some wedge-shaped parenchymal non-enhancing areas compatible with cortical infarcts, due to peripheral embolisation (segmental arteries appear normal). (c) Left renal arteriography demonstrate intimal tear and endoluminal flap protrusion, without flow limitation. (d) The thrombogenic intimal tear was secured with placement of a balloon-mounted stent (Express LD: 7×27 mm) to avoid further embolisation or development of false lumen with vessel stenosis.

dissection, as invasive surgical repair has a poor long-term outcome.¹⁸ Simple balloon angioplasty is not useful in cases of renal dissections as they have a tendency to recoil after simple dilatation.¹⁰ Stent-grafts or covered stents are recommended only in cases of vessel laceration with contrast medium extravasation, being more thrombogenic than bare metal stents.^{32–34} Partial kidney devascularisation is not considered a treatment priority by current guidelines, although some cases of segmental or accessory artery stenting are reported.^{19,32,35} NOM of traumatic renal artery occlusion may represent a viable option in cases of functioning contralateral kidney, but carries a high risk to develop nephrovascular hypertension up to 32%, whereas for patients treated with revascularisation, the risk is reduced to 12%.³⁶ The onset of renovascular hypertension is usually between 2 weeks and 8 months after renal injury and sometimes resolves spontaneously, but if severe and refractory to medications, may require nephectomy in a minority of patients (approximately 10%).³⁷

Renal artery stenosis without flow limitation

Focal renal artery stenosis after traumatic dissection (Fig. 4) may occur immediately or as a late complication,

due to fibrotic vascular remodelling. Angiography is useful to investigate stenosis when kidney hypoperfusion is suspected at CT, because dynamic information about the residual flow is provided and CT has superior spatial resolution to calculate the location and the degree of stenosis: this information is crucial for choosing the therapeutic strategy. NOM with anticoagulation is the preferred option in cases of non-critical stenosis (<70%) without flow limitation, because the dissection tends to resolve spontaneously in the majority of cases.^{30,38,39} Renal artery stenting implies single or, better, dual-antiplatelet therapy to avoid stent thrombosis, but increases the risk of bleeding in cases of associated injuries.³¹

This polytrauma patient illustrated in Fig. 4 had concomitant spleen laceration with subcapsular haematoma (AAST grade II) and head trauma; therefore, antiplatelet therapy was avoided. Angiography enabled proximal embolisation of the splenic artery with an Amplatzer vascular plug and demonstrated that the renal artery stenosis was not flow limiting. Thus, only anticoagulant therapy with low-molecular-weight heparin at the prophylactic dosage was administered during NOM without the need of endovascular stenting. Before hospital discharge 3-weeks later, renal scintigraphy demonstrated normal left kidney function. Follow-up CT 3 months later showed regression of the stenosis and regular renal artery profile. The patient did not develop hypertension during follow-up.

Thrombogenic renal artery intimal tear

Intimal tear (Fig. 5) is usually the first event in renal artery dissection as the intima is not elastic as the muscular tunica. The intimal flap inside the renal artery represents a weakness that may lead to thrombus formation and peripheral embolisation with small infarcts, as already described.⁴⁰ A delayed phase CT acquisition may be useful to differentiate a small segmental infarction, seen as a wedge-shaped area of decreased contrast enhancement with sharp margins, from a parenchymal contusion, representing a small intra-renal haematoma and usually has a round shape with ill-defined margins: infarcts remain hypodense during the excretory phase, whereas contusions tend to become isodense or manifest as focal areas of striation.^{5,17}

The treatment of this type of renal dissection is controversial as anticoagulation is the most commonly used therapy,³⁹ while intervention with endovascular stenting is suggested only in cases of flow-limiting dissection or failure of medical theraphy.⁴¹ In the context of multidisciplinary management for the patient illustrated in Fig. 5, the unstable lesion was secured immediately with a bare metal stent, to prevent further embolisation and to avoid dissection evolution towards vessel stenosis or thrombosis, because the patient had to undergo orthopaedic surgery interventions to stabilise the pelvis under general anaesthesia, with the risk of intra-operative hypotension. Antiplatelet therapy was started only 1 day after surgery: a single agent (100 mg ASA) associated with prophylactic anticoagulation was used instead of dual antiplatelet therapy. Follow-up CT demonstrated a patent renal stent 3 months later.

Conclusion

NOM with the adjunct of minimally invasive procedures, such as angio-embolisation or endovascular stenting, is currently considered the treatment of choice for all the haemodynamically stable or stabilised blunt renal injuries.¹⁴ The AAST kidney injury grade based on CT and the haemodynamic status of the patient are important to guide the decisional algorithm,^{13,16} but other factors that influence the overall prognosis must be considered in cases of renal artery dissection (time of diagnosis, associated lesions, possibility of anti-aggregation).^{11,19,31}

The therapeutic time window for revascularisation with stent placement may be variable depending on the degree/ location of arterial stenosis, on the collateral circulation, and also on the dynamic evolution of the dissection. Selective renal angiography may be a useful decision-making tool to assess residual flow and the patency of intraparenchymal vessels. Angiographic manipulation of the renal dissection carries a high risk of rupture and it should be attempted cautiously. Arterial stump embolisation may be the best choice in cases of persistent haematuria or vessel perforation during the endovascular procedure.

Conflicts of interest

The authors declare no conflict of interest.

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