Renal Artery Embolism and Renal Vein Thrombosis

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Renal Artery Embolism and Infarction

Renal artery thrombosis was first described by Von Recklinghausen in 1861 [1]. The first successful revascularization was performed by Rohl in 1971 [2]. With the advent of diagnostic modalities such as spiral computed tomography (CT) and magnetic resonance imaging, as well as the development of thrombolytics and percutaneous methods of embolectomy, the clinical approach to this condition has changed significantly over the past 30 years. Although prospective studies are lacking because of the relative rarity of this condition, an accumulation of retrospective data on treatment and outcomes exists and can provide some guidance toward optimal therapy.

Renal artery occlusion has many etiologies, some of which are listed in Table 12.1. In general, these can be divided into spontaneous, traumatic, and iatrogenic causes, which present differently and have varying approaches to management. We therefore discuss each of these categories of renal infarction separately.

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Spontaneous Renal Infarction

Cardiovascular disease is the most common predisposing condition leading to spontaneous renal infarction. Reviews have shown that 65 % of affected patients had a history of atrial fibrillation, 53 % had hypertension, and 41 % had evidence of ischemic heart disease [3]. Other medical conditions that increase the risk of spontaneous renal artery embolism and infarction include hypercoagulable states, renal artery aneurysm, and inflammatory disorders such as polyarteritis and fibromuscular dysplasia.

Incidence and Presentation

The true incidence of spontaneous renal infarction is unknown. Autopsy studies have found an incidence of 1.4 % [4], although clinically significant events are uncommon, occurring in an estimated 6.1 per million hospitalized patients [5]. Common presenting symptoms in patients with renal artery thrombosis include abdominal or back pain, nausea/vomiting, costovertebral angle tenderness, and hypertension. The most common symptom is flank pain, found in 65–77 % of cases [3, 6]. These symptoms may masquerade as renal colic, pyelonephritis, cholelithiasis, lower back disease, and myocardial infarction; often an embolic event to the kidney is not suspected. Acute hypertension at presentation is thought to be renin-mediated [7].

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Table 12.1 Etiologies of renal infarction

Spontaneous renal infarction
Cardiovascular
Atrial fibrillation
Ischemic heart disease
Mitral stenosis
Endocarditis
Atherosclerosis
Hypertension
Autoimmune
Polyarteritis nodosa
Systemic lupus erythematosis
Fibromuscular dysplasia
Behcet's disease
Henoch–Schonlein purpura
Other
Drug abuse
Hypercoagulable states
Malignancy (e.g., bronchial cancer)
Chagas disease
Polycythemia vera
Acquired renal infarction
Trauma
Blunt injury
Penetrating injury
Surgical procedures (Iatrogenic)
Renal transplantation
Cardiac valve repair
Endovascular stenting
Angiography
Interventional polymers

Diagnosis

Laboratory investigation plays a limited role in the diagnosis of renal infarction. Microscopic hematuria has been reported in 60–80 % of cases, and pyuria less frequently. Leukocytosis has been reported in 71 % of identifiable cases [6]. Most patients at presentation have or will develop an elevated serum lactate dehydrogenase (LDH) as renal tissue becomes nonviable. While highly sensitive, LDH has poor specificity in this context. Serum creatinine may be elevated, but existing data are unclear given the high rate of nephrotoxic contrast exposure in these patients, who are often imaged at presentation [7]. Although inconsistently elevated, other laboratory tests that may be found to be abnormal in renal infarction include aspartase aminotransferase, alkaline phosphatase, C-reactive protein, and fibrinogen [3].

Renal artery occlusion should be suspected when a patient at an increased risk for thromboembolism presents with the aforementioned signs and symptoms. Diagnostic confirmation can be achieved using arteriography, intravenous urography (IVU), radionuclide scintigraphy, magnetic resonance imaging, or ultrasound, but the most commonly utilized mode of reliable, rapid identification of arterial occlusion has been CT [6]. Although all highly sensitive, radionuclide scintigraphy and IVU are rarely used in this context today, and the invasiveness of renal arteriography prevents its use as a first-line study. A more current diagnostic strategy for suspected renal infarction is CT with and without IV contrast and consideration of MR angiography in those for whom IV contrast cannot be administered.

Radiological findings of an embolus include a filling defect in the renal artery and lack of enhancement of the affected kidney. In some cases, an abrupt cutoff of an enhancing renal artery may be seen in the presence of normal renal contour and a central renal hematoma [8]. Spontaneous renal emboli appear to involve the left kidney more frequently, which is attributed to the more acute angle of the left renal artery off the aorta.

Treatment

Standard treatment strategy includes anticoagulation with or without thrombolytic therapy. Percutaneous or surgical interventions are more often considered in cases of solitary kidney, bilateral involvement, complete artery occlusion, or failure of medical management. Outcome literature has been limited to case reports and retrospective reviews, and controversy still exists regarding the optimal choice and timing of treatment.

Nonoperative management for spontaneous renal infarction includes anticoagulation and fibrinolytics. Patients are typically anticoagulated with IV heparin and oral warfarin to prevent further embolic events. Thrombolytic therapy can be given systemically or locally, although local infusion with selective catheterization is the preferred approach. Typical local thrombolytic therapy involves continuous infusion of an agent such as streptokinase, urokinase, or

tissue plasminogen activator to the affected artery. There are data to suggest that thrombolytic therapy does not improve outcomes once the ischemic tolerance of the kidney has been exceeded. This is thought to occur after roughly 180 min of ischemia, although the length of time to irreversible ischemic injury is debated [9]. It is generally accepted that, under physiological temperatures, a kidney becomes nonviable following 60–90 min of total circulatory arrest [10]. Both animal studies and human retrospective reviews, however, have shown persistent renal function in longer intervals of occlusion. This variability is thought to be due to the frequent presence of collateral circulation.

Surgical repair in cases of renal infarction with a normal contralateral kidney remains controversial given the higher morbidity of open revascularization and lack of evidence supporting benefits in terms of renal function. It is, however, preferred for patients with traumatic renal artery thrombosis when surgery is done within the first few hours after injury. Those supporting immediate surgical correction via open thromboembolectomy or bypass grafting argue that renal salvageability cannot accurately be assessed, and thus all efforts should be made for immediate restoration of blood supply [11]. Surgical techniques that have been described include thrombectomy with end-to-end reanastamosis, autotransplantation, or aortorenal saphenous vein bypass graft, among others. Some investigators have suggested that attempt at surgical revascularization should be made if the presumed warm ischemia time is less than 5 h.

In cases of bilateral renal artery occlusion or renal infarction in a solitary kidney, attempt at surgical or thrombolytic revascularization is indicated regardless of ischemia time. With success defined as renal function able to sustain life without dialysis, Lohse et al. reported that successful surgical revascularizations were performed in 4 of 10 patients with bilateral renal artery thromboses [12]. Similarly, there have been reports of restoration of renal function in bilateral renal artery thrombosis using thrombolytic therapy [13, 14].

Percutaneous thromboembolectomy has been shown to be a viable option for renal artery thrombosis. Successful rheolytic aspiration via hydrodynamic catheterization has been described for renal artery and other visceral emboli [15, 16]. This and other minimally invasive, mechanical techniques hold promise and may replace surgical embolectomy in select patients.

Prognosis

In the most recent outcome review of renal artery embolism, Kansal et al. showed that renal function outcomes are favorable in the majority, with 57 % of patients regaining normal renal function and 16.7 % having mild renal impairment with creatinine less than 2 after treatment. Mortality was typically a result of recurrent embolic disease or heart disease and not due to renal complications [7].

Acquired Renal Artery Infarction

Renal Infarction Caused by Surgical Procedures

Renal transplantation and other procedures involving vascular anastomoses predispose patients to the risk of renal artery occlusion. In a review of complications of more than 1,200 renal transplants, Osman et al. found that 0.4 % of transplants are complicated by arterial thrombosis, and that ischemia and complications of treatment can lead to transplant loss [17].

The diagnosis of vascular occlusion can be challenging. Late renal artery stenosis presents with worsening hypertension and decreased renal function. Early arterial occlusion typically presents with acute oliguria, with Doppler ultrasound images suggestive of poor perfusion. In this setting, the differential diagnosis includes acute rejection, acute tubular necrosis, cyclosporine toxicity, and renal vein thrombosis (RVT) [18]. Diagnosis is confirmed with arteriography, and interventional techniques can be employed acutely.

Renal artery complications after transplant can also be managed with open revision. Takahashi et al. recently reviewed the diagnosis and management of renal allograft perfusion failure caused by dissection [19]. The authors believe interventional techniques such as stenting are

superior to open revision because of decreased treatment-related complications and less chance for graft loss.

Percutaneous endovascular procedures, such as endovascular stenting, have also led to renal embolic events. In a review of complications related to renal artery stenting for renal artery stenosis, Ivanovic et al. found that 2.6 % of patients undergoing these procedures suffered from renal artery thrombosis in the postoperative period [20].

Renal Infarction in the Setting of Trauma

Renal infarction secondary to trauma can result from either penetrating or blunt injury. Although the mechanism of injury is clear for penetrating wounds, the etiology of renal occlusion in blunt trauma is not completely understood. Most investigators believe that rapid deceleration leads to stretching and subsequent disruption of the intimal layer of the renal artery, with resultant thrombosis. This thought is supported by findings that the left kidney, which is less supported by surrounding organs and thus more susceptible to stretch injury and intimal tearing, is more frequently involved than the right [21]. Others suggest that direct trauma to the artery accounts for the thrombotic event. Renal artery thrombosis resulting from trauma is infrequent, with an incidence of 0.1 % in all blunt abdominal trauma admissions [22] and only about 400 cases reported in the literature [23].

There are several considerations to be made specific to cases of renal infarction caused by trauma. First, there is the emergent need to exclude a main renal artery laceration and other intra-abdominal injuries that may indicate immediate exploratory laparotomy. Thus, in a patient stable enough to undergo imaging, spiral CT is preferred over possibly more time-consuming studies such as arteriography. If an occlusion is discovered, the decision for immediate vs. delayed operative intervention may be obviated by a concomitant intra-abdominal injury for which the general surgeons will explore the patient.

When exploring trauma patients with suspected renal vascular injuries, early vascular control is imperative. McAninch and Carroll found that early vascular control during explorations for renal trauma reduced nephrectomy rates from 56 to 18 % [24]. Specific techniques that may be required for renal arterial injuries include direct repair or resection with end-to-end anastomosis or bypass grafting using vein or synthetic material [25].

Renal Vein Thrombosis

RVT predominantly affects two subpopulations: neonates with risk factors for clotting abnormalities and adults with nephrotic syndrome. We consider the diagnosis and treatment options of these two populations separately, discussing the disease process in adults first.

Etiology

The most common medical cause of RVT in adults is nephrotic syndrome. This association was identified as early as 1840, when Rayer described thrombosis of the renal veins and inferior vena cava in a patient with proteinuria [26]. In nephrotic patients, the combination of low serum albumin, high fibrinogen levels, low antithrombin III levels, and hypovolemia predisposes to the development of thrombotic disease [27]. Membranous glomerulonephritis has been identified as the most common nephrotic state resulting in RVT, accounting for up to 62 % of cases [28]. Other hematological abnormalities that have been inconsistently described include platelet hyperaggregability, thrombocytosis, and elevations of proteins C and S.

Presentation

Like renal artery occlusion, the diagnosis of RVT is commonly missed, especially in the presence of a normally functioning contralateral kidney. The finding is usually made when a patient with clotting abnormalities presents with worsening renal function, flank pain, or peripheral edema. In some instances, patients are not identified until they present with complications of the renal thrombus and hypercoagulable state, such as pulmonary embolism.

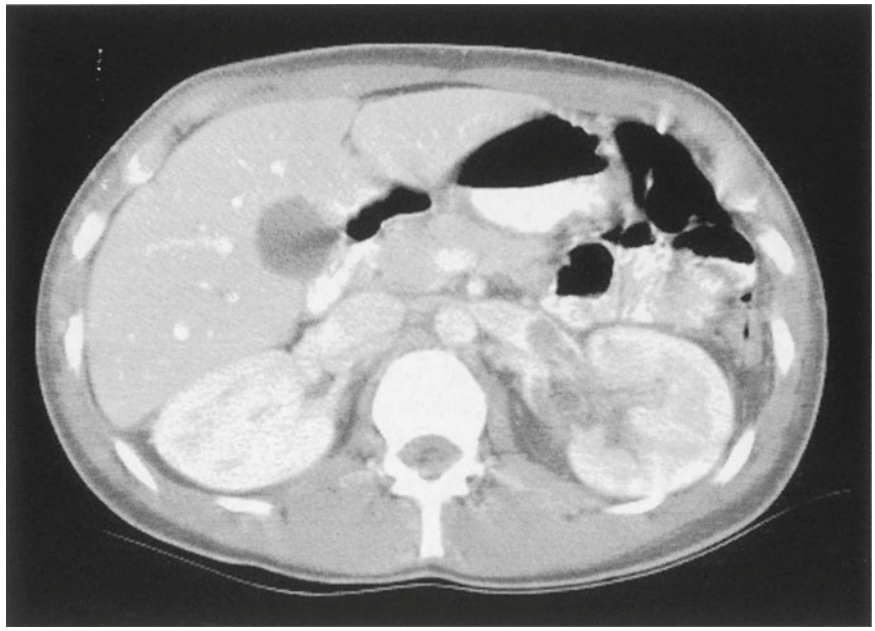

Fig. 12.1 Left main renal vein thrombus in a 27-year-old woman with the nephrotic syndrome

It should be noted that symptoms of flank pain, costovertebral angle tenderness, worsening proteinuria, and hematuria may be absent in the majority of cases. McCarthy et al. found that local symptoms were present in only 34 % of patients with RVT [29]. More commonly, the presentation is related to the nephrotic syndrome, with progressive ankle swelling and mild-to-moderate deterioration in renal function. In a prospective screening evaluation of 151 nephrotic patients, 33 had RVTs and 29 of these patients had peripheral edema only, suggesting that the majority of cases go undiagnosed or are diagnosed at a later time [30].

Diagnosis

Given the lack of specific clinical manifestations and diagnostic laboratory tests, imaging is the cornerstone of diagnosis of RVT. CT with intravenous contrast is the current imaging of choice, with sensitivity and specificity nearing 100 % (Fig. 12.1) [31]. It also allows for identification of other renal pathology. Magnetic resonance angiography (MRA) is an alternative modality that avoids radiation exposure and nephrotoxic contrast, but is costlier, more time-consuming, requires anesthesia in certain populations, and has marginally inferior sensitivity and specificity compared to CT [32]. Renal ultrasound lacks sensitivity for segmental thromboses, but has been used extensively in renal transplant recipients to detect RVTs and flow in the renal vein and artery. IVU may show swelling of the kidney from venous congestion, as well as a notching of the ureters or renal pelvis from impingement of dilated, tortuous collaterals vessels [33]. However, these findings are neither specific nor sensitive for RVT, thus IVU is rarely used in settings where more advanced imaging is available. If an interventional radiological procedure is elected, inferior venacavography and selective renal venography remain the gold standard.

Therapy

The rationale for immediate intervention in RVT in adults is to prevent further thromboembolic events and to maintain renal function. In the past, the treatment of RVT was primarily surgical, involving thrombectomy or nephrectomy. Surgical management is now rare, and anticoagulation with systemic unfractionated heparin followed by outpatient management with warfarin has

Table 12.2 Indications for thrombectomy/thrombolysis in RVT[a]

Refractory to medical intervention
Bilateral RVT
Solitary kidney
Contraindication to systemic anticoagulation
Renal transplantation
Intractable pain
Onset of complications (e.g., pulmonary embolus)

[a]Adapted from Ashgar et al. [31], with permission

been the standard treatment to prevent propagation of the thrombus [34]. The use of low-molecular-weight heparins also has been described, with the advantage of increased bioavailability, longer half-life, and potentially fewer drug interactions compared to warfarin [35]. A disadvantage of low-molecular-weight heparin is the difficulty in reversing anticoagulation should complications caused by therapy arise. Duration of anticoagulation varies from a minimum of a year to lifelong, depending upon recurrence of RVT or continued presence of risk factors [31].

Mechanical thrombectomy and/or thrombolysis via percutaneous intervention are suitable in select cases (Table 12.2). Chemical thrombolysis with agents such as streptokinase, urokinase, and recombinant tissue plasminogen activators [36] can be administered systemically or locally, although administration via catheter is associated with fewer systemic side effects. More recent advances have been made with mechanical thrombectomy via sheath access of the femoral vein under fluoroscopic guidance. In select populations, percutaneous catheter-directed thrombectomy with or without thrombolysis for acute RVT is associated with a rapid improvement in renal function and low incidence of morbidity [37].

Prognosis

The prognosis of RVT in adults has been dependent on several factors, including the presence of preexisting renal insufficiency and type of nephropathy. In a review of 27 nephrotic patients with RVT, Laville et al. reported 40 % mortality within 6 months of diagnosis with RVT, primarily due to pulmonary embolism, hemorrhagic complications, or the patient's underlying disease process. In those who survived, 12 of 16 (75 %) had stable renal function at follow-up [38]. The most important prognostic factor associated with favorable outcome was normal baseline renal function. This study also confirmed increased survival in patients with membranous glomerulonephritis compared to those with other forms of the nephrotic syndrome, such as minimal change disease and focal segmental glomerulosclerosis.

Renal Vein Thrombosis in Children

Etiology

In the pediatric population, RVT is primarily a neonatal disease, with 83 % of cases in children occurring within the first month of life [39]. It is the most prevalent non-catheter-related thromboembolism during the neonatal period [40]. The pathophysiology is assumed to be decreased renal blood flow in a child with preexisting risk factors for thrombus formation. These include severe dehydration, hypotension, cardiac disease, polycythemia, protein C deficiency, and factor V Leiden heterozygosity. Additional prenatal and perinatal risk factors include preeclampsia, maternal diabetes, traumatic birth, and fetal distress.

Presentation

Presentation of infant RVT has been classically described as the "diagnostic triad" of palpable abdominal mass, gross hematuria, and thrombocytopenia. While uncommon for all three signs to be present, a recent review showed the majority had at least one of the cardinal signs present: 56 % with gross hematuria, 45 % with palpable abdominal mass, and 47 % with thrombocytopenia [41]. Male children are affected twice as often as females and the left renal vein is affected twice as often as the right [42]. Clinical suspicion should thus also include maternal and infant risk factors, and the presence of a combination of these should warrant an investigation for RVT.

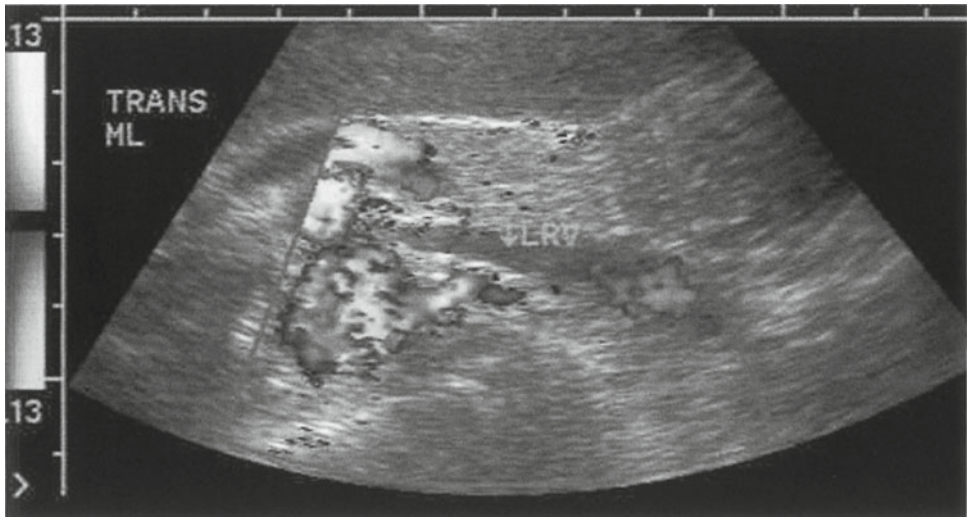

Fig. 12.2 Doppler ultrasound image of an infant with left renal vein thrombosis

Diagnosis

Once clinical suspicion is established, the best diagnostic test of RVT in children is Doppler ultrasound (Fig. 12.2). This modality is highly sensitive in infants, detecting 22 out of 23 cases in a modern study [39]. If non-diagnostic, a CT scan may be utilized. The radiological appearance of neonatal RVT includes renal enlargement and ischemia and can be confused with other causes of renal enlargement such as Wilms' tumor, pyelonephritis, and renal abscess. The radiological appearance depends on the acuity of the radiological study, because neonatal RVT evolves in appearance. Early, the kidney swells and appears echogenic on ultrasound; later, the appearance becomes more heterogeneous, with a loss of corticomedullary differentiation [43].

Treatment

Children with RVT have also been treated successfully with anticoagulation. The use of a thrombolytic agent such as recombinant tissue-type plasminogen activator has been reported in the literature [44], although intraventricular hemorrhage is a significant risk in this population, especially in the case of premature infants where deaths have been reported [45].

There are no evidence-based guidelines nor consensus for the management of neonatal RVT, although the most recent review of practice patterns shows that 40 % of patients received supportive care, 41 % received heparin or low-molecular-weight heparin, 11 % received thrombolytic therapy, and the rest some combination thereof [37]. A review of 23 children with a mean follow-up of 42 months showed that patients who did not receive anticoagulation all developed renal function impairment, while reduced renal function was seen in only 33 % of those who had received heparin [39]. However, more recent reviews demonstrate irreversible renal damage of upwards of 70 % at follow-up, regardless of anticoagulation having been administered. Long-term hypertension is found in 19 % of patients, affirming the need for close surveillance of children with RVT [40].

In summary, RVT should be suspected in neonates who have maternal or fetal risk factors with associated abdominal mass, hematuria, or thrombocytopenia. It should also be considered in adults with nephrotic syndrome who present with symptoms of colic, especially in those with membranous nephropathy. Treatment consists of supportive care and anticoagulation, with consideration of thrombolytic therapy or percutaneous interventions in select cases. With this regimen, mortality and morbidity of this condition can be minimized.

Summary

Renal artery embolism or thrombosis should be considered in the differential diagnosis of a patient with acute flank pain, particularly patients with risk factors such as atrial fibrillation, hypercoagulable disorders, or autoimmune disorders. Clinical suspicion is the key to prompt diagnosis and appropriate therapy, usually with anticoagulation or directed thrombolytic therapy.

Acquired renal artery infarction can occur from trauma or from surgical procedures such as renal transplant or vascular procedures. The specific treatment depends on the clinical situation and may be anticoagulation, endovascular procedures, or open thrombectomy or revision.

RVT occurs primarily in two populations: adults with nephrotic syndrome and infants with hypercoagulable conditions. Adults with nephrotic syndrome may present with flank pain or worsening renal function and edema. Diagnosis is usually by CT. Therapy is usually with systemic anticoagulation. The prognosis depends on a patient's renal function and whether other thrombotic complications can be avoided.

Pediatric RVT occurs most commonly in newborns of diabetic mothers or with risk factors for thrombosis such as dehydration, polycythemia, or protein C deficiency. The presentation is classically hematuria, palpable abdominal mass, and thrombocytopenia. The diagnosis is usually established with renal Doppler ultrasound. Treatment is anticoagulation or directed thrombolytic therapy, although the risk of cerebral hemorrhage is present, particularly in premature infants.

Through clinical suspicion, appropriate diagnostic testing, and prompt therapy, the late sequelae of renal vascular emergencies can be minimized.

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