Role Of Computed Tomography In Perirenal Haematoma; A Pictorial Review

S Lai, M Spanger

Citation

Abstract
Perirenal haematoma (PRH) is a rare but potentially life-threatening condition which may be traumatic, iatrogenic or spontaneous in nature. Computed tomography (CT) imaging has been shown to have a high sensitivity for detection of PRH and identification of its underlying aetiology. In this article, the CT appearance of the most common aetiologies of PRH is presented.

INTRODUCTION
Perirenal haematoma (PRH) is a potential abdominal surgical emergency, requiring rapid diagnostic and therapeutic measures. Perirenal haematoma is typically described as being confined by the dense, collagenous Gerota’s fascia, however, some anatomists hold that the inverted cone of fascia forming the perirenal space is open both superiorly and inferiorly, with continuity of the anterior renal fascia across the midline. This allows extension of the haematoma inferiorly into the pelvic retroperitoneal space (most commonly seen extension of PRH), superiorly into the bare area of liver (on the right) or subphrenic extraperitoneal space (on the left), and medially into the contralateral perirenal space. [1] There is therefore no limitation on the accumulation of haematoma, and large amounts of blood may collect before clinical signs are present.

The majority of PRHs described in the literature are secondary to moderate to major trauma or instrumentation, although cases of PRH secondary to mild continuous trauma have previously been described. [3] It is posited that this may be due to shearing forces acting on the perirenal blood vessels where they perforate the renal fascia to supply the loose connective tissue surrounding the renal pelvis. Spontaneous perirenal haemorrhage (also known as Wunderlich’s syndrome) is less common, although its rate in the community may be underestimated due to the relative lack of clinical symptoms and self-resolution of the haematoma in non-severe cases. A diagnosis of spontaneous PRH can only be made if there is no history of recent trauma, anticoagulant use, dialysis or renal transplant. The most common causes of spontaneous PRH are benign or malignant neoplasm (61.5% of 101 cases), with angiomylipoma predominating (29.1%) followed closely by renal cell carcinoma (26.1%). [3] Aetiology is unknown in roughly 5-10% of cases. [1, 4]

It is important to determine the severity and aetiology of PRH, in order to establish adequate treatment direction and avoid any additional complications of the underlying pathology (e.g. shattered kidney, renal cell carcinoma). The clinical presentation of PRH is highly variable. Although it is classically associated with Lenk’s triad (acute flank pain, tenderness on palpation and symptoms of internal bleeding), the most common presenting signs and symptoms are abdominal pain (67-83%), haematuria (19-40%) and shock (11-26.5%). [3, 5] A small flattening of the normal contour of the loin may also be present. [6] Because of the variable and often nonspecific nature of the clinical presentation, radiological imaging is invaluable in formulating the diagnosis of PRH.

While ultrasonography may be used in emergency situations, a meta-analysis performed by Zhang et al showed CT to have a much higher sensitivity of detection of PRH than ultrasound (1.00 vs 0.56) [3] due to the inability to distinguish between solid renal mass, abscess and clotted blood on ultrasound. [7, 8] Moreover, CT was able to identify the underlying pathology with moderate accuracy (sensitivity 0.57, specificity 0.82) while ultrasound was relatively insensitive (sensitivity 0.11, specificity 0.33). Computed tomography is therefore the modality of choice for suspected PRH. If CT is not available magnetic resonance imaging may be a good alternative, although clinical data on its
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efficacy is limited\(^3\). Renal angiography is thought to be unhelpful unless pathological vasculature or active bleeding is suspected.\(^9\)

This article will address the CT appearance of five of the most common presentations of perirenal haematoma: trauma, post renal biopsy, post partial nephrectomy, angiomyolipoma and renal cell carcinoma.

APPEARANCE OF PERIRENAL HAEMATOMA ON COMPUTED TOMOGRAPHY

The kidneys are especially suited to evaluation by CT as they are surrounded by retroperitoneal and perinephric fat whose absorption coefficient or \(\mu\) (-5 to -15 Hounsfield units or HU) is considerably lower than that of normal renal parenchyma (+25 to +30 HU). This difference is further enhanced with intravenous infusion of contrast material.\(^10\)

Acute PRH will present on CT as a perirenal collection with a greater attenuation value (+40 to +90 HU) than the kidney parenchyma, becoming more radiolucent over the following days. [Fig. 1.1 & 1.2] Size and extent of haemorrhage should be clearly demonstrable, with minimal contrast enhancement. Blood will be seen dissecting between the lamellar septae and fat within the perirenal space.\(^7, 11, 12\)

Figure 1

Fig. 1.1. Acute presentation of right PRH thought to be secondary to renal tumour. Area of high-attenuation haemorrhage surrounding right kidney seen on CT. Scan performed without contrast due to poor renal function.

TRAUMA

PRH is present in approximately 8-10% of trauma cases.\(^13\) Renal trauma is broadly divided by the American Association for the Surgery of Trauma (AAST) into five grades, ranked by severity of injury (Table I).\(^14\) Grades I and II are classed as minor traumas (with Grade I traumas comprising 80% of renal injuries)\(^15\) and Grades III-V as major trauma. PRH may be present from Grade II onward. While the grading system does not differentiate between causes of renal injury, PRH is most commonly associated with penetrating trauma (67%).\(^13, 16\)

Figure 3

Table I (Lee, 2006)

<table>
<thead>
<tr>
<th>Grade</th>
<th>Appearances</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Haematoma with normal imaging studies</td>
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<tr>
<td></td>
<td>Contusion</td>
</tr>
<tr>
<td></td>
<td>Non-expanding subcapsular haematoma</td>
</tr>
<tr>
<td>II</td>
<td>Non-expanding perirenal haematoma confined to retroperitoneum</td>
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<tr>
<td></td>
<td>Superficial cortical laceration ≤1cm in depth without collecting system injury</td>
</tr>
<tr>
<td>III</td>
<td>Renal laceration ≤1cm in depth without collecting system injury</td>
</tr>
<tr>
<td>IV</td>
<td>Renal laceration extending through the kidney into the collecting system</td>
</tr>
<tr>
<td></td>
<td>Splashes containing the main renal artery or vein all contained haemorrhage</td>
</tr>
<tr>
<td>V</td>
<td>Shattered kidney</td>
</tr>
<tr>
<td></td>
<td>Avulsion of ureteropelvic junction</td>
</tr>
<tr>
<td></td>
<td>Complete laceration/avulsion with/without haematoma</td>
</tr>
</tbody>
</table>

The main aim of imaging in renal trauma is to determine if there is a requirement for urgent treatment. Perirenal haematoma may rarely be seen in isolation but is more commonly seen in association with a laceration of renal parenchyma. [Fig. 2.1 & 2.2]

Renal laceration is typically seen as a focal area of linear or irregular low attenuation on contrast-enhanced CT, extending to the surface of the kidney.\(^17\) They are usually of
higher attenuation than water and do not enhance.[15]

Management of renal laceration is usually conservative unless the patient becomes haemodynamically unstable, in which case exploratory surgery may be indicated. If there is no associated major vascular injury, the majority will heal without any specific treatment.

**Figure 4**

Fig. 2.1 Trauma-induced right PRH with associated Grade III renal laceration. Patient presented with haematuria and pain following football injury to right flank. Visible defect in renal parenchyma (arrow). Nil injury to collecting system seen on CT.

**Figure 5**

Fig. 2.2 Axial view of patient in Fig 2.1. Extravasation of contrast seen within the area of haemorrhage (arrow).

**POST RENAL BIOPSY**

Perirenal haematoma is a common complication of percutaneous renal biopsy. While clinically apparent PRH occurs in less than 6% of renal biopsies,[18, 19] the rate is significantly higher on post-biopsy CT, with some authors claiming a PRH rate as high as 90.9%. A link between a drop of >1g/L in haemoglobin at six hours post biopsy and incidence of PRH has been hypothesized, but the literature is inconclusive.[18] Transient microscopic haematuria is present in almost all patients, and is associated with CT evidence of PRH in 60-80% of cases.[22]

While CT has a greater rate of detection of PRH post-biopsy than other imaging modalities,[20] radiographic evaluation immediately post-biopsy has not been shown to be predictive of clinically significant PRH (detecting less than 15%).[18] Ultrasonography has been shown to be more useful in determining patients at low risk of complication, with one study showing that ultrasonography of a haematoma at one hour post-procedure had a positive predictive value of only 43%, but a negative predictive value of 95% for development of a complication, thus making it highly predictive of an uncomplicated post-biopsy course.[22]

CT is of more value in situations where post-biopsy PRH is already clinically apparent, as it provides a more rapid and non-invasive evaluation of the location and extent of bleed than the conventional combination of urographic, ultrasonic and angiographic investigations.[10] However, as noted above, clinically apparent PRH comprises a relatively small percentage of all renal biopsies.

It must be noted that while most clinically significant bleeding usually occurs within 12 to 24 hours post biopsy, PRH has been demonstrated up to 65 days after the biopsy.[19, 21, 22] The literature suggests bed rest and observation in hospital for 12 to 24hrs post-biopsy may reduce the significance of clinically significant PRH.[18]

**POST NEPHRECTOMY**

As with renal biopsy, PRH is a relatively common post-procedural complication of nephrectomy but is rarely clinically relevant. The most common reasons for total nephrectomy to be performed in current medical practice are live renal donation or resection of renal tumour. Partial nephrectomy has been established as a viable alternative for renal masses less than 4cm in diameter.[23-25]

The rate of clinically significant PRH in laparoscopic total donor nephrectomy is approximately 6%, with one in three of these (2% of total cases) requiring further surgical intervention.[26-28] There is limited data on open donor
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nephrectomy, possibly due to the trend towards laparoscopic methods, although one study suggests a PRH rate of 1.67% (1/60). [26]

There is greater variability in data on PRH post nephrectomy for renal tumour, possibly because of the variability of surgical presentation and dependence on operator skill. However, there appears to be an increased rate of significant PRH in partial nephrectomy (PN) [Fig. 3], with a meta-analysis by Lesage et al showing a rate of 1.1% and 3.4% post radical nephrectomy (RN) and PN respectively. [29] Similar rates have been observed in several smaller studies. [30-33] This may be attributable to the increased technical difficulty of PN.

There is usually a delay of median 12 days (range 6-36) in symptom presentation of post-nephrectomy PRH, which is postulated to be due to intraoperative arterial spasm and complete hilar clamping. [32, 34] The surgical approach does not appear to impact the rate of postoperative complication. [31, 35]

**Figure 6**

Fig. 3. Left PRH post partial nephrectomy. Wedge shaped defect to lower pole of left kidney visible on contrast-enhanced CT. Some associated extravasation of contrast is noted (arrow). Collection extends along left paracolic gutter to the pelvis.

The most common cause of significant PRH post partial nephrectomy appears to be renal artery pseudoaneurysm (RAP) [Fig. 4.1], which has been demonstrated in up to 3.75% of all PN. It is more common in laparoscopic than open PN (5% vs 1.38%), [32] and usually occurs in patients with central rather than peripheral renal tumours. This may be due to an increased rate of vessels transected partially or end-on during resection, particularly at the wedge resection apex. There is no available data on the rate of RAP in total nephrectomy.

**Figure 7**

Fig. 4.1. Renal pseudoaneurysm demonstrated on lower pole of left kidney in patient from Fig. 3. on angiogram.

Because of the often nonspecific and delayed nature of presenting symptoms of RAP, its diagnosis requires a high index of suspicion. Contrast-enhanced CT has been recommended in the literature as the most accurate form of assessment of this vascular pathology. [32, 34, 36] RAP classically appears on CT as a high-attenuation, well-circumscribed focal collection of contrast isodense to the adjacent arterial vessels, with surrounding PRH. [36, 37] It most commonly appears within the renal hilum due to the increased rate of vessel transection described above. A slightly delayed nephrogram may be seen compared to the contralateral kidney. [36]

On standard CT imaging, RAP may be difficult to distinguish from urinoma, another well-documented
complication of partial nephrectomy. 3-D reconstructions using volume rendering techniques assist greatly in differentiating the two by creating a vascular map of the kidney which clearly depicts the association of the contrast collection to the branches of the renal arteries. The gold standard treatment of post-nephrectomy PRH where conservative management has failed is superselective embolization. A large retrospective study of 3685 patients by Richstone et al demonstrated an efficacy rate of 95% with nil observed postoperative complications.

**Figure 8**
Fig. 4.2 Resolution of renal pseudoaneurysm seen in Fig. 4.1 post selective embolization.

**ANGIOMYOLIPOMA**
Angiomyolipomas (AMLs) are benign hamartomas composed of smooth muscle, fat and tortuous, thick-walled vasculature, commonly confused for neoplasm on imaging. They are usually solitary, unilateral and predominantly found in middle-aged women. Pathologically, they are nonencapsulated, slow growing and enlarge in an expansile fashion, distorting rather than destroying renal parenchyma.

The high vascularity of AMLs predisposes them to spontaneous haemorrhage, with rate of PRH directly linked to tumour size (15% of all AMLs vs 51-66% if >4cm in diameter). They are the most common cause of spontaneous perirenal haematoma. It is generally recommended that PRH in the context of AML be treated conservatively to preserve the kidney. Because of the high rate of bleeding, prophylactic embolization or partial/total nephrectomy is recommended if AML is >4cm in diameter.

CT is the most reliable method of diagnosis of AML, usually demonstrating a large mass containing fatty elements (-20 to -150 HU) with areas of increased tissue density (up to 150 HU). Identification of fat within the tumour allows a confident diagnosis of AML, especially if there is little to no calcification of the lesion.

**Figure 9**
Fig. 5.1 Right PRH associated with large AML. Contrast-enhanced CT shows radiolucent fatty elements within the tumour. There is gross distortion of right kidney parenchyma.

**Figure 10**
Fig. 5.2 CT angiogram of patient in Fig. 5.1, demonstrating the tortuous vasculature of large right renal AML.

However, PRH or perirenal extension of the AML may mask the underlying fat on imaging. In cases where the fat is not visible on CT due to a relative paucity of fatty elements or obscuration by PRH, identification of the lesion...
may not be reliably confirmed by any other form of medical imaging and exploratory surgery may be indicated.\(^7,11,12\)

**Figure 11**
Fig. 6. Right PRH associated with small AML. Minimal fatty elements are seen on contrast-enhanced CT (arrow), possibly due to obscuration by the overlying area of haemorrhage.

**RENAL CELL CARCINOMA (ADENOCARCINOMA)**

While renal cell carcinoma (RCC) has a relatively low rate of spontaneous PRH, its relative frequency in the general population makes it the most common malignancy associated with PRH and the second most common cause of spontaneous PRH.

The mechanism of spontaneous PRH in RCC is unclear. Hypotheses include tumour growth leading to necrosis and subsequent rupture/haemorrhage, or increased congestion and venous pressure within the tumour as it invades and occludes branches of the renal vein leading to bleeding and rupture.\(^{41}\) Tumour size has no correlation to incidence of haematoma.\(^3\)

Renal cell carcinoma characteristically appears on CT as an irregularly shaped renal mass with a lobulated or ill-defined outer margin. Necrosis commonly causes an area of low density in the central part of the tumour and central renal sinus fat is obliterated in approximately half of all cases. The carcinoma may be isodense to renal parenchyma on non-contrast scans, with poor demarcation between the tumour mass and renal parenchyma. [Fig. 7]

However, administration of IV contrast increases density of the renal parenchyma while the tumour remains relatively unenhanced due to its lack of functional tubular elements, allowing clear demonstration of the tumour/renal parenchyma interface. Contrast-enhanced CT is therefore the imaging modality of choice for detection and staging of RCC, with an accuracy of greater than 90%.\(^{39}\)

**ROLE OF COMPUTED TOMOGRAPHY IN THE MANAGEMENT OF PERIRENAL HAEMATOMA**

Initial management of PRH is often conservative. However, if the patient becomes unstable on conservative management emergency nephrectomy, haematoma evacuation or embolization may be necessary.\(^4,42\) Once the underlying cause of the PRH is identified, treatment may be altered as required.

The 2001 meta-analysis by Zhang et al showed total nephrectomy to be the most common treatment for spontaneous PRH (68% of cases, with partial nephrectomy in a further 4%). Conservative treatment was reported in only 9% of patients.\(^19\) While many urologists believe that unexplained spontaneous PRH with a normal contralateral kidney is highly suspicious of malignancy and thus requires total nephrectomy, the relevant literature is inconclusive. Although some limited case series in the literature have demonstrated renal cell carcinoma undetected on CT at the time of unilateral PRH,\(^{43,44}\) follow up histology in the Zhang study showed malignancy in only 43% of total nephrectomies. CT may therefore be useful in reducing the rate of unnecessary intervention where the underlying lesion may be conclusively identified as benign.
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It must be noted, however, that the sensitivity of CT in detecting underlying pathology at the time of haemorrhage is only 0.57 (as previously mentioned), possibly due to the obscuration of intratumoural fat by haematoma. Where no primary tumour is seen, some authors recommend radical nephrectomy on the grounds that small renal cell carcinoma may not be apparent on CT, while others advocate it in non-fatty renal masses only (i.e. where there is a high likelihood of malignancy) with further CT following PRH resolution, on the grounds that repeat imaging following resolution or evacuation of the haematoma may be more useful in definitively excluding malignancy. Others still recommend a wholly conservative approach with follow up CT post resolution of PRH.

CONCLUSION

CT imaging has been demonstrated to be helpful in determining the presence and underlying pathology of perirenal haematoma in a number of common clinical scenarios. By identifying the aetiology and severity of haemorrhage, it may reduce the rate of unnecessary interventions.

References


Author Information

Su-Ling Lai, MBBS BMedSci
Department of Radiology, Box Hill Hospital

Manfred Spanger, MBBCh FCRad FRANZCR
Department of Radiology, Box Hill Hospital