

PARTICULAR EVOLUTION OF GIANT COMPRESSIVE PERIRENAL HAEMATOMA - CASE REPORT

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Abstract

The development of a perirenal hematoma is rare and primarily the result of trauma, malignancy, or a connective tissue disease. Spontaneous perirenal haematomas are relatively rare and its diagnosis requires the absence of recent instrumentation, surgery or trauma. We report a particular case involving the development of a giant compressive perirenal haematoma in the absence of a major trauma history.

Keywords: compressive perirenal hematoma, nephrectomy

Introduction

Spontaneous retroperitoneal hemorrhage with secondary haematoma is an uncommon condition that occurs because of bleed either from the kidney or less often from adjacent retroperitoneal structures. The most common renal causes being angiomyolipoma, and renal cell carcinoma. Vascular diseases such as polyarteritis nodosa, renal artery aneurysm, infections of kidney such as cortical abscess, pyelonephritis and renal cysts are occasional etiologic factors. Adrenal haemorrhage is seen with severe stress conditions as sepsis, burns or trauma. Patients with pheochromocytoma, adrenal carcinomas, cortical adenomas also contribute as causes of retroperitoneal haemorrhage.

Clinically, these patients have variable presentation depending on degree and duration of bleed. Nausea, vomiting, low grade fever and a decreasing haemoglobin are common findings. Mild flank and upper abdominal discomfort in case of minimal bleed to patients presenting in shock with oliguria in case of massive blood loss. Urine examination is frequently normal.^[1,2] Ultrasound is a rapid non-invasive test to localize the haematoma extent and probably look at primary pathology. Subcapsular haematomas do not reach as large a size as when bleeding is into the perinephric space,

because of the tamponade effect of the renal capsule. This tamponade effect is not possible with distensible Gerota's fascia and hence the perinephric haematoma may attain very large size.^[3] CT scan in case of subcapsular haemorrhage will demonstrate the haematoma confined by renal capsule with parenchymal flattening. With haemorrhage in to the perinephric space, the CT scan will reveal an abnormal soft tissues density with displacement, compression or obscurity of normal retroperitoneal structures. As the haematoma ages, the density may decrease, while contrast enhancement of a subcapsular or perinephric haematoma does not occur unless active bleeding is taking place.^[4] CT scan remains the gold standard not only to diagnose the cause of bleed but also to exactly localize the extent of haematoma.

Flattened renal parenchyma compressed by haematoma is clearly seen in extracapsular haematomas. Capsular arteries remain close to the capsule in subcapsular haemorrhage. In perinephric haematoma, capsular arteries are displaced away from the capsule.^{[5],[6],[7]}

A variety of pediatric perirenal/renal masses may be differentiated in the first time from kidney tumors on the basis of their clinical and imaging features.

Wilms tumor is distinguished by vascular invasion and displacement of structures and is bilateral in approximately 10% of cases. Nephroblastomatosis occurs most often in neonates and is characterized by multiple bilateral subcapsular masses, often associated with Wilms tumors. Angiomyolipoma frequently contains fat and is associated with tuberous sclerosis. Renal cell carcinoma is unusual in children except in association with von Hippel-Lindau syndrome and typically occurs in the second decade. Multilocular cystic renal tumor is suggested by a large mass with multiple cysts and little solid tissue. Renal medullary carcinoma occurs in patients with sickle cell trait or hemoglobin SC disease and manifests as an infiltrative

mass with metastases. Metanephric adenoma lacks specific features but is always well defined. Renal lymphoma is characterized by multiple homogeneous masses, often with associated adenopathy.

Case report

An 8 year old boy presented at the emergency department complaining of significant left-sided flank pain. Patient's history revealed a small lumbar trauma suffered 2 weeks ago. Vital signs showed a pulse rate of 88 beats per minute, blood pressure of 110/80 mm Hg, and temperature of 36.4 °C. Clinical examination

revealed abdominal pain in the left upper and medium quadrant.

Laboratory analysis of the patient's blood was normal, except for leukocytosis (count of 19,890/ml). Urine analysis revealed presence of albumin, numerous RBC/high-powered field and 1-3 WBC/ high-powered field, high levels of vanil-mandelic acid.

Renal ultrasonography: the ecographic aspect suggested a mixed left renal tumor with secondary hydronephrosis (fig. 1,2).

The right kidney had normal echographic aspect.

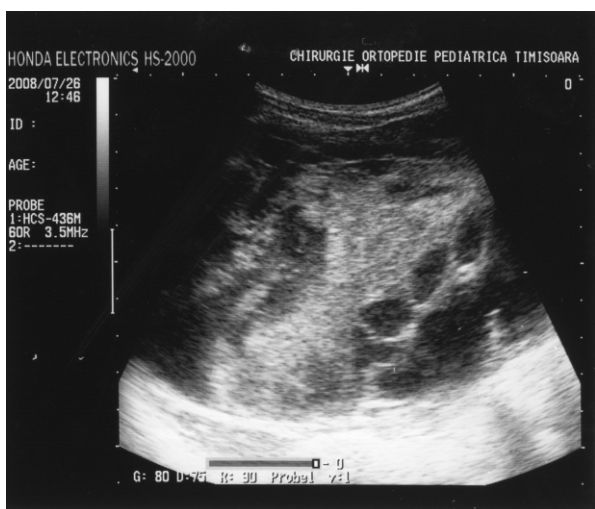


Fig. 1. Echographic aspect of left kidney.

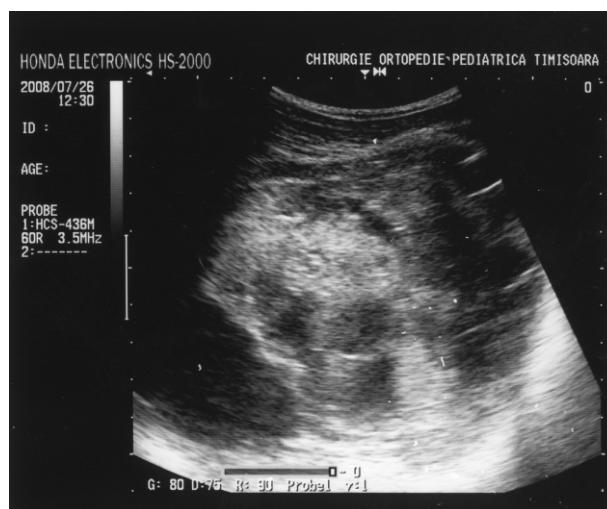


Fig. 2. Echographic aspect of left kidney.

Intravenous pyelography in the early excretory phase demonstrated normal excretion of contrast substance from the right kidney and delayed excretion of contrast material from the left kidney. Intravenous pyelography in the later excretory phase: contrast substance begins to accumulate in the dilated calyces

of the left kidney, no contrast substance was seen in the left ureter on this or other images, suggesting hydronephrosis caused by uretero-pelvic junction obstruction. The left kidney demonstrated relatively increased renal length in comparison with the right kidney.



Fig. 3. Intravenous pyelography - early excretory phase.



Fig. 4. Intravenous pyelography - latent excretory phase.

All these investigations suggested a mixed left renal tumor with secondary hydronephrosis, stage IV.

Abdominal MRI revealed left giant perirenal hematoma with secondary hydronephrosis stage IV, reduced renal parenchyma of 3 mm. (fig.5,6)

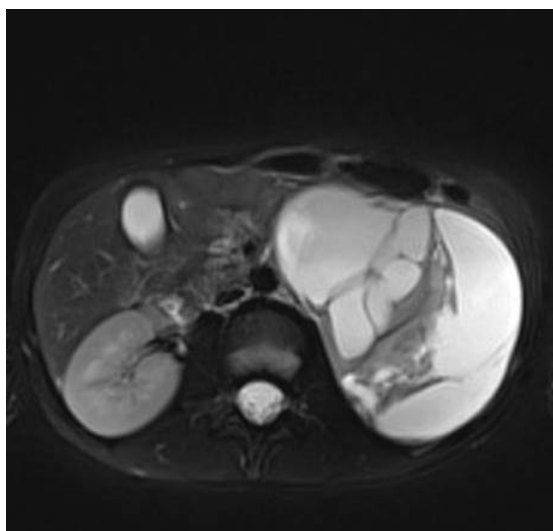


Fig. 5. Abdominal MRI.

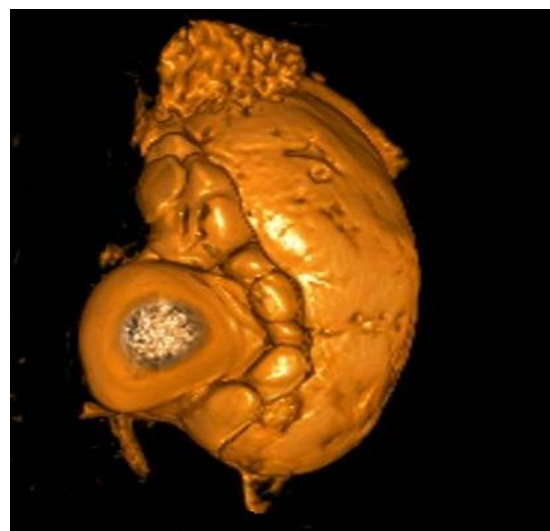


Fig. 6. Abdominal MRI (reconstruction).

Treatment in this case consisted of great median laparotomy with peritoneal organs exploration. The intraperitoneal organs showed no macroscopic pathologic modifications.

By drifting the left colon, an entry into the retroperitoneal space was made. The presence of a giant tumor (with clear margins, encapsulated and mixed structures) of the left kidney was evidenced, having on its surface well defined vasculature. Based on the results of the laboratory investigation correlated with the intra-operative macroscopic aspect, we performed a radical left uretero-nephrectomy within the oncological safety margins (left adrenalectomy, excision of peri-renal fatty mass and renal hilar lymph nodes). The macroscopic aspect of the excised tumor revealed some areas of haematoma alongside areas of “blood with no clots”

Post-surgery care consisted in administration of antibiotics (cephtriaxone), solution of parenteral nutrition (glucose, aminoacids), electrolytes, antalgics and non-steroids anti-inflammatory drugs. The evolution was favorable with swift digestive tolerance and regaining of the intestinal transit.

Histopathological findings: The post operative histopathologic results confirmed the notion of the MRI findings: perirenal haematoma.

Discussion

The development of a perirenal haematoma is rare and primarily the result of trauma, malignancy, or a connective tissue disease.

In our case the mechanism of production of compressive perirenal haematoma remain unidentified. The absence of major trauma, clinical findings and the echographic aspects of left kidney has raised the suspicion of a Wilm’s tumor. MRI showed left massive perirenal haematoma, left pelvicalyceal dilatation and a thinned renal cortex suggestive for a compressive perirenal haematoma with severe hydronephrosis. The ureter was collapsed. The hydronephrotic modifications could be produced by the progressive perirenal haematoma with compression of the upper portion of ureter. This massive perirenal haematoma was not communicating with the subcapsular parenchyma or the collecting system. The contralateral kidney was normal. In these circumstances we could not determine if perirenal haematoma was a result of a minor injury to a hydronephrotic kidney or if it was a result of a minor injury to an already existing arterio-venous renal malformation and the hydronephrosis was secondary to it.

Intraoperative we found giant perirenal haematoma which compressed the left kidney and massive destruction of kidney tissue which imposed nephrectomy. Because this patient did not show signs of acute hemorrhage, the haematoma probably appeared gradually with progressive compression and disturbances of renal blood and secondary injury of nephrosclerosis.

Histopathological examination postnephrectomy not detected tumoral changes, congenital or vascular malformations.

Conclusion

1. The perirenal hematomas are usually a result of a severe injury and have an obvious clinical manifestation.

2. In the described case, the injury was insignificant and without any clinical signs.
3. Preoperative differential diagnosis was suggestive for perirenal haematoma or mixed renal tumor, with the destruction of the renal parenchyma (limited to 3 mm).
4. The intraoperative aspect was suggestive for a mixed renal tumor, reason for which a left nephrectomy (within oncological safety margins) was performed.
5. The positive diagnosis of left giant, compressive perirenal haematoma with secondary hydronephrosis and nephrosclerosis was established examining the macro and microscopic aspects of the excised mass.

References

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