Case Report

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Spontaneous Rupture of Kidney Due to Posterior Urethral Valve– Diagnostic Difficulties

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Abstract

Background: Spontaneous kidney rupture could develop in the course of posterior urethral valve (PUV), the most common cause of outflow urinary tract obstruction in male infants. However, urinary extravasation is a rare complication among this group of children.

Case Presentation: Our case report presents diagnostic difficulties connected with spontaneous kidney rupture due to PUV in a 6 week-old infant. Due to not equivocal images, thundery course of disease and rapid deterioration in the infant`s condition, the patient required an urgent laparatomy.

Conclusion: This case showed that the investigation of renal abnormalities during early neonatal period, is very important specifically in PUV that can lead to kidney rupture.

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Key Words: Urinoma; Kidney Rupture; Urinary Extravasation; Posterior Urethral Valve

Introduction

Kidney rupture in developmental age most often takes place in the aftermath of multiorgan trauma, especially when urinary abnormalities or urolithiasis are present. Rarely spontaneous rupture of kidney is described due to hydronephrosis caused by ureteropelvic and vesicoureteric junction obstruction[1]. Also polycystic kidney disease, neoplasmatic lesions due to renal cell carcinoma, renal angiomyolipoma and transitional cell tumor could be predisposing factors. Additionally, spontaneous kidney rupture could develop in the course of posterior urethral valve (PUV). However, urinary extravasation is a rare complication among this group of children. The clinical manifestation is often nonspecific.

Our report presents diagnostic difficulties connected with spontaneous kidney rupture due to PUV in a 6-week old male.

Case Presentation

Previously healthy 6-week-old boy, born to a GIII PIII, birth weight 2900g, was admitted to the district hospital with fever, vomiting and oliguria persisting for several hours. Family history was of

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no significance. On admission, infant's general condition was poor. A distended abdomen, tense and tender on palpation, was noted. Urethral catheterization was difficult. Laboratory investigations revealed: WBC 3.7 K/u, Hb 8.4 g/dl, urea 70 mg/dl, CRP 132.3 mg/l, procalcitonine >200 ng/ml, and uncompensated metabolic acidosis. Urine microscopy revealed pyuria and few fresh red cells. Ultrasound examination showed a left kidney measuring 70 mm in length with a dilated pelvi-calyceal system and an enlarged hydronephrotic right kidney measuring 90 mm in length. Treatment was started with cefotaxime, vancomycin, dopamine and furosemide. The infant`s general condition improved, but CRP continued to rise (281.5 mg/l). Serum urea, creatinine and procalcitonine were 98.0 mg/dl, 1.0 mg/dl and 180.74 ng/ml, respectively. Blood and urine cultures were negative. A diagnosis of bilateral hydronephrosis was made.

On the 2nd day of hospitalization the infant was transferred to our Department. A distended abdomen with a right sided large palpable mass was found. Laboratory tests showed CRP 309.4 mg/l, urea 116 mg/dl, creatinine 1.81 mg/dl, Hb 10.5 g/dl and compensated metabolic acidosis. Urine output in less than 24-hours measured about 280 ml. Cefotaxime and vancomycin were discontinued and meropenem commenced. Abdominal ultrasound demonstrated left kidney 7×2.8 cm, with mild pelvic dilatation, and right kidney 11×6.6 cm with a dilated and deformed collecting system. The upper and middle calyces seemed mostly dilated with a total volume of about 216 ml, pelvic volume 3 ml. Right kidney parenchyma was compressed. The bladder was empty. Hydronephrosis was considered, but a suspicion of kidney compression by an extraneous cystic structure was also raised. Due to worsening of the infant's condition and equivocal ultrasound images, he was qualified for an urgent laparotomy.

During surgery a huge cyst was found in the peritoneal cavity, encircling almost the whole right kidney and connected to the renal capsule by its capsule. The cyst contained about 500 ml of watery bright fluid which was resected revealing that its wall was a fragment of the anterior wall of the renal capsule. Drains were set up to postoperative sites. Additionally, the bladder was noted to have a thick and hard wall.

In the following 24-hours renal function improved (urea 47 mg/dl, creatinine 0.95 mg/dl), CRP decreased to 65 mg/dl. Urine output was 2-5 ml/h. Bright yellow draining fluid, about 20 ml/h, was noted to have similar biochemical properties to urine: urea 496 mg/dl, urea nitrogen 231.8 mg/dl, creatinine 9 mg/dl. Follow up abdominal ultrasound reported a slightly wider right pelvi-calyceal system, with a small fluid collection near the upper pole (Fig. 1). Voiding cystourethrography showed trabeculated bladder wall. Cystoscopy revealed urethral structures which could correspond to disrupted posterior urethral valves. Valve ablation was performed. Histopathologically the wall of the resected cyst turned out to be the right kidney capsule and was reported as *Pseudocystis in inflammatione chronica*. Retrospectively spontaneous kidney rupture due to posterior urethral valves was diagnosed.

After 21 days of treatment, inflammatory markers and renal function normalized. Renoscintigraphy performed 6 weeks post surgery revealed cumulative renogram curves of both kidneys, the right kidney (operated) contributing 81.5% of renal function and the left kidney 18.5%. At 20 months follow up review both renal function parameters and blood pressure values were normal. Ultrasound examinations revealed persisting mild dilatation of both pelvi-calyceal systems. History regarding urinary tract infection was negative.

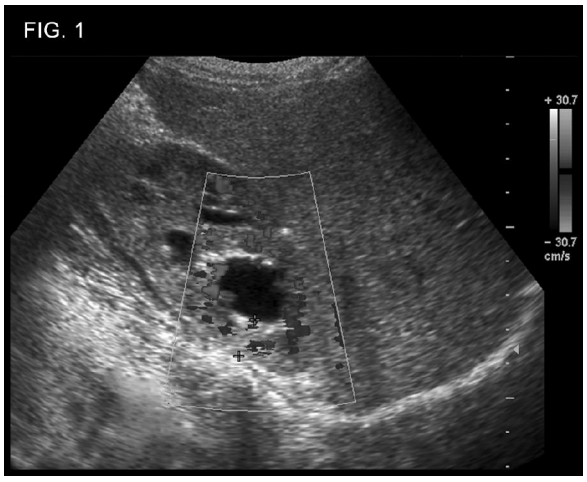

Fig. 1: A ultrasound image of fluid encircled right kidney's parenchyma

Discussion

Diagnostic difficulties in our child were connected with an ambiguous clinical image. The case of our 6 week-old infant does not blend with the symptomatology-diagnostic scheme typical for PUV. Usually suspicion of PUV is propounded prenatally. Postnatally this abnormality reveals as a general rule in the neonatal period, most often as a pelvis dilation and urinary tract infection[2]. In our case the boy was born to a healthy mother at term, in good condition and was discharged from the neonatal unit without suspicion of any anomaly. The antenatal ultrasound examinations were normal. On admission to hospital patient`s clinical condition suggested acute kidney injury caused by urosepsis due to bilateral hydronephrosis. Ultrasound performed after few hours revealed an acutely increasing fluid collection in the right side of the abdominal cavity. The image was ambiguous and required differential diagnosis from multilocular cystic nephroma, cystic nephroblastoma, cyst with septum and adrenal hemorrhage. In that situation the infant was qualified for an urgent laparotomy. Absent visible marks of ruptured kidney's parenchyma did not allow to recognise intraoperatively a urinoma (i.e. the development of a perirenal extravasation of urine). Later retrospective analysis of full clinical picture made us diagnose spontaneous kidney rupture in the course of PUV and subcapsular urine collection.

Urinoma is associated with PUV in 1% to 9% of cases. In 2008 Finnish authors published results of retrospective analysis of 17 boys with PUV and urinary extravasation. Nine children had a perirenal urinoma, six urinary ascites and two patients urinothorax. In all cases the diagnosis of PUV was made before age 9 weeks[3]. In turn Dutch authors described 10 cases of neonates with rupture of the uropoetic system. Underlying diagnosis included obstruction of the ureteropelvic junction in 3 children and PUV in 7 children[4].

It is not known if urinoma is correlated with severity of obstruction. In our patient difficulty with urethral catheterization, thickened bladder wall described in diagnostic imaging and confirmed intraoperatively, argued for a pronounced subvesical barrier.

Therapeutic management of spontaneous kidney rupture due to PUV has not been established yet. Usually percutaneous drainage of the urinoma and urethral catheterization are sufficient[5]. We opted for laparotomy in our patient because of equivocal images, thundery course of disease and rapid deterioration of the infant`s condition.

It is still under discussion whether urinoma could be a protective mechanism in obstructive uropathy. Claahsen-van der Grinten`s et al analysis revealed a better function of ruptured kidney in renal scintigraphy compared to the other side[4]. Our presented case seems to confirm these observations. The right kidney function (with urinoma) was estimated as 81.5% and the left kidney 18.5% in renoscintigraphy. In turn Patil et al study did not confirm protective role of urinoma for the ruptured kidney function. On the contrary they observed deterioration of its function[5]. Once again, retrospective study by Finnish authors reported that urinoma had neither harmful nor protective influence on kidney function[3].

Conclusion

The above described case is an example how important is investigation of renal abnormalities during early neonatal period, realizing the fact that in some cases PUV can lead to kidney rupture.

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