

CONSIDERATIONS ON A CASE WITH ACUTE RENAL FAILURE OF UROLOGIC NATURE IN INFANCY

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Abstract

The paper presents the case of a 6-month old infant hospitalized in Clinic II Pediatrics for congenital bilateral ureteral stenosis with subclinical evolution before this age. Clinical course revealed the occurrence of the urinary tract infection associated with the malformation, with unfavorable and rapid evolution to acute pyelonephritis, and acute renal failure with demise in the 5th day of hospitalization. The case was referred to our clinic from a Pediatric Ward where he was admitted for a respiratory infection and the diagnosis of polycystic kidney was suspected based on an ultrasound examination.

Keywords: congenital bilateral ureteral stenosis, acute pyelonephritis, acute renal failure.

Introduction

Congenital bilateral ureteral stenosis is a rare abnormality of the urinary tract, more frequent in boys, ureteral stenosis being situated wherever between the ureteropelvic and the ureterobladder junctions¹.

Clinical signs represent an outcome of the complications occurred because of the urinary obstruction, and are recurrent urinary infections, ureterohidronephrosis (pseudo tumor kidney), chronic renal failure and more rare acute renal failure.¹

Excretory urogram examination represents the most important method of examination for a certain diagnosis. It shows the uretero-pyelo-calyceal suprastenotic dilatation with a delay or even absence of elimination of the contrast substance.

Case presentation

The 6-month old infant, G.P., was admitted to our clinic for: fever and vomiting. Family history revealed young and apparently healthy parents. He was the third

child, with normal gestational age, birth weigh 3300g, 2 months breast feeding and then artificial feeding with cow milk. Two days before admittance in our clinic, the child was hospitalized in another pediatric department for Bronchopneumonia being treated with association of antibiotics. Based on an ultrasound examination, a suspicion of polycystic kidney was established. No information regarding diuresis before admittance was available.

Clinical examination showed an infant with 3850g, fever (T=39⁰C), bad general state, no appetite, sever pallor, warm extremities, enlarged abdomen, liver with inferior margin 2 cm below the right rib. At the examination of the medium right part of the abdomen a 4/6cm pseudo tumor was revealed, with lombar contact, firm, wavy surface, mobile, and another 4/4 cm pseudo tumor was palpable in the hypogastrium and pubian area, firm, immobile, with a smooth surface. Oligo-anuria was present during the entire evolution.

Biologic tests showed: Hb=6,6g%, Er=2,400 000/mmc, L=5,200/mmc, ESR=120/141mm, uree=178mg%, creatinine=1,4mg%, uric acid=9,7mg%, Natrium=146→135→119mEq/l, K=6,5mEq/l, ph=7,07, BE=-19,7mmol/l, pCO2=30,3, albuminury, leucocitury, Uroculture with E. Coli>100,000/ml, sterile hemculture.

Renal ultrasonography in different sections revealed sinusal transonic images into the renal parenchyma, wiped pyelocaliceal shadow, and disseminated hyperreflectogen images in the cortico-medullar area. Right kidney appeared with a mixed structure, with hyperefectogen images up to the cortico-sinusal level, with very slight differentiation parenchima-sinusal. Between the liver parenchyma and the renal parenchyma, the renal capsule is highly reflectogene, with wavy contour and invasion of psoas (Fig. 1).

Fig.1 Renal ultrasonography-
Left lumbar transversal section-
Transonic images that go into
the left renal parenchyma.



Bad general state of patient and the presence of acute renal failure did not allow us to perform the excretory urogram. In the absence of the excretory urogram, the diagnosis of that moment was: Congenital anomaly of kidney (probably

bilateral polycystic kidney); Acute pyelonephritis; Acute renal failure. The evolution was unfavorable despite the treatment, with profound alteration of general state and decease within 5 days from admittance.



Fig 2. Macroscopic examination- Both kidney increased in size, wavy surface, bilateral hidroureter. Morphopathologic exam actually allowed us to visualize the patient lesions.

Macroscopy revealed both kidney increased in volume and weight (right kidney 8,5/3,5/5cm, left kidney 6,5/3,5/3 cm) colored in red, highly waved surface, with an aspect of bilateral hydroureter (Fig 2). On the section, the calices appeared dilated, filled with purulent liquid. The left ureter had a narrowing of the lumen through a spin at 3,5cm from the ureteropyelic junction, whereas the right ureter was impermeable, with a severe stenosis 1,5cm from the ureteropyelic junction. Bilateral ureterohydronephrosis and

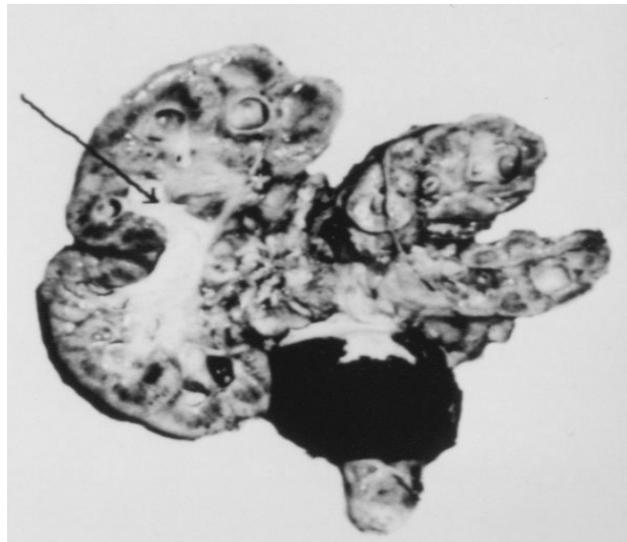


Fig. 3 Macroscopic exam - Dilated calice, filled with purulent liquid.

increased urinary bladder with large walls purulent liquid and necrotic-hemorrhagic mucosa were also noticed (Fig. 3).

Microscopy showed confluent interstitial inflammatory lesions with congestion, interstitial edema, polymorphonuclear infiltration, suppurative necrosis, endotubular necrotic and granulocytic cylindries. (Fig. 4, 5).

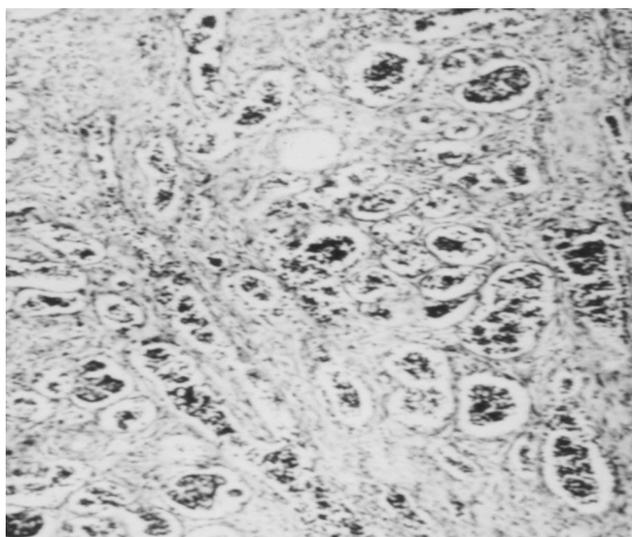


Fig. 4. Microscopic exam - Confluent interstitial inflammatory lesions, polimorphunuclear infiltration, suppurative necrosis.

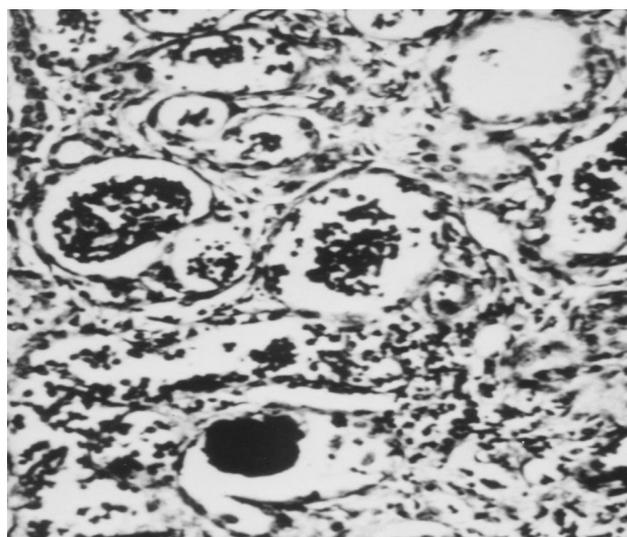


Fig. 5. Microscopic exam - Endotubular necrotic and granulocitar cylindries.

Discussion

The case was admitted to our clinic with digestive symptoms and fever, which after investigations proved to be the clinical and biological picture of the acute renal failure, following an acute pyelonephritis.

Ultrasound examination did not establish a precise diagnosis, but a suspicion of congenital renal anomaly was clear, most probable a cystic disease of kidney.

Among these, the most frequent disease in children is multicystic dysplastic kidney which goes with increased kidney, palpable at clinical examination, wavy surface, severe and rapid evolution to acute renal failure and decease. In this case, a giant hydronephrosis is developed with the decrease of renal parenchyma to a blade size.^{1,4,5}

A second frequent anomaly is represented by polycystic kidney (Potter I polycystic kidney). The kidneys are also palpable at clinical examination, increased in size,, wavy surface, but the onset occurs later on in life with signs of portal hypertension (hepatosplenomegaly) through the severe congenital hepatic fibrosis and with chronic renal failure progressively installed.

Polycystic kidney type Potter IV (partial or intermittent obstruction of the urinary flux) presents the most elements similar with our case: palpable kidney, wavy surface, bilateral hidroureter, hypertrophic urinary bladder, severe prognostic with evolution to acute renal failure. This

disease was excluded based on the macroscopic and microscopic examination.^{1,4,5}

Another aspect that should be taken into account is the eventuality of a preexistence of chronic renal failure stage II partly compensate on which acute renal failure occurred. Actually, renal malformations determine chronic renal failure at first, acute renal failure occurring as a complication of the chronic stage in the context of hydro-electrolytic perturbation witch was the situation of our case. The patient presented oliguria within 2 weeks ago, at the same moment with the onset of the respiratory disease, and the azotemia increased gradually, simultaneous with hydroelectrolytic and acido-basic perturbations.

Conclusions:

1. The case represents a regrettable diagnosis error
2. The delayed diagnosis was determined by multiple factors: few clinical signs at the domicile, ambiguous results at ultrasound examinations, and the rapid evolution of pyelonephritis in the absence of therapy to acute renal failure.
3. Excretory urogram with radiopaque medium performed in due time would have been allow a precocious diagnosis and a rapid surgical correction treatment

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