Scientific Research Publishing

Open Journal of Pediatrics, 2023, 13, 791-797
https://www.scirp.org/journal/ojped
ISSN Online: 2160-8776
ISSN Print: 2160-8741

Posterior Urethral Valves without Reflux Associated with Bilateral Renal Cortical Atrophy: Diagnosis and Management

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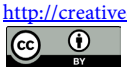
How to cite this paper: Kaba, B., Ouo-Ouo, K., Hassimiou, C.S., Aminata, B.M., Emmanuel, C., Lamine, D.M., Binta, D.F., Moustapha, D.M., Moustapha, K. and Pé, B. (2023) Posterior Urethral Valves without Reflux Associated with Bilateral Renal Cortical Atrophy: Diagnosis and Management. *Open Journal of Pediatrics*, **13**, 791-797.
https://doi.org/10.4236/ojped.2023.136087

Received: August 22, 2023
Accepted: November 4, 2023
Published: November 7, 2023



Abstract

The objective of this case report was to highlight the characteristics of posterior urethral valves observed in a 3-year-old patient and to describe their management. The case of a 4-year-old patient treated for a posterior urethral valve with bilateral renal cortical atrophy was reviewed. The diagnosis was confirmed by abdominal ultrasound, cystography and abdominal CT. He was referred to Martinique (a tertiary health establishment) for effective support. From the acute situation to the fortuitous discovery, its understanding must be deepened because of the potential immediate symptomatic impact in the form of renal colic which can be associated with sepsis, as well as in the long term on renal function. This observation is intended to help the attending physician to initiate his diagnosis and treatment.

Keywords

Valves, Urethra, Posterior

1. Introduction

The valves of the posterior urethra, a congenital obstacle to the posterior urethra, represent the most frequent cause of obstruction under the bladder in children. This anomaly, rare and generally isolated, accounts for up to 50% of the malformative causes of end-stage renal failure in children. Prenatal diagnosis is now possible more than 4 times out of 5. Before this possibility of antenatal diagnosis, the age of diagnosis was a good indicator of the severity of the obstruc-

tion. In 25% to 50% of cases the valves of the posterior urethra still lead to renal failure.

Posterior urethra valve (PUV) is a congenital anomaly that affects the whole genitourinary system with consequent renal impairment [1].

Posterior urethral valve mortality has fallen from 50% in the 1950s to about 5% today (only in neonates with renal failure and pulmonary hypoplasia) [2]. There is no known cause for this anomaly which affects 1/5000 to 1/8000 boys.

2. Case Report

He presented in my health facility on account of recurrent episodes of abdominal pain associated with vomiting and constipation of one month duration. Elsewhere, pollakiuria is observed.

On examination: Weight: 17 kg, saturation: 100%, HR: 106 beats/min, FR: 32 cycles/min, PA: 124/92/min, temperature: 37.9 degrees Celsius.

General condition preserved, stable hemodynamics, good fluid status, eupneia in ambient air, normal cardiorespiratory auscultation, no transit disorder.

Entry assessment: hemoglobin level: 10.9 g/dl; leukocytes: 22,500/mm^3; CRP: 76 mg/l; PCT: 4.5 ng/ml; creatinine 56 micromol/l sodium: 140 mmol/l; potassium: 2.8 mmol/l; ECBU: leukocytes: 15/mm^3; red blood cells: 11/mm^3; Sterile urine culture.

Abdomen without standing preparation: abdominal distension, presence of air in the right hypochondrium, diffuse coprostasis, doubt about the hydro-aerial level in the ascending colon.

Abdominal CT scan for suspicion of occlusion: bladder globe with a bladder with irregular diverticular thickened walls evoking a struggling bladder. Significant bilateral pyelocaliciel and ureteral dilation (pelvis measured at 24 mm on the left and 23 mm on the right) with more marked bilateral cortical atrophy on the left. Hypotonia with colic and hail fluid retention without visible obstacle. Peritoneal effusion of weak to average abundance more marked on the left.

Supported:

This is how we proceeded to the rectal enema, the establishment of an indwelling urinary catheter, the administration of analgesic, Diffu k, a probabilistic antibiotic therapy was established by Metronidazole: 180 mg × 3 per day, Cefotaxime 900 mg × 3/day stopped on D8 until CRP normalization, Gentamycin: 90 mg/day stopped on D2.

Monitoring progress:

On the endocrine level: looking for biological signs in favor of a peripheral neuropathy that could explain the struggling bladder. TSH achievement: 2.20; T4: 16.6; B6: 11 nmol/l (N: 17-209).

On the hematological level: VGM: 72 fl; MCHC: 30%; reticulocytes: 22,350/mm^3; iron: 2 micromol/l; Transferrin: 2.10 g/l; saturation factor: 4%; Ferritin: 107 micromol/l; Vit B9: corrected by hematocrit: 269 nmol/l; Vit B12: 115 nmol/l; hemoglobin electrophoresis: HbA1: 82%; HbF: 1.6%; HbA2: 2.4%. In view of these results, a regenerative hypochromic microcytic anemia was suspected and sup-

plementation with Foldin at 5 mg/day.

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On the digestive level: supple depressible abdomen without hepatosplenomegaly without defense or contracture. Elsewhere we note constipation which is well relieved by symptomatic treatment.

On the urinary level: installation of a permanent urinary catheter, diuresis estimated at 1200 ml/kg/hour, urinary ionogram: Na: 57 mmol/l; potassium: 8 mmol/l; chlorine: 16 mmol/l; Creatinine: 2 mmol/l; Protein: 0.42 g/l;; Albumin/creatinine: 210 mg/mmol; pH: 5; Density: 1.015; Albumin: negative; Glycosuria: negative; ketone: +; negative nitrite; blood ++++; urobilin: negative.

Control ECBU: leukocytes: 360/mm^3; red blood cells: >1000/mm^3, positive urine culture Enterococcus faecalis.

Cystography: known diverticular (fighting) bladder. Homogeneous bladder opacification, with discreetly significant repletion compared to the previous examination. On per-micturition images: good opening of the bladder neck. Stenosis of the membranous urethra with upstream ureteral dilation. No abnormality of the penile urethra. In conclusion: stenosis of the membranous urethra with upstream urethral dilation related to the valves of the posterior urethra. No notable vesicoureteral reflux.

Neurologically: normal examination, a cerebromedullary MRI was performed to rule out a neurological bladder. Spine: no spinal cord or cerebral abnormality, but note the presence of chronic left maxillary sinusitis.

In total, the diagnosis retained was: valves of the posterior urethra without reflux, proteinuria associated with bilateral renal cortical atrophy and anemia due to iron and B9 deficiency.

A transfer to Martinique was organized for support.

3. Discussion

Posterior urethral valves are one of the main obstructive uropathies in boys [3] [4] [5], with a variable incidence depending on the series, ranging from 1/5000 male births according to Perks [6] to 1/25,000 for Atwell [7]. The incidence of Posterior Urethra Valve (PUV) compared to other malformative uropathies is also variable. The diagnosis of PUV must of a dilation of the posterior urethra which can be objectified by morphological ultrasound from the 24th SA. This can also detect abnormalities of the urinary tract associated with renal dysplasia

(as seen in severe oligohydramnios) [8].

Prenatal diagnosis and therefore early management theoretically prevents infectious and hydro-electrolytic complications on the one hand and preserve the renal parenchyma on the other [9].

The major picture, grouping together oligohydramnios, renal insufficiency, respiratory distress, large kidneys and globe of the bladder, initially described by Potter, has become exceptional thanks to the contribution of morphological ultrasound.

Apart from prenatal diagnosis, the circumstances of discovery vary greatly. In the neonatal period, VUP can be suspected in the face of abnormalities in the micturition stream, late urination, oliguria, pyuria or even urinary ascites, but it is mainly extra-urinary signs, therefore misleading, which take center stage up to respiratory distress secondary to pulmonary hypoplasia. In infants and older children, the mode of revelation is dominated by urination disorders and especially urinary infection which can lead in the more or less long term to renal failure.

Retrograde cystography revealed: stenosis of the membranous urethra with urethral dilation upstream in relation to the valves of the posterior urethra. No notable vesicoureteral reflux. It can be practiced from birth without inconvenience. The valves have a variable appearance depending on their type: double convex image downwards (type I) or horizontal or oblique line (diaphragm: type III). The direct image of the valves corresponds to a downward and forward oblique linear sharpness or a cup-shaped abutment.

This image is under montane. There is a disparity in the urethral caliber with dilation of the upstream urethra and reduction in the caliber of the downstream urethra. Reflux into the prostatic utricle and sperm ducts is possible. The abnormal presence of a subcervical chamber in the form of a dilation of the posterior urethra upstream of the obstacle is a pathognomonic sign of PUV. Hypertrophy of the bladder neck, a bladder with a thick and irregular wall or, on the contrary, a large, atonic bladder with a gaping neck are also indirect signs of PUV [10].

The other examinations, whether radiological or isotopic, will make it possible to assess the morphological and functional impact on the upper appliance. Cystoscopy is the "key" examination for the diagnosis of PUV because it allows a direct study of the cervix, the bladder wall and the ureteral orifices, often in an ectopic position. It also and above all allows the classification of valves and their treatment by directed electrocoagulation [10].

Anatomical constraints can hamper the performance of cystoscopy: very tight foreskin, very thin meatus and narrow balanic urethra, not admitting even the smallest endoscope. It is then necessary to carry out gentle dilation of the distal urethra using fine beniqués or soft bougies 6, 8 or 10 Ch. A small meatotomy is sometimes necessary but carries a subsequent risk of scarring stenosis of the meatus. It is also possible to leave a No. 6 urinary catheter in place 24 to 48 hours before the endoscopic procedure.

The treatment does not only consist in cutting the valves and removing the obstacle, it must also take into account the associated lesions which may later require an appropriate action if there is a threat to renal function. The age of the patient at the time of sectioning of the PUV is a very important prognostic factor because the more the pathology has evolved, the greater the risk of bladder lesions [11] [12] [13].

The prognosis of PUV depends on several factors.

Prenatally, the presence of antenatal renal dysplasia and an anteroposterior diameter of the pyelon greater than 10 mm are considered factors of poor prognosis. Postnatally, certain factors can contribute to the deterioration of renal function, in particular vesicoureteral reflux and its corollaries, hyperpressure in the excretory cavities and urinary tract infection. The level of serum creatinine before the removal of the obstacle is a good reflection of the subsequent evolution: a serum creatinine higher than 90 micromol/L the 1st year constitutes an element of poor prognosis. In the advanced forms, which are unfortunately frequent, the bladder involvement, of a neurogenic nature, only partially regresses after the removal of the obstacle and poses enormous problems for management [14].

Overall, progression to renal failure is seen in significant proportions ranging from 16% of cases for Macher [15] to 45% of cases for Warshaw [16].

In most recent studies, there no longer seems to be a significant difference in prognosis between transplants for renal failure related to posterior urethral valves or other etiologies on the essential condition of properly managing the morphological abnormalities and especially functional problems of the bladder, the management of which is long, painful and full of hazards constituting a cause of failure in a good number of cases [17] [18] [19].

4. Conclusion

Relatively frequent PUVs are one of the most serious malformative uropathies in boys, which can lead to irreversible deterioration of the upper urinary tract. Prenatal ultrasound diagnosis significantly modified the presentation of the valves of the posterior urethra. The current therapeutic doctrine is: treat the valves, only the valves and monitor the evolution. The evolution is linked to the etiopathogenesis of PUV which makes it an affection not limited to the supramontanal urethra and the bladder but to the urinary tree in its entirety. Moreover, despite the treatment of the valves, neurogenic lesions often persist in the bladder which can evolve on their own account and impose variable therapeutic attitudes according to the type, mode of presentation and especially the date of appearance of the symptomatology.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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