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# *Urine Ascites Complicating Obstructive Uropathy: Report of a Case*

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**Summary**

**Adedokun HPA. Urine Ascites Complicating Obstructive Uropathy: Report of a Case.** *Nigerian Journal of Paediatrics* **1984; 11**:129. A bladder perforation producing urine ascites in a child with posterior urethral valves is reported. The diagnosis of urine ascites was made at micturating cysto-urethrography, but the patient died shortly after the procedure. It is emphasised that this procedure should be carried out early in all neonates, especially males, with neonatal ascites as high mortality rates are reported in cases due to urine.

## Introduction

Urine ascites is a recognised complication of congenital obstructive uropathy, especially posterior urethral valves.[1] Such an event is rarely due to spontaneous bladder perforation. Urine ascites may be rapidly fatal and mortality rates of between 70 and 100 percent have been reported.[2,3] This report describes a case of urine ascites from a perforated urinary bladder and highlights the diagnostic value of micturating cysto-urethrography in such cases.

## Case Report

An 8-month old male child first presented at a peripheral hospital with a history of abdominal distension for a month, constipation for five days and haematuria for one day. Examination revealed a grossly distended abdomen as well as a suprapubic mass which, on rectal examination, was felt to protrude posteriorly, thus partially obstructing the rectum. The mass was thought to arise from the bladder. The child was therefore, referred to the University College Hospital (UCH), Ibadan, with a provisional diagnosis of a bladder tumour. On further questioning at the UCH, the child's mother admitted that his urine volume was often small and that he cried excessively during micturition. Besides, his development had been slow, and he was still unable to sit unsupported.

On examination, he weighed 5.5kg and was marasmic. The abdomen was distended with a girth of 45cm at the level of the umbilicus and 47cm at a point 5cm above the umbilicus. There was moderate ascites. The spleen and the liver were not palpable, but a slightly tender, non-mobile mass was felt extending from the suprapubis to the umbilicus. Rectal examination did not reveal any mass. The other systems were

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normal. The clinical impression was bladder distension from posterior urethral obstruction. A rhabdomyosarcoma of the bladder was also considered as a differential diagnosis.

Laboratory studies showed serum electrolyte levels to be within normal range, but the urea was elevated to 64mg/100ml (10.7mmol/L).

*Radiological examination*

Initial plain abdominal radiograph showed abdominal distension with ascites and bowel loops "floating" centrally. A vaguely defined double density, suggestive of a full bladder, was also seen within the pelvis. It was planned to precede excretory urography by micturating cysto-urethrography, so that vesico-ureteric reflux might be observed at the latter examination. However, the child died before the former examination could be performed. The micturating cysto-urethrogram revealed a small capacity bladder, with marked hypertrophy and trabeculation of its wall and a few diverticulae. The posterior urethra showed bulbous dilatation with a relatively narrow anterior urethra consistent with urethral obstruction from posterior urethral valves (Fig.1). No vesico-ureteric reflux was seen, but contrast material was noticed to leak from the anterior aspect of the bladder dome into the peritoneal cavity. A late abdominal radiograph (Fig. 2) showed generalised smearing of the abdominal viscera, particularly bowel loops and the liver, with positive contrast material producing a positive contrast parietogram.

Immediate surgery was not possible because the patient was considered too ill for general anaesthesia. He was managed with bladder drainage via a trans-urethral catheter, as well as fluid and electrolyte therapy, but he eventually died. An autopsy was not done.

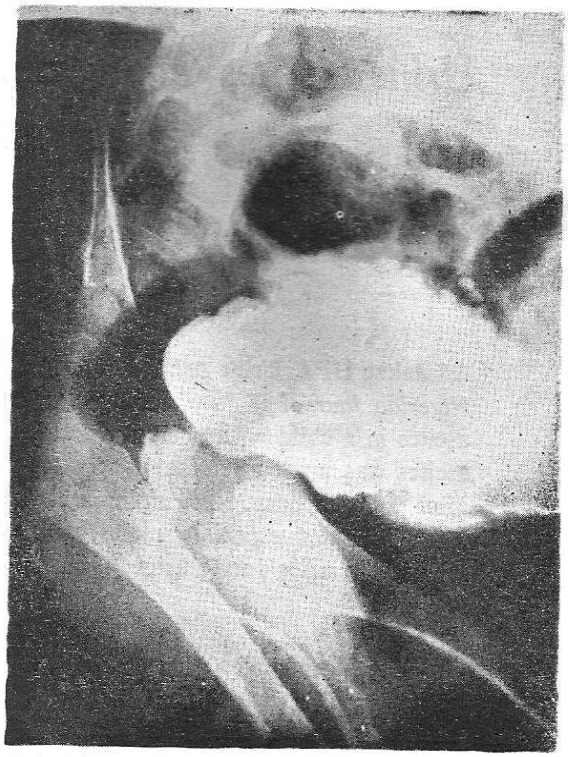

*Fig 1* *Micturating cysto-urethrogram (MCU) Note bulbous dilatation of the posterior urethra from posterior urethral valves. The bladder wall is trabeculated and there is extra-vesical leakage of contrast medium.*

## Discussion

Ascites in the neonatal period may be associated with gastro-intestinal, porto-biliary or urinary tract abnormalities,[1] but obstructive uropathy, particularly from posterior urethral valves, is probably the most important cause.[4] Of seven cases of neonatal ascites reported by Odita and Omene,[3] four were due to urethral obstruction from posterior urethral valves. In another recent report of six cases of neonatal ascites, all were due to urinary tract abnormalities: four due to posterior urethral valves and two due to intrinsic renal pathology.[5]

There are three possible pathways for escape of urine into the peritoneal cavity. First, urine may transude through the walls of a thinned and dilated urinary tract.[4,6] Secondly, perforation may occur in the upper renal tracts, commonly in a calyceal fornix with peri-renal extravasation

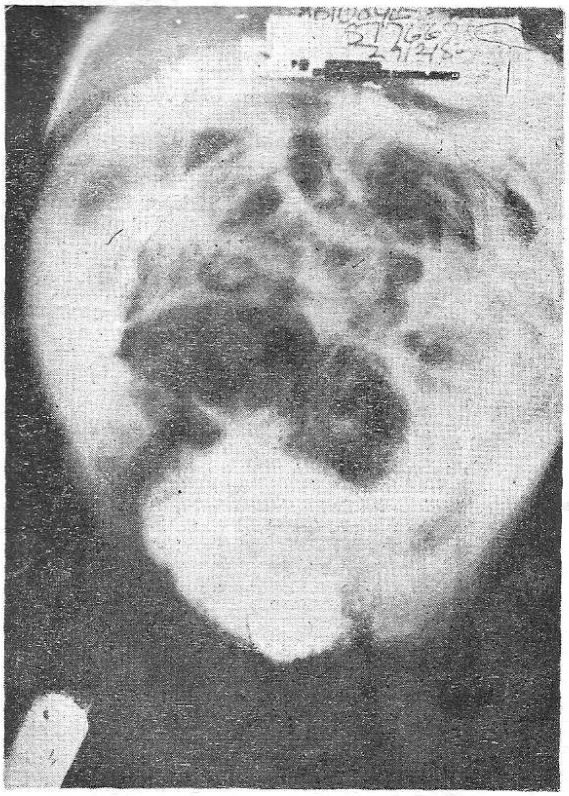

*Fig 2 Abdominal radiograph obtained 10 minutes after MCU. There is generalised smearing of the peritoneum and viscera with leaked contrast medium (positive and contrast parietogram). Note the well-defined liver outline.*

and eventual discharge into the general peritoneal cavity. Dockray[7] has described an important radiological P-sign which may be seen in such cases. The O-part of the letter "P" is formed by the kidney creating a filling defect within the extravasated contrast medium under Gerota's fascia. The dilated ureter then adds a tortuous leg to complete the letter "P". Swain and Tucker,[8] and Odita and Omene[3] have documented cases to confirm this method of ascites formation. Rarely, the extravasated urine could spread into the mediastinum and then rupture into the pleural cavity, producing a "urino-thorax'.[9] Lastly, though very rare, the urine could escape from a perforation in a bladder diverticulum or saccule,[10] as probably occurred in our patient.

When bladder perforation occurs early, especially in utero, continuous release of bladder pressure with formation of urine ascites prevents hypertrophy of the bladder and cystography often shows a smooth-walled bladder. Late perforation, as in the present case, is associated with marked bladder hypertrophy and diverticula formation. The bladder perforation in our patient probably occurred at the time of the first hospital attendance, when the mother gave a history of haematuria. However, the possibility of iatrogenic perforation could not be completely excluded. Although the immediate cause of death is unknown, chemical peritonitis from the contrast medium is a possibility.

In conclusion, it is worth stressing that posterior urethral valves may pose serious diagnostic problems, especially in cases without a history of poor urinary stream which is an important symptom in this disease. Ogunbiyi, Oduwole and Akingbehin[11] have reported a typically elusive case in which the diagnosis was finally made after four years and only after the child had received a course of cytotoxic drugs for suspected Wilm's tumour. It is therefore, suggested that a micturating cysto-urethrogram should be done in all neonates, especially males, with ascites.

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