CASE REPORT

Urinary Ascites in Newborn – A Rare Case Report

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Abstract:

Urinary Ascites in the newborn is a very rare condition. It is commonly secondary to posterior urethral valves (PUV) which are membranous folds extending from proximal urethra leading to obstruction to urine flow. Here we report a two days old male neonate delivered full term in a hospital who presented with gross, uniform tense distension of abdomen with massive scrotal oedema, secondary to massive urinary ascites as a result of rupture of renal calyces. In addition there was a peri-renal urinoma.

Keywords: Calyceal Rupture, Neonatal urinary ascites, Posterior Urethral Valves,

Introduction:

Isolated posterior urethral valves are a common urinary anomaly in male neonates. However, only a few cases of urinary ascites have been reported so far. This occurs due to calyceal rupture [1] or rupture of dome of urinary bladder in-utero both being rare causes [1, 2]. Isolated bladder rupture without antecedent cause is extremely rare [3]. Early detection and surgical intervention may prevent ensuing renal failure.

Case report:

We report a two days old male full term neonate weighing 3kg at birth, delivered in hospital to an oligohydramniotic mother without any intranatal problem. The baby presented with gross tense abdominal distension (Fig. 1) associated with massive scrotal and mild penile oedema (Fig. 2). Scanty dribbling of urine was observed.

The baby was well hydrated and there were no signs suggestive of pulmonary hypoplasia or Potter's

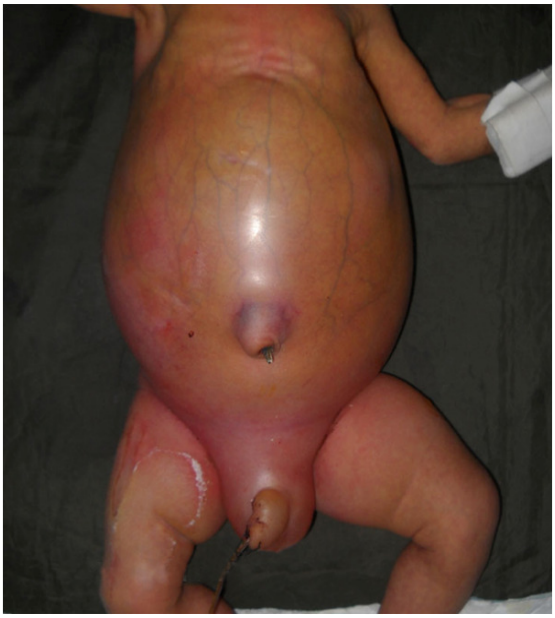

Fig. 1: Photograph Showing Gross Abdominal Distension

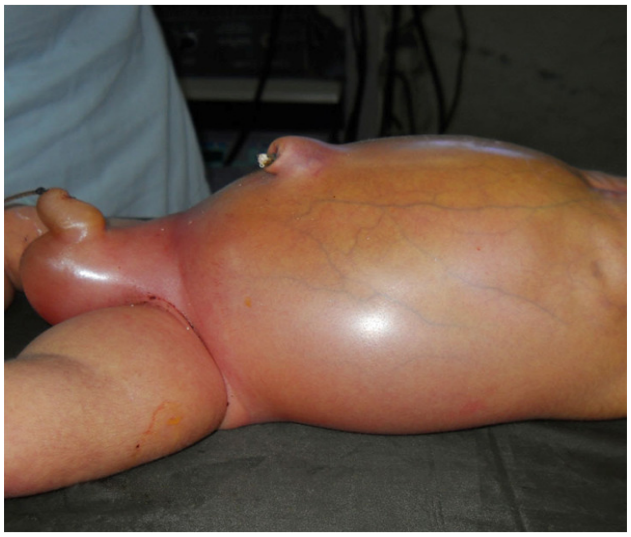

Fig. 2: Photograph Showing Tense Ascites with Scrotal Oedema

facies. Per abdominal examination revealed positive fluid wave confirming massive ascites and palpable urinary bladder. Bedside diagnosis of posterior urethral valves was suspected by placing catheter per-urethra wherein there was dribbling by the side of the catheter instead of in the lumen of the catheter.

Findings of Ultrasound detected

Massive ascites were with normal architecture of right kidney with cortico-medullary differentiation. (CMD). Left kidney showed loss of CMD, dilatation of calyces, and upper calyceal rupture with peri-nephric urinoma. Urinary bladder showed thickened wall with poor filling. (Fig. 3)

Renal function tests on admission showed blood urea 50 mg% and serum creatinine 1.08 mg%. Urine routine examination was normal and culture was sterile.

Initial management included –

Intravenous fluids with maintenance of electrolytes balance, antibiotics – (Ceftriaxone), urinary catheterisation and abdominal paracentesis follwed by glove drain insertion. After stabilisation and improvement, a micturating cysto-urethrogram (MCU) was performed and showed (Fig. 4) the underlying pathology of urinary tract.

Namely thimble shaped bladder with crenations and small capacity. It is also detected Grade V vesico-ureteric reflux on left side with extravasation of urine into peritoneal cavity with presence of posterior urethral valves (PUV).

The baby underwent cutaneous vesicostomy as the urethral calibre precluded cystoscopic management. Two months later cystoscopic fulguration of valves was undertaken. At 6 months follow-up the baby had adequate weight gain and development, and normal renal function.

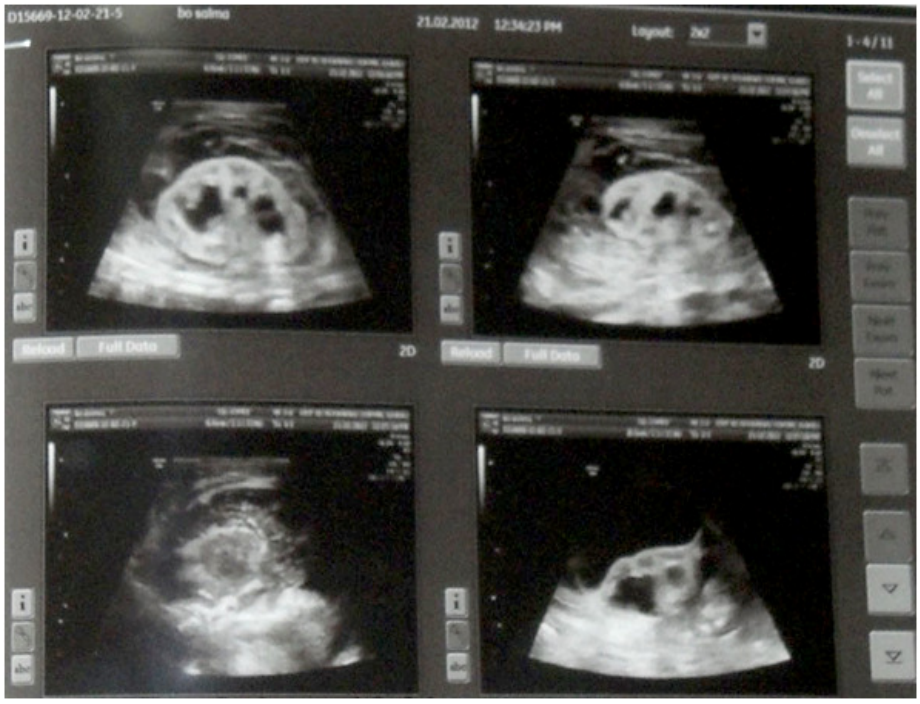

Fig. 3: Photograph of Ultrasonography Showing Massive Ascites with Loss of CMD, Dilatation of Calyces, Upper Calyceal Rupture and Peri-Nephric Urinoma on Lt. Side of Kidney & Bladder Shows Thick Wall with Poor Filling

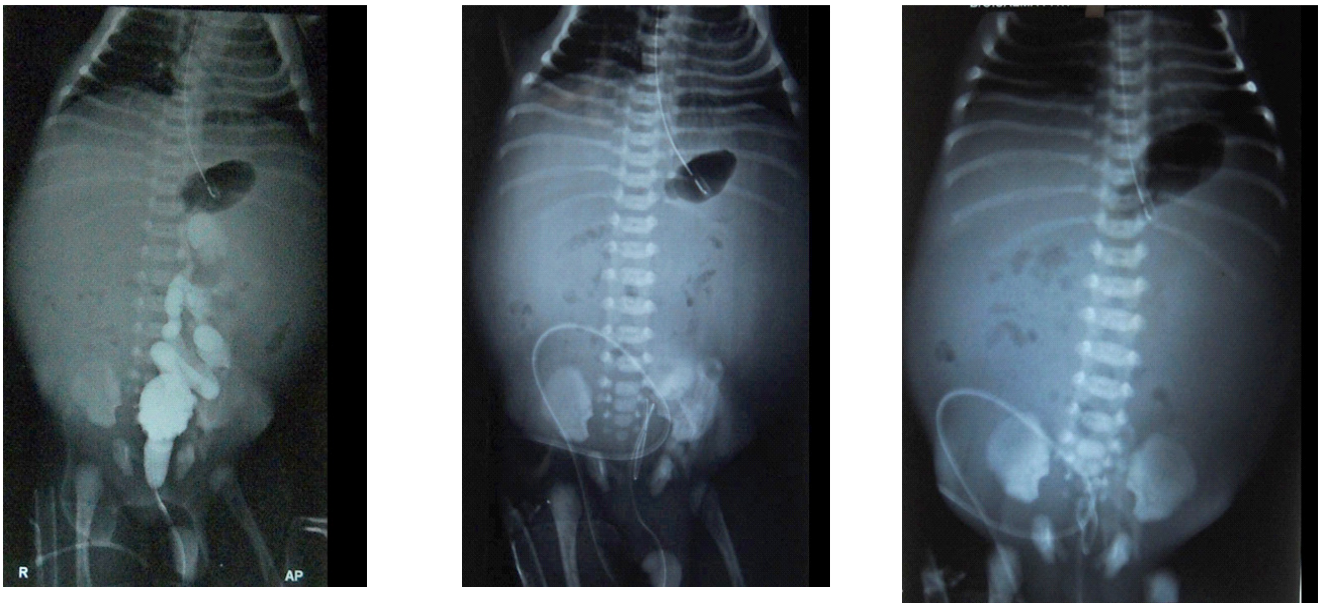

Fig. 4: Photograph of MCU showing Crenated Thimble Shaped Urinary Bladder, Grade V Vesico-Ureteric Reflux on Left Side, Extravasation of Urine into Peritoneal Cavity and Presence of Posterior Urethral Valves.

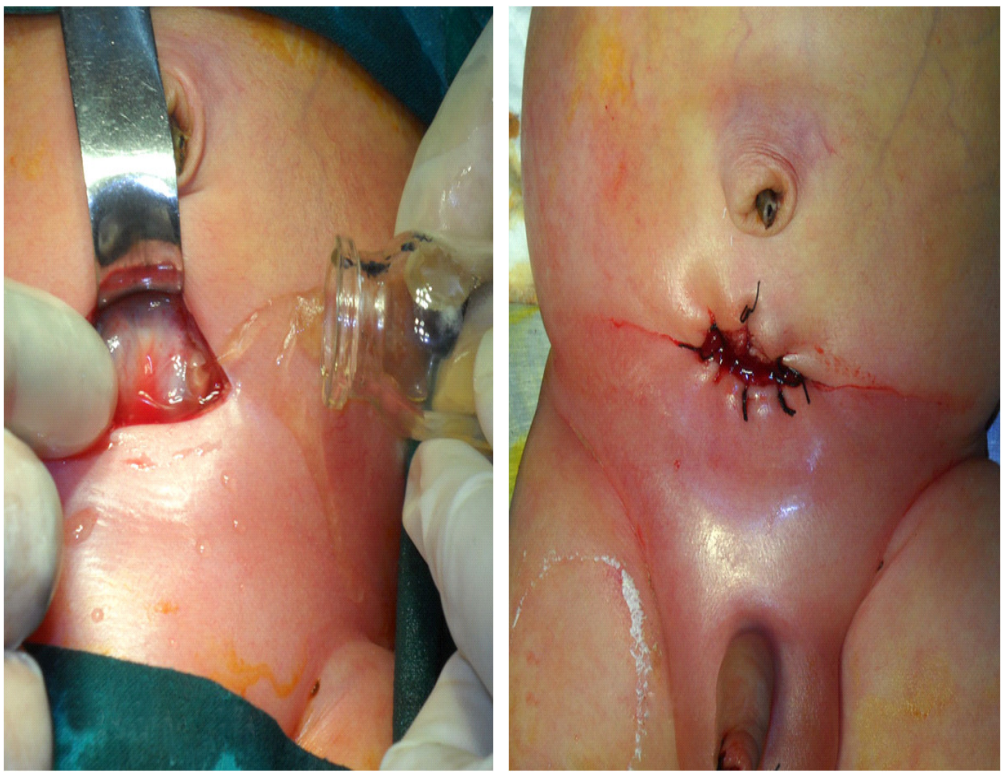

Fig. 5: Photograph showing Cutenous vesicotomy operation

Discussion:

Posterior urethral valves are exclusively present in males, the incidence being 1 in 5000 to 1 in 8000 [4, 5]. They are membranes obstructing the posterior urethra extending from the verumontanum to distal membranous urethra. The exact aetiology is unknown. It is postulated that PUV result from failure of posterolateral migration of urethra-vaginal folds with fusion and incomplete dissolution of urogenital membrane.

Antenatally PUV cause decreased urinary output and consequent oligohydramnios which may lead to pulmonary hypoplasia and Potter's syndrome in the neonate. In addition there is varying degree of associated renal dysplasia which may lead to renal failure.

Severe bladder outlet obstruction due to PUV may also cause back pressure changes such as hydroureteronephrosis. In severe cases a 'pop-off' event such as rupture of fornix [6] or dome of bladder occurs leading to urinary ascites [7].

In the newborn PUV may present as –

- Distended urinary bladder
- Hydronephrosis
- Uro-Sepsis
- Ascites
- Respiratory distress due to pulmonary hypoplasia
- Renal failure

Around 40% of ascites in the neonates is due to urinary pathology [8], commonly PUV. Ascitic aspirate usually shows electrolyte and creatinine levels similar to those of serum due to the large absorptive surface of peritoneum with high dialysing capacity. A keen search should be made for urinary conditions in such cases. An X-ray abdomen may show centrally placed small bowel loops. Ultrasound may show calyceal or bladder rupture along with thickened bladder wall and 'key-hole sign' suggestive of PUV. An MCU should be done in all cases suspected of PUV [1, 9]. Long term outcomes of PUV are favourable with prompt management of bladder pressure with adequate drainage. However around 25% of children end up with End Stage Renal Disease (ESRD) needing renal replacement in later life. Infants with urinary ascites and urinomas have worse prognosis compared to isolated PUV due to high bladder pressures.

Conclusion:

Ascites in the newborn is a rare condition with different causes such as biliary, liver, cardiac, chylous and urinary diseases [1, 9]. Ascitic fluid aspirate analysis may be inconclusive. There should be a high index of suspicion for a urinary cause by ultrasound which may demonstrate rupture of calyx or bladder. This may direct the investigation by specific method such as a micturating cysto-urethrogram in this case. Given the poorer prognosis and morbidity associated with both urinary ascites and posterior urethral valves, prompt investigation alongside stabilisation must ensue.

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