Renal pelvis haematoma causing pelviureteric obstruction: a first case of Antopol-Goldman lesion in a neonate

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Dear Editor,

Neonatal hydronephrosis is a relatively common problem. Most are diagnosed by routine antenatal ultrasound screening, with an estimated frequency of between 0.6% to 1.4% (1,2). The most common cause is pelvi-ureteric junction obstruction (PUJO), which accounts for more than 60% of the neonatal hydronephrosis with genuine pathology. Vesicoureteric reflux, vesico-ureteric junction obstruction are the other causes (3). The pathology for PUJO is usually due to an intrinsic stenosis, followed less commonly by extrinsic compression (4).

In adults, Antopol and Goldman first described a subepithelial haematoma of renal pelvis causing urinary obstruction in 1948 (5). In subsequent publications by others, hematuria was reported to be the usual presenting symptom, with radiological investigations showing a renal mass. Unfortunately, many of these lesions were mistaken as renal neoplasms and the patients were subjected to unnecessary nephrectomies (6,7). Here we report a preterm baby who presented with pelvi-ureteric junction obstruction caused by a haematoma. We believe that this is the first reported case of Antopol-Goldman lesion in the pediatric population.

Our patient was a girl born prematurely at 29 weeks of gestation by spontaneous vaginal delivery. Antenatal ultrasound did not show any abnormality.

The premature labor was due to antepartum haemorrhage from placental previa. One dose of antenatal steroid was given before delivery. Her birth weight was 1.2 kg.

She was cyanotic and apnoeic at birth and required intubation and mechanical ventilation, and was given two doses of surfactant. Our patient was found to have renal impairment since day 1 of life, with the level of creatinine climbing to 140 umol/l on Day 5 of life. There was no oliguria nor macroscopic haematuria. She was

treated as having dehydration with intravenous fluid resuscitation and her renal function slowly improved subsequently. No ultrasound was done at this point because of improving renal function.

Abdominal distension was noted on day 11. Abdominal X-ray showed evidence of pneumatosis intestinalis, which was compatible with necrotizing enterocolitis. This was treated with conservatively and our patient made an uneventful recovery. Feeding was resumed after 5 days. On Day 21 of life, physical examination revealed a right upper quadrant mass measuring around 2 cm in diameter. Ultrasonogram of the abdomen showed a grossly hydronephrotic right kidney measuring 5.2 x 4.8 x 3.2 cm. A solid mass (2.1x1.4x1.8cm) was noted at right renal hilum and extending into right ureter (Figure 1A). There was no significant vascular flow demonstrated in the lesion. The left kidney was unremarkable. Routine blood tests, including the clotting profile, were normal. Computed tomography (CT) of the abdomen was performed to ascertain the nature of the mass at right renal hilum. This showed a mildly hyperdense lesion inside the dilated right renal pelvis causing hydronephrosis. No contrast enhancement was noted inside the mass (Figure 1B). A radiological diagnosis of renal pelvis haematoma causing pelvi-ureteric junction obstruction was made. As there was significant PUJ obstruction with evidence of cortical thinning, right-sided percutaneous nephrostomy was performed. The antegrade pyelogram showed complete obliteration of right renal pelvis. The initial urine drained from the nephrostomy contained clots, which was compatible with the initial diagnosis. Serial ultrasound performed subsequently showed decreasing size of the mass lesion, suggestive of resolving hematoma. A repeated right-sided antegrade pyelogram was done 1 month later. This showed prompt contrast drainage to urinary bladder and no

intra-luminal filling defect could be detected (Figure 2). The nephrostomy tube was removed and the patient was discharged 2 weeks later. Reassessment ultrasound at 3 month of age showed only mildly dilated right renal pelvis, with an otherwise normal urinary system. No mass lesion was detected. Renal MAG3 scan was also performed at 3 month of age. It showed satisfactory function and perfusion over both kidneys with no evidence of obstruction.

In the paediatric population, the most common cause of PUJO is due to intrinsic narrowing. Intra-luminal obstruction is rare in neonates, with only a few reports found with acute renal failure caused by obstructive candidal bezoars (8,9). Although the Antopol-Goldman lesion was first reported in 1948, it remains a rare entity in adults and has never been reported in neonates. Most of the affected patients presented with haematuria and filling defects in the renal pelvis on radiological investigations. Due to the possibility of renal cell carcinoma, most adults required nephrectomy before coming to the correct diagnosis. Although some patients did report a history of minor trauma before development of this lesion, there has never been a definite cause that can be attributed to this pathology.

In our case, the echogenic mass lesion detected on US showed that it was not arising from the adjacent renal tissue. Furthermore, there was no doppler flow detected inside the lesion nor was there contrast enhancement on CT to suggest the presence of tumor blood vessels. Reviewing the history in our patient, we believe that the stormy course soon after birth, together with deteriorating renal function, would suggest the possibility of the haematoma having been present since birth. Since renal cell carcinoma was extremely rare in neonates, this diagnosis was not considered and a conservative approach adopted. We are not able to provide a definite explanation for

the cause of the haematoma in our patient, although premature labour and antepartum haemorrhage could have contributed towards the formation of a haematoma. In conclusion, we report here the first case of an Antopol-Goldman lesion in a neonate and hope that this case can raise the awareness of clinicians.

Reference

1. Woodward M, Frank D. Postnatal management of antenatal hydronephrosis. *BJU* Int 2002; **89(2)**:149-56.
2. DiSandro MJ, Kogan BA. Neonatal management. Role for early intervention. *Urol Clin North Am.* 1998; **25(2)**:187-97.
3. Aksu N, Yavascan O, Kangin N, et al. Postnatal management of infants with antenatally detected hydronephrosis. *Pediatr Nephrol*. 2005; **20(9)**:1253-9.
4. Dewen PA, Ng KP, Ashwood PJ. The relationship of age to pathology in pelviureteric junction obstruction. *J. Paediatr. Child Health.* 1998; **34(4)**: 384-386.
5. Antopol W, and Goldman L.Subepithelial hemorrhage of renal pelvis simulating neoplasm. *Urol Cutan Rev.* 1948; **52**: 189-195.
6. Kim SJ, Ahn HS, Chung DY, Kim YS, Lee EJ, Park KH. Subepithelial hematoma of the renal pelvis simulating neoplasm (Antopol-Goldman lesion). *Urol Int*. 1997;**59(4)**:260-2.
7. Iczkowski KA, Sweat SD, Bostwick DG. Subepithelial pelvic haematoma of the kidney clinically mimicking cancer: report of six cases and review of the literature. *Urology.* 1999; **53(2)**:276-9.
8. Hari P, Srivastava A, Gupta AK,et al. Neonatal renal failure due to obstructive

candidal bezoars. *Pediatr Nephrol*. 1997; **11(4)**: 497-8

9. Laufer J, reichman B, Graif M et al. Anuria in a premature infant due to ureteropelvic fungal bezoars. *Eur J Paediatr.* 1986; **145**:125-7

Figure legends:

Figure 1 - Imaging studies of the right kidney of the patient demonstrating the obstructing lesion at the right renal pelvis. (A) Ultrasound showing gross hydronephrosis and an echogenic mass lesion inside the renal pelvis (arrow). (B) Post-contrast oblique coronal computed tomography showing the mass lesion causing outflow obstruction (arrow).

Figure 2 - Right antegrade pyelogram showing contrast drainage to the urinary bladder with no evidence of intra-luminal lesion.

Fig 1A

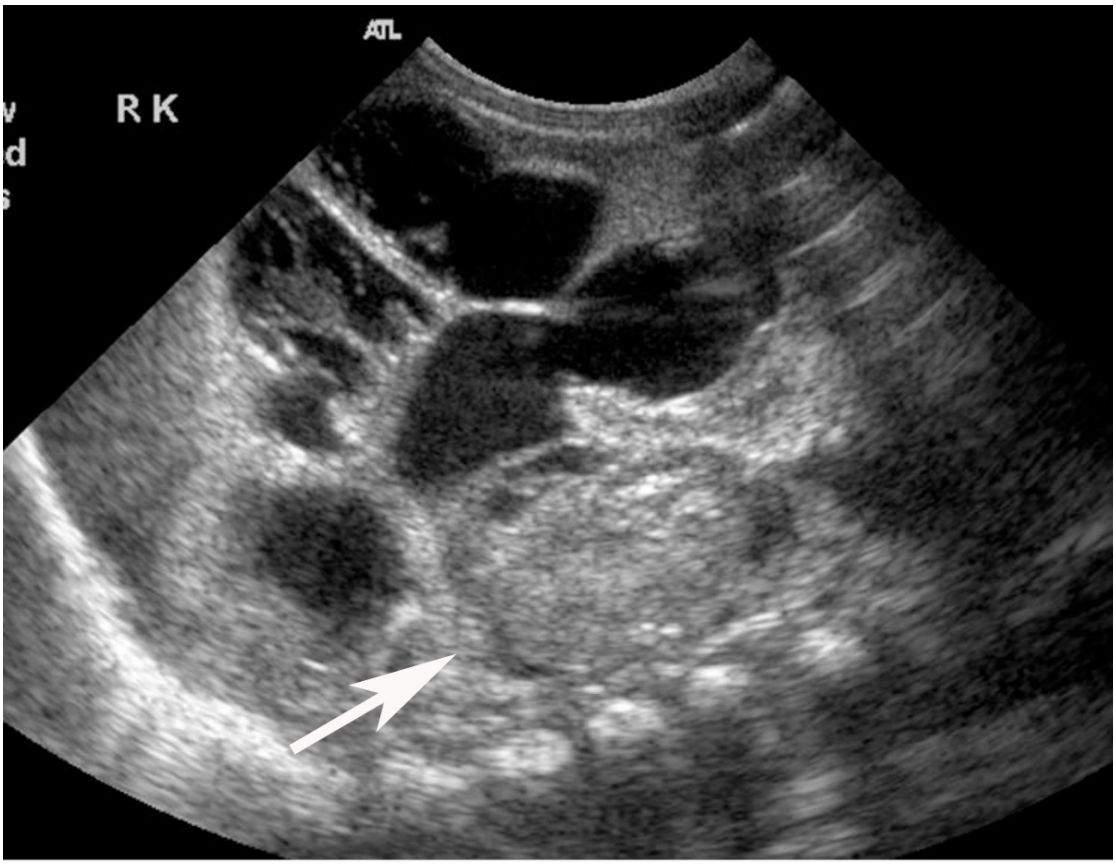

Fig 1B

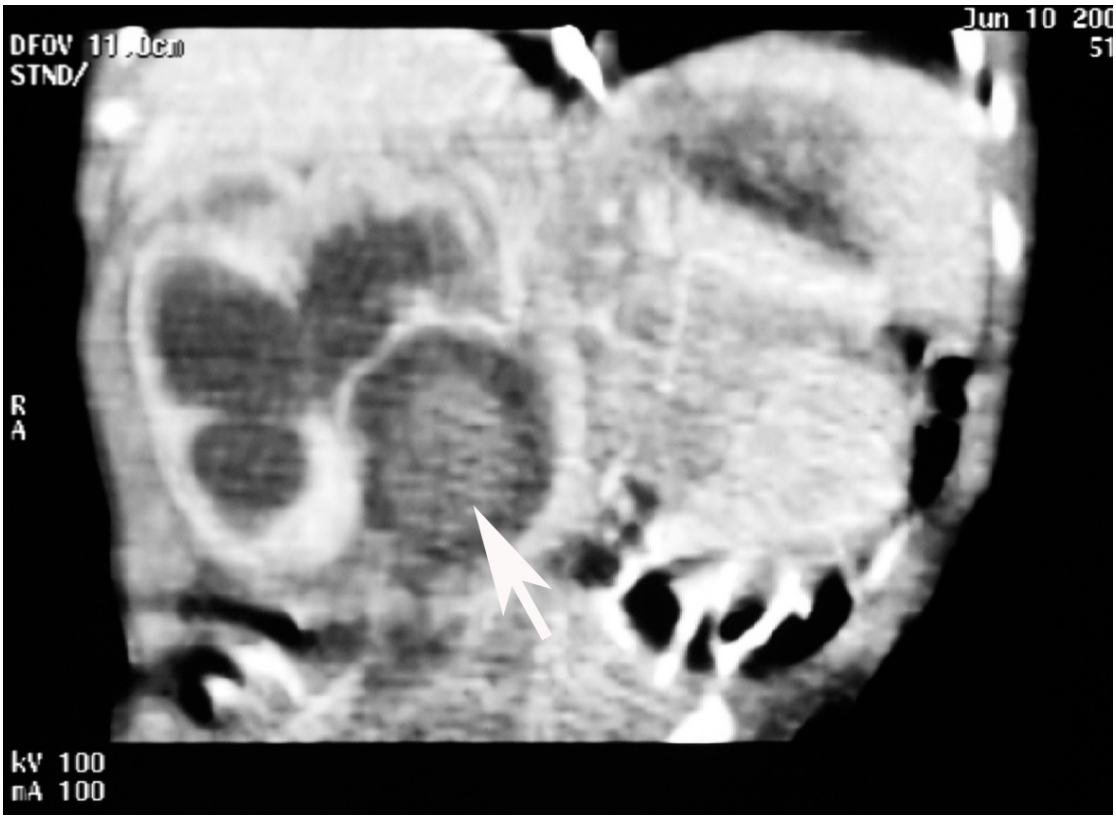

Fig 2