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Case Report

Giant Hydronephrosis in a Newborn Causing GIT Obstruction: An Unusual Association

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Abstract

Hydronephrosis in a newborn is typically asymptomatic. It is one of the most common and usually benign anomalies detected on ultrasonography, with only a minority causing significant problem requiring urgent intervention. We present a neonate with giant congenital hydronephrosis presenting with feed intolerance due to duodenal obstruction. The baby was successfully managed at our centre and discharged on full feeds.

Keywords: Giant Hydronephrosis; Newborn; GIT Obstruction

Introduction

Hydronephrosis of the newborn is a commonly encountered challenge in nurseries. Prenatal screening with ultrasonography can detect antenatal hydronephrosis, which is graded by trimester and anterior-posterior diameter of the renal pelvis: most are mild. However, in rare instances it may go undetected either due to twin pregnancy, with one fetus obstructing optimum visualization of the second fetus, or technical and manual errors. Usually asymptomatic, hydronephrosis may sometimes be large enough to press on surrounding structures and present with atypical features. With heavy reliance on antenatal ultrasonography to foresee such anomalies present congenitally, it may be considered later in the differential when evaluating a newborn with obstructive symptoms.

Case Report

We report a neonate, second and smaller twin, with antenatally undetected hydronephrosis, which was severe enough to cause duodenal obstruction resulting in feed intolerance. This female neonate was born to a primigravida mother, booked pregnancy, and was a product of IVF conception. Our patient was second of the two twins, born at term gestation, with low birth weight and was smaller of the two twins. Baby passed urine and stool within the first 24 hours of life. Due to sick condition of mother post caesarian section, the babies were kept in nursery. When feeds were started on day 1 of life with expressed breast milk, this neonate started having vomiting containing the ingested milk. Physical examination was significant for abdominal fullness, and the baby while not lethargic, had suboptimal activity for a term baby. An open NG tube was kept in place. Lab workup showed abnormal renal function with creatinine of 3.4 mg/dl, hyperkalemia: 5.6 mEq/l. Sepsis screen was negative. There was no metabolic acidosis. Urine output was age adequate. We attributed the deranged creatinine as a reflection of mother's levels, since the workup was done on first day of life. Radiographic imaging was done next, including abdominal X ray which showed gaseous distension of the bowel loops with fullness of the mid abdomen. Child was managed conservatively with IV fluids and IV antibiotics at this stage. The blood culture report did not grow any organism after 48 hours of incubation. Urine cultures were also negative. But the baby continued to vomit after feeds. Repeat creatinine was 1.5 mg/dl. USG abdomen with KUB was done on day 3 of life, which revealed gross right renal hydronephrosis with moderately dilated right renal ureter and early cortical thinning. There was also mild to moderate left renal hydronephrosis with distended bladder, suggestive of vesico-ureteric junction obstruction bilaterally. There was addition obstruction of

the duodenum by the enlarged kidneys which we attributed was the reason for feed intolerance in our child. Conservative management was continued with IV Fluids, IV antibiotics, monitoring of urine output with a urethral catheter in situ. By the 5th day of life, the renal functions and electrolyte imbalance had normalized, and feeds were restarted. This time she tolerated feeds with infrequent episodes of vomiting. Follow up USG abdomen with KUB on day 7 of life showed resolution of the intestinal obstruction seen in the previous scan, with moderate right renal hydronephrosis, and mildly dilated entire right ureter with abrupt cut off at the right vesico-ureteric region. There was no hydroureteronephrosis on the left side. Repeat scans have been scheduled at 1 month of age. Baby was discharged on antibiotic prophylaxis.

Discussion and Conclusion

Bowel obstruction due to giant hydronephrosis is a rare condition. Most cases of huge hydronephrosis in a newborn are detected antenatally, however this finding was missed in our case. Many definitions for giant hydronephrosis have been reported in the literature, but none in neonates. Historically, only 50% of huge hydronephrosis cases are properly diagnosed because of its non specific clinical presentation. There are no long-term follow-up and outcome data in the literature on giant hydronephrosis available especially in neonates including its definition [1]. 41 - 88% of antenatal hydronephrosis is transient hydronephrosis [2], with another 30% occurring due to ureteropelvic junction obstruction, followed by only 5 - 10% cases in which vesicoureteral junction obstruction was implicated [2]. So far, to the best of our knowledge, only one case of giant hydronephrosis presenting as intestinal obstruction in neonates has been reported [3], where urgent renal drainage with pyelostomy was required. The cause was implicated to be ureteropelvic junction obstruction.

Our report presents an interesting case of GIT obstruction caused by giant hydronephrosis secondary to likely VUJ obstruction in a female child. Further tests have been planned to confirm the primary etiology of hydronephrosis. Due to unusual nature of presentation, this differential is not readily considered when evaluating for causes of feed intolerance in NICUs over the world, and this is the point we want to emphasize. Our patient required no urgent invasive procedure. This could be due to transient nature of antenatally acquired hydronephrosis which is the most common etiology in literature till date [2]. However, unless this condition is considered in our differentials, corrective procedures, if required, can be delayed causing avoidable morbidity and even mortality. Our case report emphasizes the need for watchful consideration in neonatal hydronephrosis as many are likely to self resolve, pertinent need for antenatal detection particularly in cases where it is likely to be missed, and consideration of hydronephrosis, in etiology for feed intolerance in sick neonates in the NICU.

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Conflict of Interest

None.

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