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DYSTOCIA DUE TO FETAL ABDOMINAL DISTENSION: ANTICIPATION AND TIMELY ACTION.

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ABSTRACT Dystocia due to fetal abdominal enlargement is a rare entity causing obstructed labour. Here we present a case of rare congenital anomaly of the foetus, autosomal recessive polycystic kidney disease that leads to fetal abdominal distension causing dystocia. We managed it by ultrasound-guided fetal abdominal tapping of fluid easing the vaginal delivery. This patient was registered in the early third trimester and presented in labour with an enlarged fetal abdomen diagnosed on bedside USG. With the advent of high-resolution ultrasound, polycystic kidneys in the fetus can be picked up in level II scans, and also enlargements of the fetal abdomen and unexpected obstructed labour can be prevented. These women can be offered counselling and timely obstetric intervention so that labour emergencies like dystocia due to soft tissues are prevented in the future.

KEYWORDS : Dystocia, Fetal abdominal distension, Polycystic kidney disease.

INTRODUCTION

The causes of dystocia have been described classically as a fault in the power, passenger, or passage. The passenger is the fetus which can cause obstructed labour by malposition, macrosomia, congenital anomalies, or enlargement of fetal parts. Dystocia due to fetal abdominal enlargement is comparatively rare. One of the first case of dystocia due to fetal abdominal distension (ascites) was reported by Mauriceau`s and et al in 1681[1]. Dorland has described six entities of fetal abdominal enlargement which are – a collection of fluid in the peritoneal cavity or true ascites, distension of fetal bladder, ureter or kidney and pelvis which in turn produces abnormally enlarged abdomen, congenital polycystic kidneys, cystic enlargement, and tumours of the liver, distension by the fluid of the genital tract in the female[2]. The ultrasound diagnosis criteria of polycystic kidney disease include bilateral changes of large hyperechogenic kidneys with poor cortico-medullary differentiation. Macrocysts (>10 mm) in the polycystic kidney disease are unusual and suggest multicystic dysplasia; whereas bilateral cysts of 5–7 mm are more common in these cases. Prenatal ultrasound can detect congenital anomalies and enlargements of the fetal abdomen and unexpected obstructed labour can be prevented.

Autosomal recessive polycystic kidney disease is a rare condition with a poor prognosis[3]. Ultrasound examination can show bilateral polycystic kidneys with massively increased abdominal circumference[3]. This condition is rare and carries a guarded prognosis if the renal tissue is thinned out due to cysts because it indicates minimal functioning kidneys. Hence termination of pregnancy can be offered in women detected early. However, women who have advanced pregnancy, the possibility of labour dystocia should be kept in mind.

CASE DISCUSSION

A 30-year-old primigravida, married for two years, non-consanguineous marriage, registered at 32.5 weeks of gestation with an ultrasound report done at 32 weeks suggestive of a single live foetus of 31.1 weeks of gestation. Bilateral enlarged polycystic kidneys of foetus (Figure 1 and 2).

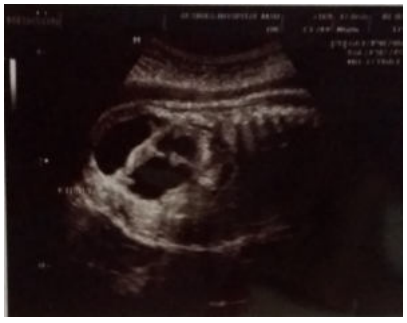

Figure 1- Ultrasonography suggestive of enlarged kidneys with cystic structure in axial view.

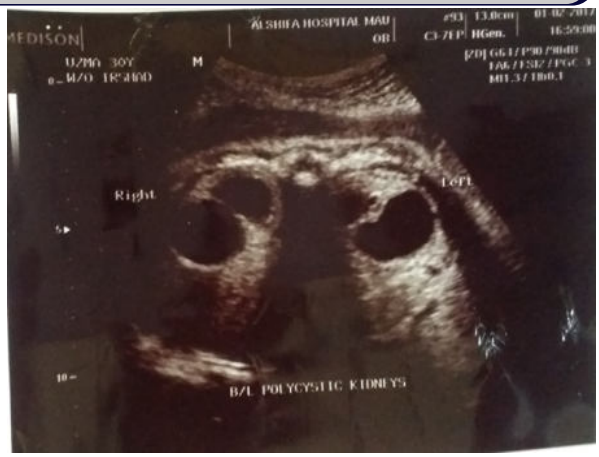

Figure 2- Showing the bilateral fetal polycystic kidneys in Sagittal view

Patient was advised a review detailed anomaly scan and paediatric urology reference. However, patient did not follow up as advised and came in labour at 33.4 weeks of gestation at 2:00 am. A review scan was done in the labour ward which showed severely bilateral polycystic kidneys with thinned out renal cortex and also fetal ascites. There was severe oligohydramnios with AFI- 1 cm. She was getting uterine activity of 1/10/15-20 seconds. Her Per vaginum examination showed cervical os 1 cm, 30% effaced, vertex, the station at |-3|, membranes present and flat, pelvis adequate. Her intra-partum monitoring showed fetal heart rate decelerations up to 80b/min with poor variability. The woman and relatives were informed regarding the poor prognosis of the fetus because of bilateral polycystic kidneys with thinned out renal cortex. The women and relatives gave negative consent for caesarean section and insisted on vaginal delivery. The labour was then augmented by oxytocin. She progressed further and became fully dilated and fully effaced at 9:30 am. However, by this time, her fetal heart were absent. At 10:00 am, the fetal head delivered and further delivery was difficult. The shoulders were delivered with difficulty but further traction failed to deliver the trunk and abdomen. The patient was in agony as labour was obstructed with the fetus incompletely delivered. The distended fetal abdomen could not be accessed vaginally as it was high up, the maternal pelvis was entirely occupied by fetal thorax. The preterm fetal vertex had easily negotiated the maternal pelvis and there was dystocia due to the enlarged fetal abdomen. (Figure 2). The fetal abdomen had to be immediately decompressed to allow further delivery. However, decompression from the vaginal end was not possible as the cervix was tightly over the chest, and the abdomen was not felt even with difficulty.

Immediately, decision to abdominally decompress the fetal abdomen was taken. The labour ward bedside ultrasound was kept ready as the

difficulty was anticipated in this delivery. Informed consent of abdominal tapping was taken from the patient for fetal abdominal decompression. The abdomen was cleaned, painted with a betadine solution, and draped. Under all aseptic precautions, an 18 gauge spinal needle was inserted in fetal abdominal ascites under ultrasound guidance and this was attached to an intravenous set and fetal ascetic fluid was removed. The needle was then redirected towards the cystic kidneys and fluid from it too drained out. The total amount of 200 ml fluid was drained and then in the next strong uterine contraction, the rest of the trunk, abdomen, and entire baby delivered at 10:40 am. The placenta and membranes were too delivered out completely. Meanwhile, 10 units oxytocin started in drip and after the delivery of the placenta, Inj. methyl ergometrine was given intramuscularly as atonic postpartum haemorrhage was expected after difficult and obstructed labour. Cervical tracing and vaginal tracing was done as there were many manoeuvring and manipulations. However, there were no lacerations nor extensions of episiotomy.

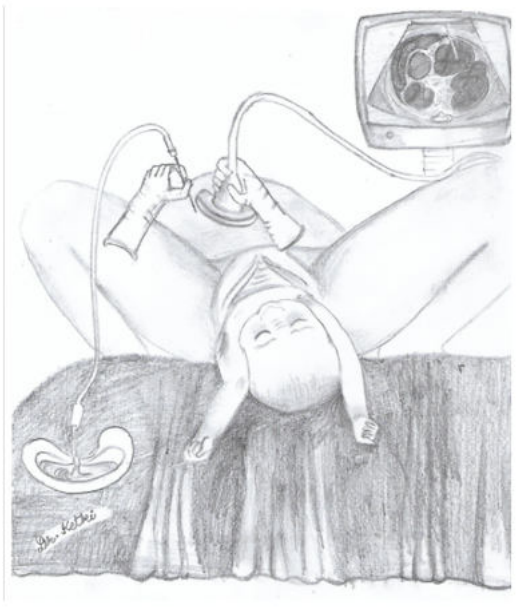

Figure 3: Figure 3 depicts the events during labour, fetal obstruction can be noted at the level of fetal abdomen after delivering shoulder, fluid from polycystic kidneys were drained under ultrasonography guidance.

Gross examination of the fetus - 2.4 kg male fresh stillborn fetus with an enlarged abdomen. No other external congenital malformations were noted. The relatives were however not willing for clinical autopsy. The patient was given intravenous antibiotics for two days and lactation suppression. She was discharged on day 3 with advice regarding contraception, peri-conceptional counselling, and early registration in the next pregnancy.

DISCUSSION

Fetal ascites as a cause of dystocia is rare and not many cases have been reported in history. Jhaveri et al have described a case of dystocia in India way back in 1973 due to abdominal distention in a case of persistent cloaca[4]. The fetal abdomen was decompressed after a portable X-ray and also thorax had to be perforated and evisceration performed for delivery of the baby[4]. Parveen Haider et al reported two cases of fetal dystocia is one of the cases, outlet forceps were applied for delivery, however, the head became decapitated and the rest of the baby had to be delivered by lower segment caesarean section after draining fluid from the fetal abdomen. The second case was a fetus with gross fetal ascites with breech presentation. She was delivered vaginally after draining the fetal ascites vaginally. Neelam Swaroop et al reported a case of a malformed fetus with Prune Belly syndrome with multi-septate cystic fetal hydronephrosis and distended fetal bladder with dystocia which was managed by abdominal tapping and decompression of the fetal abdomen[6].

In our case, the dystocia was caused at the level of the fetal abdomen by enlarged polycystic kidneys with fetal ascites. Polycystic kidney disease is a genetic condition characterized by the accumulation of fluid-filled cysts in the kidney and other organs[7]. It can be inherited as an autosomal recessive polycystic kidney disease (ARPKD) or autosomal dominant polycystic kidney disease (ADPKD). The incidence of ARPKD ranges from 1: 6000-1:40,000 live births[8, 9]. Since clinical autopsy could not be done, whether there were associated cysts in the liver or hepatic portal fibrosis and pulmonary hypoplasia could not be commented upon. Since the parents and previous generations were not having any polycystic kidney disease, the mode of inheritance could be autosomal recessive polycystic kidney or a new mutation.

With the advent of high-resolution ultrasound, polycystic kidneys in the fetus can be picked up in level II scans and also associated congenital anomalies. These women can be offered counselling and timely obstetric intervention so that labour emergencies like dystocia due to soft tissues are prevented in the future. Also in emergency situations, obstetrician should be vigilant for timely action to handle such emergency situations. As destructive procedures are obsolete in this era, ultrasound guided procedures provide the cake walk for such difficult deliveries.

DECLARATIONS

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