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Ultrasound of solid fetal neck mass before and after delivery

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Abstract

Fetal tumors are very rare congenital anomalies. Their incidence is around 1 in 12 000 to 1 in 30 000 births. Among them, neck tumors represent an important group. This is a very specific and unique fetal and neonatal problem. Initially, like other fetal tumors, neck masses may negatively affect the fetal cardiac function because of increased cardiac afterload and preload. Secondly, they may affect the patency of fetal airways, including trachea, which may have fatal consequences after birth. In addition, big neck tumors, especially teratomas, may cause fetal lung hypoplasia, because of wedging the lungs into the apices of chest cavity. Finally, they have great cosmetic effect on the child.

A differential diagnosis of the fetal neck tumor or cystic lesions include lymphangioma, hemangioma, goiter, teratoma and neuroblastoma.[1]

Keywords: Neck Tumor, Fetal, Teratoma, Fetal Goiter.

Introduction

Fetal thyroids mature at approximately 12 weeks of gestation, but active secretion of hormones does not occur before the middle of the second trimester, at around 17 weeks. A fetal goiter is any increase greater than two standard deviations above the 95th percentile. It is a rare pathology and is present in one in 40,000 pregnancies.[2]

Congenital hypothyroidism rarely causes a clinically significant neck mass in newborns. We present the case of a newborn with congenital hypothyroidism and significantly enlarged goiter and discuss imaging considerations.[3]

Case Presentation

A 26-year-old non consanguineously married woman (G3, P2) booked antenatal case with no pre-diagnosed fetal anomaly or tumor had come for routine fetal growth scan. No history of fever, rash, drug usage during current

pregnancy. Her thyroid function tests indicating subclinical hypothyroidism, blood parameters were within normal limits. No gestation induced diabetes or hypertension. She had undergone previous C-sections.

TSH	4.706 mIU/L
FREE T3	3.36 pg/Ml
FREE T4	0.94 ng/Dl

Table 1: Thyroid profile of mother during pregnancy. Raised TSH s/o of subclinical hypothyroidism.

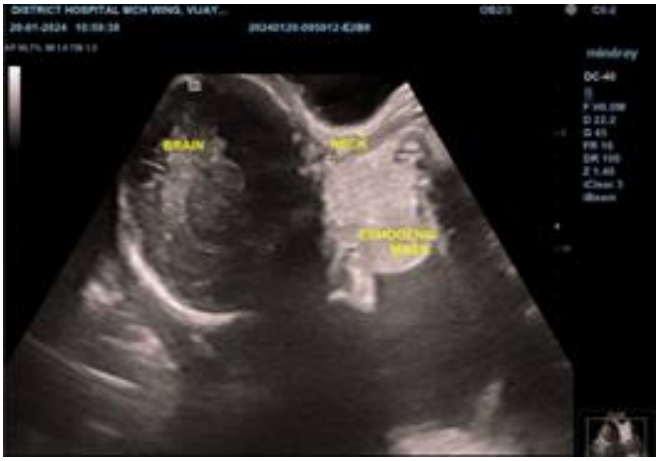

Figure 1: Echogenic mass noted in anterior neck.

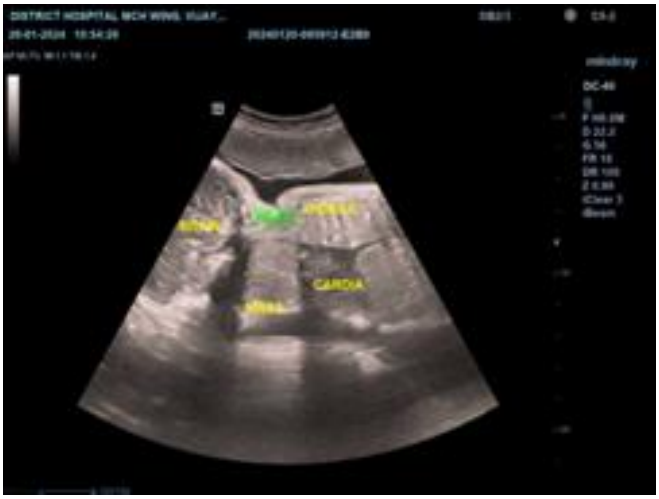

Figure 2: Sagittal view showing cranial to caudal: brain, neck mass and thorax with cardia.

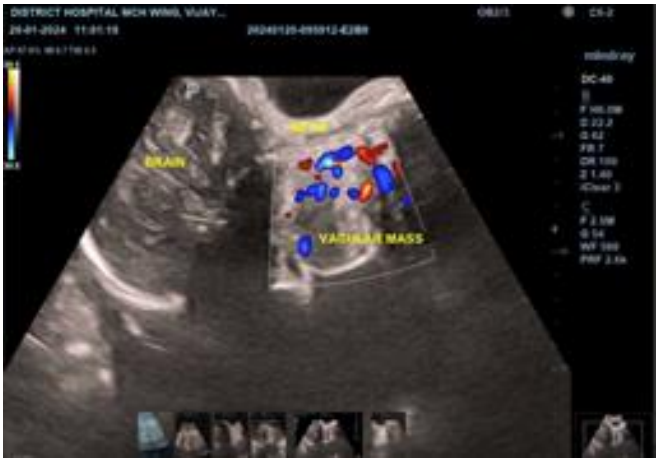

Figure 3: Significant internal vascularity noted within the neck mass

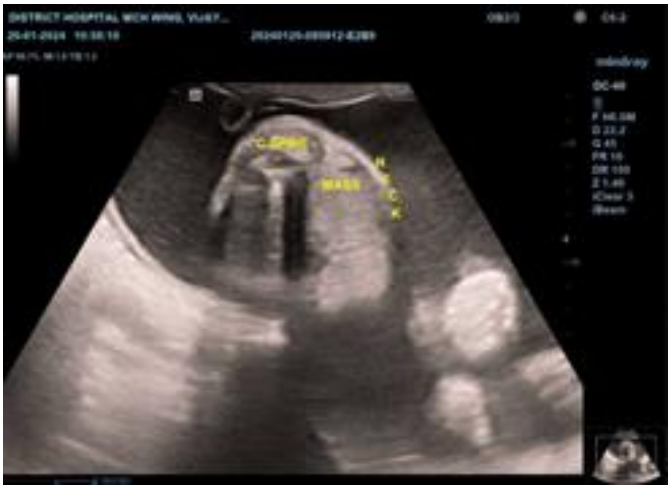

Figure 4: Axial section with cervical spine and anterior neck mass. (posterior acoustic shadow of c spine seen)

USG Findings: Incidental finding of a well-defined echogenic solid anterior neck mass measuring 8 x 8 cm, color Doppler revealed significant vascularization compatible with fetal goiter. Aminotic fluid was also increased for gestational age (35 weeks 6 days)

Congenital goitre and cervical teratoma were given as differentials considering significant internal vascularity and solid nature of the mass. The case was followed up till delivery of the baby.

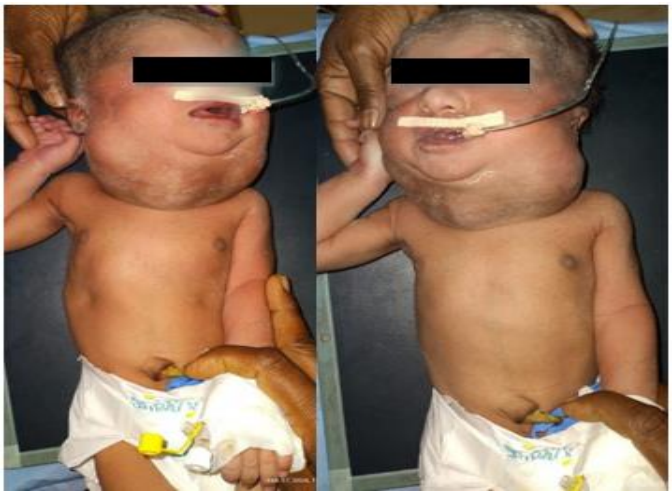

Figure 5: Immediately after birth neonate seen having huge anterior neck mass.

TSH	>100 mIU/L
T3	0.46 ng/ml
T4	1.86 mcg/Dl
Anti TPO Abs	5 IU/Ml

Table 2: Neonatal thyroid profile and autoantibody titre. Raised TSH levels, indicating hypothyroid status of the neonate.

USG of the neonatal neck reveals

Figure 6: USG showing enlarged hypervascular thyroid gland.

Enlarged bilateral thyoid lobes, isthumus showing increased vascularity compressing on underlying tracheal airway.

Treatment and results: The neonate was put on Thyroxine 40mcg/day X 10 days of birth

↓

dose tapered over hospital stay of 1 month

Thyroxine 25mcg/day (at time of discharge)

Maintenance of 12.5 mcg/day was given post discharge. Infant has significant reduction in the size of swelling by the time of discharge and looks like this.

Figure 7: Complete resolution of thyroid swelling in response to treatment.

Discussion and Conclusion

Fetal goiter is a diffuse enlargement of the fetal thyroid gland that can occur regardless of the status of the thyroid's function. However, it has been reported that it is often associated with a change in thyroid function. It is an extremely rare condition that affects one out of 30,000–50,000 pregnancies.[4]

Usually, dyshormonogenesis is responsible for Fetal goiter. Blockage of hormone biosynthesis increases fetal TSH levels, and this leads to Fetal goiter. Fetal goiter can lead to hyperextension of neck, polyhydroamnios, and compression of vascular structures in neck and

edema. Mental and motor retardation also have been reported. These reasons make fetal hypothyroidism important for early diagnosis and appropriate hormone-replacement treatment.[5]

While thyroid dysgenesis remains the most common cause of congenital hypothyroidism, the incidence of dyshormonogenesis has been increasing over the last few decades. Whereas dyshormonogenesis accounted for only 15% of congenital hypothyroidism diagnosed in the early days of newborn screening, 30–40% of infants diagnosed by current newborn screening strategies have a eutopic thyroid gland consistent with a form of dyshormonogenesis.

While the term dyshormonogenesis has classically referred to discrete defects in the cellular machinery of thyroid hormone synthesis leading to (often goitrous) congenital hypothyroidism, increasing recognition of the wide spectrum of severity of such defects makes it reasonable to define dyshormonogenesis as inadequate thyroid hormone production from a eutopic thyroid gland.[6]

To date, no reports have linked early postnatal treatment to improvements in goiter size, airway maintenance, or swallowing function. There are limited published descriptions of intra-amniotic administration of L-thyronine to treat fetal hypothyroidism and reduce or stabilize goiter size, but knowledge of its effect on goiter size and neonatal outcomes is sparse.Figueiredo et al followed a similar patient with perinatal goiter who was treated with intra-amniotic levothyroxine injections and started on immediate treatment postnatally, but measurements of goiter were not tracked. Case reports note that older children with large goiters despite appropriate treatment for hypothyroidism, as well as worsening goiter size upon stopping levothyroxine supplementation.

Surgical treatment of congenital goiter is rare and is utilized for extreme cases of aerodigestive compression that are unable to be conservatively managed. Thyroidectomy is also reserved for cases with concerning features of malignancy of the thyroid on imaging and is used in exceptional circumstances. Another surgical option includes tracheostomy for severe airway compression, failure to extubate safely, or severe tracheomalacia refractory to positive pressure. These options are discussed thoroughly with the entire care team before proceeding and fortunately, did not need to be explored in this patient.

We found that allowing time for the goiter to decrease in volume with thoughtful medical management alone allowed for safe extubation once there was reduced compression on the trachea and aerodigestive tract and a deferred need for surgical intervention. Over time, the goiter continued to shrink to a more cosmetically appealing and functional size with meticulous correction of hormone deficiency with levothyroxine supplementation as appreciated on serial ultrasonography. Our case supports the approach of expedient and aggressive medical management of congenital hypothyroidism with goiter to allow for significant volume reduction and reduction of compression on the aerodigestive tract to lessen the need for surgical intervention.[7]

Statements and Declarations

Permissions

We certify that the submission is original work and is not under review at any other publication.

Authors have seen and agree with the contents of the manuscript and there is no financial interest to report.

Figures, tables are not published elsewhere The authors have no conflicts of interest to declare.

Informed consent

Parent of the child has been explained in vernacular language the need of follow up scans and images taken for the case.

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