CASE REPORT

Tumour lysis in newborn: spontaneous or secondary to antenatal steroids?

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SUMMARY

Malignancies are rare in the early neonatal period. Common congenital tumours include malignant teratoma and neuroblastomas. Tumour lysis syndrome is a serious condition usually seen after commencement of chemotherapy for a malignancy. Rare case reports of spontaneous tumour lysis have been reported though not in the newborn period. We report here an instance of tumour lysis syndrome in a newborn with congenital rhabdoid tumour, where the cause was either spontaneous or related to antenatal steroid exposure.

BACKGROUND

Neonatal malignancies are rare. They account for less than 2% of childhood malignancies.^{1,2} Tumour lysis syndrome is a medical emergency requiring immediate treatment.³ It refers to a myriad of metabolic abnormalities—hypocalcaemia, hyperphosphataemia, hyperuricaemia, hyponatraemia, hyperkalaemia, elevated lactate dehydrogenase (LDH), high creatinine and urea. It is commonly seen after starting chemotherapy in tumours with high cell turnover. Rare case reports of spontaneous tumour lysis have been reported. It is often seen in solid tumours with high cell load like Burkitt lymphoma or acute leukaemia with high cell load.

CASE PRESENTATION

The baby described was born to a 27-year-old primigravida mother who was referred from a private obstetrician at 36 weeks with polyhydramnios and a neck mass detected in the fetus. Anomaly scan done elsewhere at 22 weeks was reportedly normal. Ultrasound done here showed polyhydramnios with an amniotic fluid index of 44 cm with a 7×6 cm left-sided neck mass. There was growth restriction with an expected fetal weight of 1800 g. The neck mass was well defined, solid, situated on anterolateral aspect of the neck on the left side with no retrosternal extension. Possibility of oesophageal compression by the mass was suspected and plan was to do an ex utero intrapartum treatment (EXIT) procedure and deliver the baby.⁴

Since the gestational age assessment was based on last menstrual period (LMP) rather than an early dating scan, the fetal weight of 1800 g either represented severe growth restriction or wrong dates. Hence, mother was given a course of antenatal steroids (betamethasone). Baby was delivered via an EXIT procedure⁴ with her airway secured by 7 min.

INVESTIGATIONS

MRI done after stabilisation showed a mass on the left side of the neck with rarefaction of the middle cranial fossa floor, and left carotid sheath was embedded within the tumour. It protruded into and compressed the nasopharynx and oropharynx. Histopathology and immunochemistry established a diagnosis of malignant rhabdoid tumour.⁵

From the time of admission, she had metabolic acidosis with elevated lactate and was started on inotropes. Screening echocardiography showed severe myocardial dysfunction with a thickened myocardium suggestive of hypertrophied cardiomyopathy. Baby had oliguria since admission. Since she had a malignant tumour along with persistent lactic acidosis and clinical features of acute kidney injury, a diagnosis of tumour lysis was considered, and she was evaluated for the same. She had hyponatraemia, hyperkalaemia, hyperphosphataemia, hypocalcaemia, hyperuricaemia and elevated LDH—all of which were consistent with tumour lysis syndrome (values in table 1).

DIFFERENTIAL DIAGNOSIS

Clinically, the considered differential diagnoses were teratoma and soft-tissue sarcoma.

Baby was ventilated on admission to the neonatal unit. She was noticed to have a neck mass (10×7 cm) located on the left side of the neck extending to the face superiorly. She also had a nodule in the right popliteal fossa.

She was on multiple inotropes—dopamine, dobutamine and vasopressin. Her lactic acidosis was not responding to medical treatment, and she developed oliguria and later became anuric by 60 hours of life. Her creatinine was high, 1.33 mg% on day 3.

She was given intravenous fluids, as well as a single dose of rasburicase and allopurinol. Aluminium and magnesium hydroxide were given for the hyperphosphataemia.

OUTCOME AND FOLLOW-UP

She had persistent lactic acidosis and her laboratory parameters worsened. Baby remained anuric from 60 hours of life. In view of possible intracranial extension, surgeons felt that surgical options were limited. Parents were explained in detail about options of peritoneal dialysis, chemotherapy and difficult surgical removal. In view of the poor prognosis, parents opted for palliation; supports were withdrawn and she passed away on day 4 of life.

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Table 1 Investigations

	Day 1	Day 2	Day 3
Sodium (mmol/L)	137	123	127
Potassium (mmol/L)	5.5	6.3	5.4
Calcium (mg/dL)	7.36	6.3	5.21
Phosphorus (mg/dL)		8.8	4.8
Uric acid (mg/dL)		14.8	17
LDH (U/L)			13 120
Creatinine (mg/dL)	0.89		1.33

LDH, lactate dehydrogenase.

DISCUSSION

Neonatal malignancies are rare. They account for less than 2% of childhood malignancies.¹ Commonly seen neonatal malignancies include neuroblastoma, teratoma, tumours with genetic predisposition like Wilms tumour, retinoblastoma and other solid tumours like central nervous system (CNS) tumours and soft-tissue sarcoma. Some malignancies are found in syndromic babies like congenital leukaemia in Down syndrome.²

Tumour lysis syndrome refers to metabolic abnormalities seen in tumours with high cell turnover. It is usually seen after starting chemotherapy, with the dead tumour cells releasing electrolytes, protein and nucleic acids.³ This will lead to hyponatraemia, hyperkalaemia, hypocalcaemia, hyperphosphataemia, hyperuricaemia and elevated LDH. Most of these are excreted by the kidneys and when there is an overload, it leads to kidney failure. It also leads to myocardial dysfunction and seizures.^{6,7}

Paediatric malignancies like acute lymphoid leukaemia, Burkitt lymphoma, neuroblastoma and central nervous tumours are known to cause tumour lysis after initiating chemotherapy.⁶

Cairo-Bishop criteria based on biochemical and clinical parameters is used to define tumour lysis syndrome. The biochemical parameters include uric acid, potassium, creatinine, calcium and phosphorus while clinical parameters include features of renal failure, seizures, cardiac arrhythmias and sudden death.⁸

Tumour lysis is a medical emergency requiring immediate treatment. Treatment options include hydration with intravenous fluids, xanthine oxidase inhibitors like allopurinol or recombinant uricolytic agents like rasburicase which decrease uric acid, specific measures for treating hyperkalaemia (potassium free fluids and diet, potassium binding resins, salbutamol inhalation, glucose insulin infusion and dialysis if refractory to medical treatment). Hyperphosphataemia is treated with phosphate binders like aluminium hydroxide and hydration with phosphorus free fluids. Phosphorus binds with calcium, leading to hypocalcaemia and metastatic calcifications. Peritoneal or haemodialysis is offered to those who are refractory to conventional hydration and medication. If left untreated, the metabolites accumulate in the body and cause renal failure; interfere with functioning of liver and CNS, cause myocardial dysfunction, arrhythmias and ultimately death.⁹

Spontaneous tumour lysis is a rare but known entity. It is often seen in solid tumours with high cell load like Burkitt lymphoma or acute leukaemia with high cell load.¹⁰ A retrospective study from Taiwan showed that 1.1% of acute renal failure in adults were as a result of spontaneous tumour lysis presenting with acute renal failure, elevated LDH and hyperuricaemia. These patients were later found to have malignancy.¹¹

Tumour lysis in newborn is rare. Neuroblastoma and acute leukaemia are the most common malignancies which can cause

tumour lysis. Spontaneous tumour lysis has not previously been documented in the newborn period. Treatment involves hydration, allopurinol, phosphate binders, treatment of hyperphosphataemia and dialysis in case of acute kidney injury. Rasburicase may be useful in neonates; however, the prognosis in neonates with tumour lysis is probably worse, especially in preterm due to immature renal functions and pre-existing tubulopathy that worsens the biochemical parameters.¹²

In the baby described, tumour lysis syndrome occurred spontaneously in the absence of chemotherapy. An alternate possibility was that the antenatal steroids given to the mother could have precipitated the tumour lysis syndrome. There are no proven cases where antenatal steroids caused tumour lysis in a fetus/newborn with a malignancy. In this instance, it seems a strong possibility, since tumour lysis manifested so soon after birth.

Learning points

- ▶ Spontaneous tumour lysis is a rare but known entity. However, it has not been described in newborns.
- ▶ It is possible that antenatal steroids given to the mother could have caused the early tumour lysis.
- ▶ This possibility should be taken into account when planning delivery for a preterm fetus with a strong antenatal possibility of a malignant tumour.
- ▶ Strong clinical suspicion would lead to an earlier diagnosis and management, leading to a better outcome.

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