alkalinization is of minimal therapeutic value, as the solubility of xanthine is only minimally enhanced at alkaline pH. Our patient showed excellent treatment response over a long follow up of nine years, which is in line with the short-term follow-up response reported in the literature [4,5].

To conclude, HX is a rare disorder of purine metabolism which should be suspected in children presenting with orange colored graveluria, hypouricemia, hypouricosuria and radiolucent renal stones. Molecular testing is essential for exact phenotyping, and should be pursued in all cases. Such children show excellent response to treatment with low purine diet and increased oral hydration, as exemplified in the case described.

Contributors: SK: collected and analyzed the data and drafted the manuscript; AG: conceptualized the case report, drafted and edited the manuscript; RS: collected and analyzed the data, drafted and edited the manuscript. RK: conceptualized the case report and edited the manuscript.
Funding: None; *Competing interest*: None stated.

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Developmental Delay with Intermittent Twisting of Neck

The hallmark of cerebral palsy (CP) is the presence of pyramidal or extra-pyramidal signs [1]. There are many disorders that can mimic CP [2]. One such mimicking condition is high cervical cord compression due to anomalies of the spinal cord [3].

A two-year-old boy, second of twins born of non-consanguineous marriage, was brought with inability to stand. He was delivered at eight months of gestation with a weight of 1.5 kg and no significant neonatal complications. His motor milestones were significantly delayed compared to his twin and spasticity was noticed from six months of age. The parents reported stiffness of his neck and limbs which was more on waking up, which would decrease within a few minutes. There were no seizures or regression of milestones. He was diagnosed to have mixed (spastic-dystonic) cerebral palsy. His language and social skills were age appropriate. At presentation, he was using two- word phrases and had attained daytime bowel and bladder control.

On examination, weight, height and head circumference were within normal limits. There were no obvious dysmorphic features. His upper segment to lower segment ratio was 0.92 suggestive of truncal shortening. His vision and hearing were normal. There was hypertonia in all the four limbs and brisk deep tendon reflexes. The plantar responses were extensor bilaterally. Examination of the other systems was unremarkable.

Lateral *X*-ray of the neck (***Fig.* 1a**) showed anterior dislocation of C1 vertebra. The pre-dentate space was widened and measured 13 mm. MRI did not show any

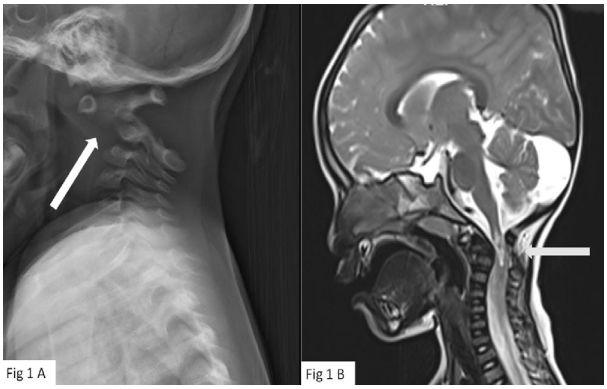

***Fig.* 1** (*a*) *X-ray Cervical spine lateral view flexed position showing widened pre-dentate space (white arrow); (b) MRI T2 weighted image sagittal section showing compression of the cord at C1 level (grey arrow).*

periventricular or basal ganglia changes. MRI of the cervical spine (***Fig.* 1b**) (confirmed atlanto-axial dislocation (AAD) causing compressive myelopathy at C1 level, without any other spinal malformations. Neurosurgeon prescribed neck collar, and advised follow-up for cervical spine stabilization. In view of the truncal shortening and AAD, he was also advised evaluation for skeletal dysplasia, but the parents deferred it to a later date.

Conditions which mimic CP should be considered – when there is absence of definite preceding perinatal insult; there is family history of developmental delay and spasticity; there is developmental regression or onset of new clinical signs of upper motor involvement; and when there is associated significant ataxia, muscle atrophy, or sensory loss [2]. Although neuroimaging is not essential for making a diagnosis of CP, MRI brain is abnormal in more than 80% of children with CP [4]. Current Western guidelines recommend MRI in children suspected to have CP [1]. The imaging not only uncovers the pathogenic patterns responsible for the CP but can also detect structural malformations of the brain and neuro-metabolic problems which resemble CP [4].

Despite significant perinatal risk factors, the intermittent abnormal neck stiffness warranted meticulous examination and evaluation [3], which revealed AAD can be idiopathic or due to traumatic, inflammatory or genetic disorders like Down syndrome, achondroplasia, cleidocranial dysplasia and Morquio syndrome [4]. Neurological manifestations of congenital AAD in children result from progressive compression of the cervicomedullary junction and present as progressive quadriplegia. Patients with myelopathy may go undiagnosed for a long period because of very slow progression of the disease process [3] and maybe mistakenly diagnosed as CP. Trauma or sudden movement can worsen symptoms in AAD. In the reported child, the increase in stiffness upon getting from sleep could possibly be due to the fact that while he was lying down, neck positioning could have caused an increase in stiffness. Poor cervical posture during sleep could cause increased biomecha-nical stresses on the structure of the cervical spine and could result in cervical pain and stiffness [5].

This case highlights compressive myelopathy as a differential for CP, and underscores the importance of a good history-taking in all patients, especially those labelled as cerebral palsy.

Contributors: Both authors were involved in clinical care, literature search and manuscript preparation.
Funding: None; *Competing interest*: None Stated.

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Congenital Chylothorax with Lymphatic Malformation and Successful Antenatal and Postnatal Management

Neonatal chylothorax is an abnormal accumulation of lymphatic fluid in the pleural space which can be either congenital or acquired. Nearly 90% of all *in utero* pleural effusions are chylothorax [1]. The estimated incidence of congenital chylothorax is 4 per lakh [2], with mortality ranging from 30-50%. [3]. We herein report a late preterm girl identified antenatally at 31 weeks of gestation with severe bilateral pleural effusion for which thoracoamniotic shunt was placed and subsequently diagnosed with congenital chylothorax after delivery.

A 37-year-old lady, G3P1A1L1 was admitted at $36^{5/7}$ weeks for delivery of hydropic fetus. Antenatal follow up had been uneventful till 31 weeks when ultrasonography showed hydropic changes in the fetus with bilateral pleural effusion and subcutaneous edema. A therapeutic fetal pleurocentesis was done with amniocentesis. Chromosomal analysis and microarray on amniotic fluid was negative. Mother had a negative indirect coomb's test, with serology negative for VDRL, and TORCH. Parvo