cause of gastric outlet obstruction in the 2- to 12-week-old age group. Pyloric stenosis leads to progressive and projectile vomiting.

It affects 2–3 per 1000 infants in Northern Europe but less common elsewhere in the world. Males (especially first borns) are affected approximately 4–6 times as often as females. The offspring of a mother, and, to a lesser extent the father who had pyloric stenosis are at higher risk for pyloric stenosis (Nelson Textbook of Paediatrics, 20th ed, Elsevier Saunders, 2016).

Impetus

- To stimulate discussion and further research on extreme electrolyte
- imbalances babies can tolerate.
- To demonstrate importance of slow correction of electrolyte imbalance over
- 72 hours.
- To consider Pyloric Stenosis as one of the differential diagnosis for
- constipation in early infancy.

Case Report We present a 9-week baby boy presenting with constipation and worsening vomiting for a week. He had very severe metabolic alkalosis (Ph-7.67), hypokalaemia (2.5 mmol/L) and hypochloroemia (60 mmol/L). He was amazingly still breathing spontaneously with no apnoea. His electrolyte imbalance was slowly reversed over 72 hrs and later he was transferred to a tertiary surgical care unit for corrective pyloromyotomy. He recovery was uneventful post surgery. We would like to share baby's severe electrolyte imbalances and stimulate research on how much extremes of metabolic alkalosis infants can tolerate.

Abdominal ultrasound Pylorus appears moderately thickened measuring to 2.1 cm in length (normally up to 1.5–1.7 cm). Pyloric transverse diameter measures to 1.7 cm (normally up to 1.3 cm) and pyloric muscle thickness was 8.9 mm

Learning Points

- Slow correction of electrolyte imbalance is critical in treatment of pyloric stenosis.
- Observe very closely for apnoea in babies with severe metabolic alkalosis
- Always think of Pyloric Stenosis in infants presenting with constipation. Pyloromyotomy is performed only after baby's metabolic alkalosis is normalized.

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P105 FLOPPY INFANT: FORGOTTEN OR NEGLECTED SYMPTOM OF CELIAC DISEASE

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10.1136/archdischild-2019-epa.460

Introduction Celiac disease (CD) or gluten-sensitive enteropathy is an autoimmune disease of the small intestine precipitated by the ingestion of gluten in genetically susceptible individuals. The prevalence is 1 of every 120 to 300 persons. The association between neurological symptoms and celiac

disease was first described by Cooke and Smith in 1966. Initiation of a gluten-free diet reverses the mucosal lesion and corrects malabsorption with symptomatic improvement.

Case report A 16-months old girl presented to the University children hospital with acute hypotonia and suspected Guillain-Barre syndrome. Two months before admission, she developed vomiting and watery diarrhea, which lasted for few days. Two weeks afterwards, acute symmetrical weakness of upper and lower limbs was occurred, so the patient was admitted to regional hospital. Her past medical history was irrelevant. She had normal growth and normal milestones development. She was able to walk before her first birthday. There was no history of honey and canned food use. She had negative family history of a similar condition. Her physical examination showed a floppy, irritable and afebrile child with body weight of 9900 g (25–50th percentile). Both upper and lower limbs were very weak with no reflexes. After her laboratory tests, abdominal ultrasound and brain CT, the diagnosis of GB was established and the treatment with IVIG was initiated. Her neurological symptoms improved slightly, but after five doses her neurological condition deteriorated with appearance of pretibial and dorsal pedal edema, and then she was sent to our hospital. On admission, she was 16-month old girl, body stature 82 cm (90th percentile), body weight 8450 g (10th percentile), BMI 13.7 kg/m². Subcutaneous fatty tissue was significantly reduced, she also had severe generalised edema. Her abdomen was distended without signs of hepatosplenomegaly. Neurological examination revealed severe general hypotonia of limbs with reduced reflexes, without axial weakness. Routine laboratory revealed normal CBC, hypoproteinemia, hypoalbuminemia with normal kidney and liver function. Inflammatory parameters, the biochemistry and cytology of cerebrospinal fluid, and MR of lumbosacral region were normal. The electro-neuromyography was not performed. Clinical aspect and laboratory results indicated celiac disease. Very high anti-endomysial and anti-transglutaminase antibodies and positive genetic test (HLA DQ2) confirmed clinical suspicion. No duodenal biopsy was performed. During investigation, the therapy with corticosteroids, albumin and furosemide were started. After celiac disease was diagnosed, gluten free diet (GFD), vitamin B complex and folic acid were started with gradually decreasing of corticosteroid therapy. Neurological improvement appeared after five days. After 3 weeks, she improved in her nutritional and psychological aspects.

Conclusion We should think about CD, since many patients have atypical disease.

P106 DIAGNOSIS OF ACUTE MYOSITIS VS DUCHENNE MUSCULAR DYSTROPHY BY TIMING OF BLOOD'S ENZYME PROFILE

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10.1136/archdischild-2019-epa.461

Introduction Elevated Alanine Aminotransferase (ALT) and Aspartate Aminotransferase (AST) enzymes would sway a doctor toward thinking there's a liver problem; a high CK with high ALT and AST levels suggests that something's going on in the muscle. A correct diagnosis may prevent extensive investigations or procedures. In boys one of the worst case scenarios is Duchenne Muscular Dystrophy (DMD) a severe