Table 1: Thyroid profile following accidental consumption of Levothyroxine

Time	Thyroxine (T4)*	Triiodothyronine (T3)	Thyroid Stimulating Hormone (TSH)
3 months prior to overdose	6.6	104	12.7
10 hours after overdose	30	154	0.49
46 hours after overdose	25.4	216	0.05
70 hours after overdose	19.3	158	0.01
118 hours after overdose	11	97	0.10

Normal rang: T4: 6.4-13.3µg/dl; T3: 94-241 ng/dl; TSH: Nl=0.7-64.0µu/ml

and hypertension. Annual report of the American Association of Poison Control Centers' National Poison Data System of 2008 revealed that out of 9,006 unique exposures to thyroid preparations only 3 cases had major adverse outcome and there were no deaths[4]. It has been documented that there is no correlation between the amount of levothyroxine ingested and the onset and severity of the symptoms as well as the serum concentrations of both triiodothyronine (T3) and thyroxine (T4)[2,5-7]. In study by Golighty et al one child with massive ingestion (13mg) never developed any complications whereas ingestion of 1.8mg developed tachycardia[5]. Serum T4 levels can help only in verifying the occurrence of the ingestion[5,6]. In many pediatric levothyroxine ingestion study series either they did not develop symptoms or showed only minimal symptoms[2,3-6]. In a study by Livotiz et al only four out of 78 children developed symptoms and T4 levels in three of these four children were 32.8, 30 and 26.4 µg/dl, respectively[2] which were similar to T4 levels in our child. Literature has supported a conservative management based on minimal symptoms[2,5,6]. T4 values cannot be used to guide treatment, and prompt clinical monitoring and evaluation is necessary[5]. Propranolol is used in the presence of features of toxicity[3,5]. Julio Pardo opines thyroxine overdose needs very close monitoring after gastrointestinal decontamination and conser-vative treatment like propranolol, prednisone, etc should be started as soon as the patient becomes symp-tomatic to avoid the development of a thyroid storm[7]. In children with overdosage of levothyroxine, there is production of reverse T3 which is inactive, thereby protecting from the toxicity and this pathway is enhanced by steroids supporting addition of steroids to the treatment regimen[3,8]. To conclude although pediatric levothyroxine overdose rarely leads to serious toxicity, any symptoms should be managed accordingly as they arise.

Key words: Levothyroxine; Hypertension; Propranolol; Dexamethasone; Tachycardia

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Gastric Perforation Associated with Congenital Diaphragmatic Hernia in a Neonate

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Neonatal congenital diaphragmatic hernia (CDH) is a complex anomaly, the clinical course of which depends on the timing and duration of herniation

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and the amount of viscera herniated. Acute gastrointestinal complications of neonatal CDH are mainly caused by the incarceration of the stomach and intestines[1], which are significantly reduced by prenatal diagnosis and appropriate neonatal treatment. Neonatal gastric perforation associated with CDH is an unusual surgical emergency, the symptoms of which may be quite nonspecific[1], and the radiographic findings may show pleural effusion and massive hydroperitoneum[2,3]. Rapid diagnosis and appropriate treatment are essential. We present a 4-day-old neonate with gastric perforation and CDH, who was treated successfully.

A 2.85 kg male infant was born at term by Cesarean section. The US at 25 weeks was normal. Milk feeding was accepted 2 hours after birth. He presented with recurrent non-bilious vomiting after meals during the first 3 days. Stools were passed during this period. On day 4 of life he had tachypnea and abdominal distension and greenish vomiting and his condition deteriorated rapidly. Then he was admitted to our hospital. On physical examination there were marked abdominal distension and decreased bowel sounds. The auscultation revealed decreased air entry on the lower left side of the chest. A nasogastric tube was passed and it drained bilious aspirate. A supine thoracoabdominal radiography showed an opacified left lower lung field resembling pleural effusion and the presence of gas in the left lower chest with shifting of the mediastinum to the right and a diffuse ground-glass opacity with paucity of bowel gas (Fig. 1). An erect thoracoabdominal radiography showed changes similar to the supine radiography except for the presence of gas fluid level in the left hemithorax (Fig. 1). Subsequent US showed left hydrothorax and an abundant volume of peritoneal fluid. Oral feeding stopped and he was intubated soon after admission. Prompt laparotomy was performed, which revealed the posterolateral diaphragmatic hernia, with a hernia orifice measuring 4×3 cm and consisting a large part of the stomach. A perforation, 4 cm in diameter, was located at the back wall of greater curve of the stomach. An abundant amount of yellow fluid was identified in the peritoneal cavity. The defect of the diaphragm was closed primarily and the perforation was repaired by two-layer closure. He was extubated on the seventh postoperative day and discharged from hospital

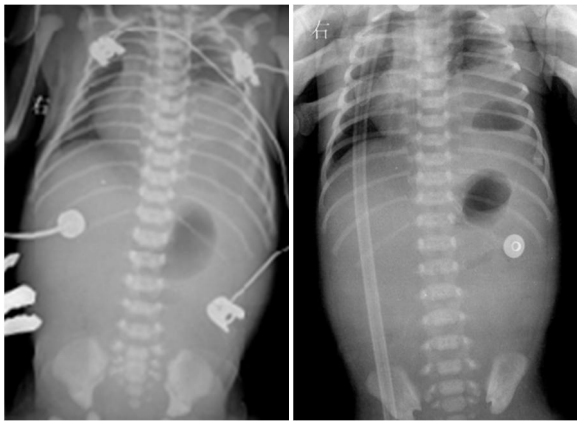

Fig. 1: Left: Supine radiography showing an opacified left lower lung field with a cyst and a diffuse ground-glass opacity with paucity of bowel gas; **Right:** Erect radiography showing gas fluid level in the left hemithorax.

35 days after admission. He recovered well after seven months of follow up.

There have been only a few case reports of neonatal CDH presenting with gastric perforation[1-3]. Two cases were discovered before birth[2,3]. Our case highlights the clinical course and radiography of neonatal gastric perforation associated with CDH, which are important in the diagnosis. There was one similar case observed after birth, symptoms of which were gastrointestinal rather than respiratory in origin[1]. In our case, the symptom of CDH before gastric perforation was recurrent non-bilious womiting rather than tachypnea, which is quite nonspecific, and the clinical course such as tachypnea, abdominal distension, greenish vomiting and rapid deterioration of the neonate came with gastric perforation. The radiography of neonatal CDH often shows an opacified hemithorax with mass effect and contralateral shift of the mediastinum. Bowel gas is lacking in the abdomen and can be identified in the chest[4]. In our case, the radiography showed an intrathoracic cyst rather than loops of bowel in the chest because the hernia consisted only a large part of the stomach. Pleural effusions and massive ascites indicated gastrointestinal complication associated with CDH, which is similar to some other cases[2,3]. Our case suggests that congenital diaphragmatic hernia should also be suspected in the neonate presenting with recurrent womiting. When massive ascites and pleural effusions are noted in association with CDH, gastrointestinal perforation should be considered. Early diagnosis

and fluid resuscitation and prompt surgical treatment are imperative.

Key words: Gastric Perforation; Neonate; Congenital Diaphragmatic Hernia

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Hypophosphatemic Rickets and its Dental Significance

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I read with interest Rabbani et al's paper entitled "Dental problems in hypophosphatemic rickets, a cross sectional study" in the fourth issue of Iran J Pediatr 2012[1]. Clinical oral manifestations of hypophosphatemic rickets often include premature tooth exfoliation, hypoplastic enamel and dental infections. Apical rarefaction, rickety bone trabeculation and absent or abnormal lamina dura are frequent radiographic findings[2].

Single or multiple abscesses relate to the enlarged pulp chamber and pulp horns reaching the dentino-enamel junction or even the external surface of the tooth[2, 3]. Defective enamel is also abraded or fractured easily[2]. Prophylactic pulpectomy and stainless steel crown placement have been recommended in affected patients[2,3].

Since premature tooth exfoliation is sometimes a feature in this inherited abnormality[2], the paper could also be paid to the investigation of this case. Also in the mentioned article, it is not clear under what circumstances the oral examination was done and what index has been used to assess the inflammation of the gingiva.

One of the common dental findings of the affected patients according to the above mentioned paper is taurodontism. Taurodontism is a dental anomaly characterized by a tendency for the body of the tooth and pulp chamber to enlarge at the expense of the root, leading to a elongated pulp chamber[2-4]. This developmental condition results from improper level of horizontal invagination of the Hertwig's epithelial root sheet[5] and seen in approximately 2.5- 3.5% of the population as an isolated trait[4].

Taurodontism is found with conditions such as X-chromosomal disorders, Down syndrome, otodental dysplasia, trichodento-osseous syndrome, Mohr syndrome, microcephalic dwarfism, amelogenesis imperfecta (type IV) and ectodermal dysplsia[2-5].

Diagnosis of taurodontism is based on radiographic examination and its clinical significance is disclosed when pulp therapy is indicated[2]. Since this anomaly frequently occurs multiplely[4], hence it was better to report the frequency of bilateral and multiple taurodontism of posterior teeth in the paper.

Key words: Hypophosphatemic Rickets; Taurodontism; Dental Problems

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