

# Transient Functional Intestinal Obstruction In A Newborn: The First Clinical Manifestation of Congenital Hypothyroidism

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## Abstract

Pseudo intestinal obstruction is an unusual initial presentation of congenital hypothyroidism. We describe a newborn boy infant who, on the third day of life, developed clinical and radiological evidence of intestinal obstruction. He was initially treated conservatively. Meanwhile, results of neonatal screening suggested congenital hypothyroidism which was confirmed to have permanent primary congenital hypothyroidism, due to thyroid hormone dysmorphogenesis. Replacement therapy with Levothyroxine led to marked improvement.

Keywords: Congenital hypothyroidism, Pseudo intestinal obstruction, Levothyroxine  
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## Introduction

Congenital hypothyroidism (CH) is a common endocrine disorder [1,2]. The introduction of neonatal screening programmes for CH in the 1970's is now regarded as a highly cost effective strategy to detect the disease before the clinical manifestations [2]. An unusually high incidence of various congenital anomalies has been reported in association with thyroid dysgenesis.3 Micro and Megacolon, and colonic motility impairment have been reported in association with hypothyroidism [3-6].

The purpose of this report is to indicate the fact that, CH may be a cause of transient functional intestinal obstruction in the early neonatal period. This may assist early diagnosis and appropriate management of such conditions.

## Case Report

This infant boy is the third child of a 26-year-old Yemeni mother. The parents were consanguineous and had no history of thyroid dysfunction nor cystic fibrosis. The pregnancy had been normal and no evidence of polyhydramnios. The mother had not received any medication containing iodine or antithyroid agent. The boy was born at 36 weeks of gestation by spontaneous vaginal delivery with Apgar Scores of 8 and 9 at 1 and 5 minutes, respectively. At birth the infant weighed 3.1 kg and was 50 cm long with unremarkable examination. He was started on breastmilk and reported to be slow feeder with frequent regurgitation. At day three, he was noticed to have abdominal distention with delayed passage of meconium. Bowel sounds were sluggish. Plain radiograph of the abdomen (Fig. 1) showed dilated loops of bowel with multiple fluid levels. Sepsis work-up was unremarkable with normal serum electrolytes. Contrast enema was normal with free passage of meconium. The patient was treated conservatively by intravenous fluids and nothing by mouth with open nasogastric tube drainage. Hirsch-prung's disease was unlikely diagnosis at this stage.

The hospital course was complicated by indirect hyperbilirubinaemia which was treated by phototherapy. At day seven, an abnormal cord screening for congenital hypothyroidism revealed thyroid stimulating hormone (TSH) of 413 mU/L (normal; <20) and thyroxine (T4) of 24 nmol/L (normal; 110-285) which was confirmed on repetition, (TSH 530

mU/L and T4 36 nmol/L). The epiphyses of the distal femora and proximal tibia were not visible on a radiograph. Sweat chloride test was unre-markable, and ruled out the possibility of cystic fibrosis. He was started on Levothyroxine 37.5 µg/day after which the boy was noted to suck well, became more active and stooling regularly.

At 3years of age, and after 4 weeks off thyroxine therapy, 99mTc pertechnetate thyroid scan showed normally located thyroid gland with increased uptake (Figure 2) suggesting thyroid hormone dysmorphogenesis. This was con-firmed by perchlorate discharge test (PDT) [7]. His growth has continued to follow the normal curve for age on thyroxine.



Figure 1. Radiograph of the abdomen showed dilated loops of bowel, with multiple fluid levels.

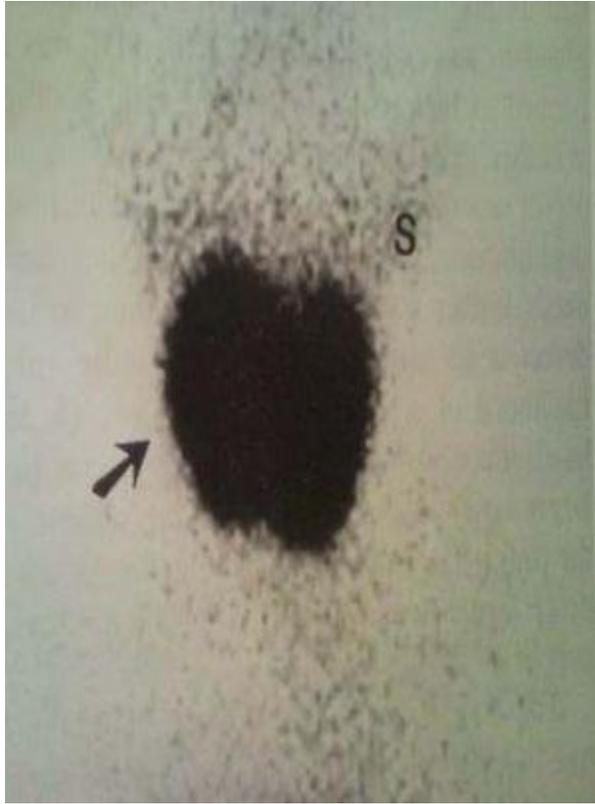


Figure 2: <sup>99m</sup>Tc pertechnetate thyroid scintigraphy re-vealing a eutopic enlarged gland (arrow) which is showing increased uptake, (s) salivary gland.

## Discussion

The diagnosis of permanent primary congenital hypothyroidism, in this newborn infant, was established on the bases of high TSH and low T4 values (both in the screen-ing and confirmation samples). The course of clinical and laboratory investigations during follow-up were another supportive evidence. Thyroid gland scintigraphy and perchlorate discharge test (PDT) indicated thyroid hormone dysharmonogenesis, inherited as an autosomal recessive disorder. Meconium ileus associated with cystic fibrosis, sepsis, and electrolyte abnormalities which could be excluded by the appropriate investigations [8-10].

Hypothyroidism is generally not considered to be manifest during the early neonatal period, however, the longer thyroid replacement therapy is delayed postnatally in infants with CH, the more clinical signs appear [11]. Feeding problems are certainly common, but are usually associated with multiple other findings. Intestinal obstruction is extremely rare and regarded as an unusual initial presentation [4]. Furthermore, paralytic ileus associated with longstanding lack of thyroxine, could be potentially disastrous if not recognized on time, as it simulates a surgical emergency [8-10,12]. Knowledge that intestinal obstruction may be an early sign of CH in the neonate is important and may facilitate early diagnosis, hence, avoiding devastating sequelae. The pathogenesis of such intestinal disorder is complex and poorly understood. Shafer et al [13] reported that gastro-intestinal transit in hypothyroid patients was slow. This also supported by Goto and associates [6]. Kowalewski and Kolodej [14] reported the decreased myoelectrical and mechanical activity of stomach and small intestine in hypothyroid dogs. Although, further studies still needed, improved motility after thyroxine replacement therapy indicates that it is the function rather than the morphology which was affected and supports further the role of adrenergic receptors and beta adrenergic responsiveness [6,15].

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