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Letters to the Editor

☐ UPGRADE OF CHILAIDITI SIGN TO SYNDROME: ARE THERE ANY PREDISPOSING FACTORS?

☐ To the Editor:

We have read with great interest the article, "Chilaiditi syndrome-what's air doing there?" by Hussain et al. regarding this uncommon (incidence 0.025–0.28%) radiological and clinical entity (1). The radiological sign is an asymptomatic visceral interposition, most often of the colon, between the diaphragm and the liver, referred to as the Chilaiditi sign, and the clinical presentation of symptoms is known as the Chilaiditi syndrome.

We believe that the title of the article mentioned above may be the most accurate description for this source of clinical and diagnostic excitement among young physicians that can be noticed surrounding the entire relevant literature. Chilaiditi syndrome was originally described by Demetrius Chilaiditis in 1910, a radiologist of Greek origin located in Vienna (2). Chilaiditi is a grammatical alteration of the original name in the Greek language.

The expression "thinking outside the box," regarding the famous puzzle of nine dots created by the early 20th century British mathematician Henry Ernest Dudeney, might be applied here, as we considered Chilaiditi syndrome as a quite interesting "box" in regard to diagnosis and pathophysiology. Possible preexisting, sometimes undiagnosed or modified through time, morbidities such as developmental or endocrinological disorders in patients with the radiological diagnosis of colonic subphrenic interposition may coexist and remain underestimated.

We recently dealt with a 10-year-old boy admitted due to febrile lower respiratory tract infection. His medical history included hypothyroidism on thyroxine replacement therapy, and learning difficulties due to which he was attending a special school. Moreover, he suffered from constipation that was well controlled with dietary adjustments and occasional administration of laxatives. His height was in the 15th percentile but his weight in the third percentile. Thoracic and abdominal radiography both in supine and upright positions revealed interposition of the colon between the diaphragm and the liver, known as the Chilaiditi sign (Figure 1).

According to the literature, the Chilaiditi sign becomes the Chilaiditi syndrome when there are obvious features from the gastrointestinal tract (such as abdominal pain, anorexia, nausea, vomiting, or constipation) due to strain or obstruction of the interposed visceral segment between the liver and the right hemidiaphragm (3,4). Other clinical features of Chilaiditi syndrome are associated with the respiratory system and include restlessness, respiratory distress, tachypnea, and sometimes severe chest pain; these features are often secondary to the presence of intestine at an abnormal location, which interferes with free movements of the diaphragm (3,4).

The comparison between the case reported by Hussain et al. and our own may trigger speculation on the conception of the criteria for the conversion from solely a sign to a syndrome, as shown below (1).

Though similar in presentation, there were certain differences observed between the patient presented by Hussain et al. and our patient (1). The first was a well-nourished patient with rather temporary symptoms. Beyond the common respiratory infection, abdominal

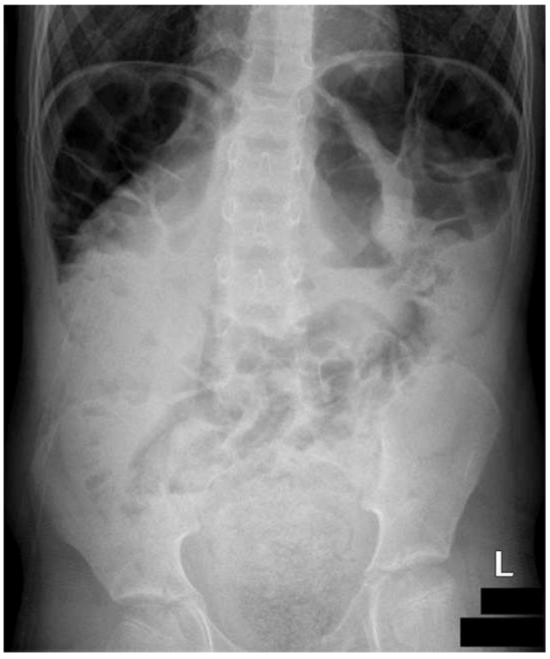

Figure 1. Intestine with morphological characteristics of colon interposed between the diaphragm and the liver in upright abdominal radiography.

distention was transitory, and there was a history free of further diseases. In our patient, the colonic compromise was chronic, with hypothyroidism and mental morbidity, while being in the third percentile for weight, indicating serious growth underdevelopment. These observations may highlight the role of respiratory involvement as a factor of comorbidity that may not always be secondary to intestinal pathology, but may be caused by other preexisting and underestimated factors, that is, the learning difficulties and hypothyroidism. Nonconformal and creative thinking "outside the box" may lead to the diagnosis or, if already known, to the reevaluation of developmental issues and associated medications as well as endocrine disorders that may affect both the motility of the bowel and the progression of a respiratory infection. These could therefore be regarded as the underlying predisposing or contributing factors of the presumed intestinal etiology of Chilaiditi syndrome.

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☐ THE CHICKEN OR THE EGG – CHILAIDITI AND CONSTIPATION

☐ To the Editor:

We would like to thank the editor for sharing with us the case titled "Upgrade of Chilaiditi sign to syndrome: are there any predisposing factors?" The authors present an interesting case of a young boy with a known case of hypothyroidism on thyroxine, presenting with constipation. The incidental finding of Chilaiditi syndrome in a patient with hypothyroidism raises an interesting conundrum: chronic constipation due to hypothyroidism that may have caused Chilaiditi syndrome vs. primary Chilaiditi sign that may have been followed with constipation complaints. Both situations are probable and have been recorded in the literature (1). Perhaps a second look at the patient's history may aid in distinguishing between the two.

In either case, as mentioned before, Chilaiditi syndrome has an important list of mostly acute differentials that need to be considered prior to attributing the symptoms solely to the syndrome. These include, but are not limited to, bowel obstruction, diaphragmatic hernia, or intussusception (1,2). In this patient, a thyroid panel would also be warranted in the full work-up, as is evident by the report submitted.

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☐ MINERALOCORTICOIDS AS A TREATMENT FOR SELECTED CASES OF REFRACTORY HYPERKALEMIA

☐ To the Editor:

Peacock et al. documented diabetes as a possible cause of hyperkalemia in 27% of the 203 subjects in the multicenter prospective observational study of hyperkalemia (1). This observation has important implications, given the fact that diabetic nephropathy is a risk factor for type 4 renal tubular acidosis, and associated hyporeninemic hypoaldosteronism, which may be unmasked by drugs such as trimethoprim, leading to severe hyperkalemia (2,3). In the report by Hussain and Chowdhury (2016) (3), of a 75-year-old woman with type 2 diabetes currently on trimethoprim, the administration of glucose/insulin infusion and intravenous sodium bicarbonate could only bring down her admission serum potassium