

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.

Xenophon Sinopidis, PHD, MD
Despoina Gkentzi, PHD, MD
Eirini Kostopoulou, PHD, MD

Ageliki Karatza, PHD, MD, Professor
Gabriel Dimitriou, PHD, MD, Professor
University of Patras, School of Medicine, Patras, Greece

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THE CHICKEN OR THE EGG – CHILAIIDITI 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.

Sara Hussain, MBBCH
Emergency Department
Rashid Hospital
Dubai Health Authority
Dubai, United Arab Emirates

Shabbir Hussain, MD
Iranian Hospital
Dubai, United Arab Emirates

Sahar Hussain, BPHARM, PHARMD
Dubai Pharmacy College
Dubai, United Arab Emirates

<http://dx.doi.org/10.1016/j.jemermed.2019.07.014>

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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