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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REFERENCES

1. Hussain S, Hussain S, Hussain S. Chilaiditi syndrome-what's air doing there? J Emerg Med 2018;55:e131–2.
2. Evrengül H, Yüksel S, Orpak S, Özhan B, Agladioglu K. Chilaiditi syndrome. J Pediatr 2016;173:260.
3. Moaven O, Hodin RA. Chilaiditi syndrome: a rare entity with important differential diagnoses. Gastroenterol Hepatol (NY) 2012;8:276–8.
4. Ghani S, Course CW, Bodla HP. From sign to syndrome: Chilaiditi. Arch Dis Child 2017;102:1117.

☐ 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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REFERENCES

1. Moaven O, Hodin RA. Chilaiditi syndrome: a rare entity with important differential diagnoses. Gastroenterol Hepatol (N Y) 2012;8:276–8.
2. Van Den Heede K, Van Slycke S. The Chilaiditi syndrome: another Greek tragedy? Case report and short review of literature. Acta Chir Belg 2014;114:352–4.

☐ 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

level of 8.8 mEq/L to levels in the range 6.0 to 7.0 mEq/L. Nevertheless, within 48 h of administration of fludrocortisone 100 μg bid by mouth, plasma potassium was restored to the normal range (3). Further investigations revealed a serum creatinine level of 191 μmol/L (2.16 mg/dL), and a random serum cortisol of 423 nmol/L (15.3 μg/dL) (3). Tacrolimus is another inducer of Type 4 renal tubular acidosis (RTA), and the resulting hyperkalemia may be refractory to intravenous furosemide, oral sodium polystyrene, intravenous sodium carbonate, and glucose-insulin infusion (4). In the report by Sahu et al. (2017) (4), however, serum potassium fell from 6.2 mmol/L (6.2 mEq/L) to the normal range 24 h after the administration of fludrocortisone 0.1 mg/day. With continued use of fludrocortisone, the serum potassium subsequently fell to 4.5 mmol/L (4.5 mEq/L) (4).

Heparin-induced hyperkalemia also has a Type 4 RTA-dependent etiology, and it also resolves after administration of oral fludrocortisone (5). The association of hyponatremia (116 mmol/L; 116 mEq/L) and hyperkalemia (7.2 mmol/L; 7.2 mEq/L) (the latter resistant to intravenous insulin/glucose infusion and to oral cation exchange resins) was attributable to the heparin analogue, pentosane, in a 65-year-old woman with a mistaken provisional diagnosis of adrenal crisis. Both her hyperkalemia and hyponatremia responded to the combination of intravenous hydrocortisone, 100 mg every 6 h, intravenous sodium bicarbonate infusion, and nebulized salbutamol, with the consequence that, 7 days later, her serum sodium had risen to 144 mmol/L (144 mEq/L), and serum potassium had fallen to 3.4 mmol/L (3.4 mEq/L). Her random pretreatment serum cortisol was > 1650 nmol/L (59.81 μg/dL), thereby negating the provisional diagnosis of adrenal crisis (6). Accordingly, there might be a role for the use of high-dose hydrocortisone to exploit the mineralocorticoid action of that agent for the purpose of bringing down the serum potassium level if the patient is too ill to take oral fludrocortisone (7).

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REFERENCES

1. Peacock WF, Rafique Z, Clark CL, et al. Real world evidence for treatment of hyperkalemia in the emergency department (REVEAL-ED): a multicentre, prospective, observational study. J Emerg Med 2018;55:741–50.
2. De Fronzo RA. Hyperkalemia and hyporeninemic hypoaldosteronism. Kidney Int 1980;17:118–34.
3. Hussain S, Chowdhury T. Severe hyperkalemia due to trimethoprim-induced type 4 renal tubular acidosis in diabetic nephropathy. Pract Diabetes 2016;33:228-a.
4. Sahu MK, Singh SP, Das A, et al. High blood tacrolimus and hyperkalemia in a heart transplant patient. Ann Card Anaesth 2017;20:270–1.
5. Brown G. Fludrocortisone for heparin-induced hyperkalemia. Can J Hosp Pharm 2011;64:463–4.
6. Jolobe OM. Hyperkalaemic paralysis. Age Ageing 2003;32:556–7.
7. Ramsahoye BH, Davies SV, el-Gaylani N, Sandeman D, Scanlon MF. The mineralocorticoid effects of high dose hydrocortisone. BMJ 1995;310:656–7.

☐ RESPONSE TO LETTER TO THE EDITOR

☐ To the Editor:

Thank you for the interesting comments by Dr. Jolobe in this issue of the Journal (1). Although his review of the use of mineralocorticoid treatment of hyperkalemia has a plausible mechanism of action, the limitation to refractory hyperkalemia excludes this strategy for the overwhelming majority of emergency department (ED) cases. Our study investigated the initial ED treatment of hyperkalemia, and had a primary endpoint defined as the serum potassium level after 4 h of treatment (2). Because the definition of refractory hyperkalemia requires a failure of standard therapy, it is outside the purview of our study to provide insight regarding treatment with mineralocorticoids. Further, we are not aware of a single prospective study using mineralocorticoid therapy for the ED treatment of hyperkalemia. We cannot support the use of mineralocorticoid therapy as an appropriate intervention until there are prospective safety and efficacy data in the ED environment.

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REFERENCES

1. Jolobe OM. Mineralocorticoids as a treatment for selected cases of refractory hyperkalemia. J Emerg Med.
2. Peacock WF, Rafique Z, Clark CL, et al. Real world evidence for treatment of hyperkalemia in the emergency department (REVEAL-ED). J Emerg Med 2018;55:741–50.

☐ MEDICO-LEGAL ANALYSIS OF TWO RECENT CASES OF BODY PACKING

☐ To the Editor:

We read with interest the two recent reports published in your journal, discussing the unusual case of a body packer concealing both a synthetic cannabinoid and cannabis,