described (Oster et al, 1976; Fang and Huang, 1996). They proposed three possible mechanisms for hypothyroidism-induced renal tubular acidosis:

1. Alteration of the cortical and/or medullary collecting tubules
2. Reduction in cortical sodium reabsorption leading to diminution of the degree of luminal negativity and producing a voltage-dependent defect which would lead to a concurrent impairment in potassium secretion and result in hyperkalaemia
3. An increase in membrane permeability, which allowed back-diffusion of hydrogen ions or possibly bicarbonate.

Deficiency of thyroid hormone might inhibit adenylate cyclase enzyme, thus resulting in a reduction in cortical sodium re-absorption producing a voltage-dependent defect of renal tubular acidosis. Interestingly in a study on rats with induced hypothyroidism, thyroid hormone was found to be responsible for altered expression of several renal acid base transporters (Mohebbi et al, 2007).

Conclusions

This case report highlighted the reversibility of renal tubular acidosis after treatment of the underlying autoimmune hypothyroidism and subsequent prevention of recurrence of hypokalaemic periodic paralysis in this patient. BJHM

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LEARNING POINTS

- Type 1 renal tubular acidosis leading to hypokalaemic periodic paralysis can be a very uncommon presentation of autoimmune hypothyroidism in young people.
- Persistence renal tubular acidosis despite treatment should prompt a search for a secondary cause such as hypothyroidism.
- Treatment of hypothyroidism can prevent the recurrence of renal tubular acidosis.
- The commonest association of secondary distal renal tubular acidosis is with Sjögren's syndrome.
- A defect in the hydrogen adenosine triphosphatase pump, reduced K+ absorption and membrane permeability are proposed mechanisms for hypothyroidism-induced renal tubular acidosis.

Stomal metastasis of colorectal cancer

A 74-year-old woman attended the accident and emergency department with clinical features of sub-acute large bowel obstruction. She had undergone Hartmann's procedure for obstructing sigmoid cancer 2 years previously.

There was stomal stenosis with an impassable stricture (*Figure 1*). Trucut biopsy of the lesion revealed adenocarcinoma of colonic origin. A staging computed tomography scan demonstrated metastatic disease at the stoma and in the liver and lungs (*Figure 2*).

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Metachronous tumours arising from the colonic mucosa often present as stomal recurrence and at least 10 cases have been described (Shibuya et al, 2002; Chintamani et al, 2007). This case is unique because of the location of the metastatic lesion and the time after resection of the primary (2 years).

There are no clear guidelines for the optimum curative therapy for stomal recurrence of colorectal cancer. The patient was offered stomal re-fashioning and palliative chemotherapy with radiation. BJHM

Figure 1. Macroscopic picture of stenosing stomal lesion.

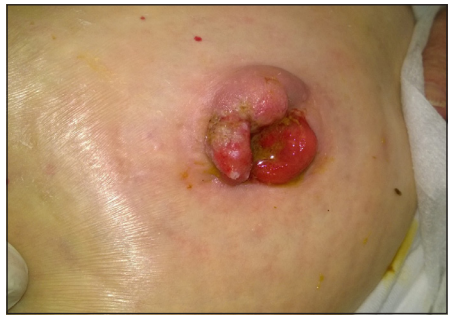
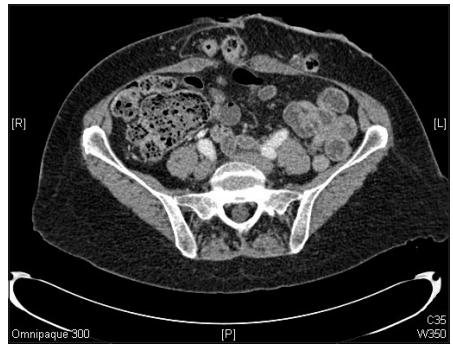


Figure 2. Computed tomography scan showing stoma stenosis.



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