CASE REPORT

A double conundrum: concurrent presentation of Hashimoto's thyroiditis and ulcerative colitis

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SUMMARY

We describe a case of a 31-year-old man who presented with a 3-day history of crampy abdominal pain, anorexia, malaise and diarrhoea of increasing frequency, with the passage of both mucus and haematochezia. The patient's biochemical investigations revealed hyponatraemia, hypothyroidism and elevated inflammatory markers. The patient underwent an ultrasound and fine-needle aspiration of the thyroid and was diagnosed as having Hashimoto's thyroiditis. He was started on thyroxine and fluid was restricted. He was also investigated for alternative causes of hyponatraemia. With improvement in his hyponatraemia, the patient underwent a colonoscopy with biopsies confirming a concurrent diagnosis of ulcerative colitis. He was started on mesalazine and prednisolone and discharged from hospital. He suffered a flare shortly after discharge and was readmitted to hospital. He was again discharged, on a higher dose of prednisolone, with outpatient follow-up at our hospital's gastroenterology clinic.

BACKGROUND

While an association between ulcerative colitis and Hashimoto's thyroiditis was first noted in 1962,¹ very few case reports have appeared in the literature.^{1–8} Furthermore, our case study is unique in that it looked at the simultaneous occurrence of Hashimoto's thyroiditis and ulcerative colitis in a patient with no other comorbidities, unlike previous case studies.

Previous case studies have looked at the relationship between Hashimoto's thyroiditis or ulcerative colitis along with other comorbidities (including autoimmune hepatitis, type 1 diabetes mellitus, Turner's syndrome and vitiligo), but not at Hashimoto's thyroiditis and ulcerative colitis concurrently as a pair.^{1–8}

Additionally, we wrote this case study up because the status of thyroid abnormalities (specifically Hashimoto's thyroiditis) as a form of extraintestinal manifestation of ulcerative colitis has neither been fully explored nor clarified.⁹ Hashimoto's thyroiditis also shares some immunological overlap in terms of pathogenesis (explored in discussion) and can induce symptoms including constipation, joint pains and muscle cramps, which can mask or worsen the symptoms and disease progression of ulcerative colitis.⁹ As such, further research to explore a possible relationship between the two diseases could be of potential therapeutic benefit to patients.

CASE PRESENTATION

A 31-year-old man presented with a 3-day history of crampy abdominal pain, anorexia, malaise and diarrhoea of increasing frequency with the passage of both mucus and haematochezia. Just prior to admission, he was reporting up to six loose motions a day with small amounts of rectal bleeding.

The patient also reported of urgency and tenesmus with regard to his bowel movements. He denied any loss of weight and reported some nausea but no vomiting. He had not been overseas for the past 6 years. He denied any recent sick contacts. He was an occasional smoker and social drinker. He admitted to intravenous drug use in his teenage years and reported previous marijuana use. He was not taking over the counter nor prescribed drug medications.

INVESTIGATIONS

In the emergency department, the patient was noted to be slightly febrile (37.7°C). He was also tachycardic with a heart rate of 110 bpm and hypotensive with a blood pressure of 90/50 mm Hg. He was started on slow intravenous fluid therapy and his heart rate fell to 70 bpm and blood pressure stabilised around 110/80 mm Hg.

On examination, the patient displayed generalised tenderness on palpation especially worse in the left iliac and lumbar region. Bowel sounds were normal and present. There was no evidence of organomegaly, peritonitis, guarding or rebound tenderness. His chest was clear and there were dual heart sounds with no murmurs. There were no maculopapular, nodular or discrete lesions that were obvious on the patient's legs or forearms (ie, erythema nodosum) and no ulcers in the patient's mouth. The patient displayed evidence of neither axillary nor cervical lymphadenopathy.

In the emergency department, the patient's full blood examination was unremarkable. His haemoglobin was noted to be 119 g/L (normal range 130–180), he had a white cell count of $6.6 \times 10^9/L$ (normal range 4.0–11.0) and his platelets were $205 \times 10^9/L$ (normal range 150–450). His urea electrolytes and creatinine revealed hyponatraemia with a sodium level of 123 mmol/L (normal range 135–145), potassium of 4.1 mmol/L (normal range 3.5–5.2), urea of 3.1 mmol/L (normal range 2.5–8) and estimated glomerular filtration rate of 87. The patient's C reactive protein was 33 and the erythrocyte sedimentation rate 26 (both elevated) on admission. His HIV and hepatitis serology came back negative over the next few days. The patient's

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liver function tests (LFTs) also showed some mild derangement with elevated aspartate aminotransferase, alanine aminotransferase and alkaline phosphatase of 92 U/L (normal <41), 82 U/L (normal <51) and 180 U/L, respectively (normal range 30–120).

In addition to our patient's hyponatraemia, his thyroid function tests (TFTs) were also consistent with hypothyroidism. His free T4 was noted to be 6.1 pmol/L (normal range 9.0–25.0), free T3 2.3 pmol/L (normal range 3.5–6.5) and a thyroid-stimulating hormone (TSH) 37.9 mIU/L (normal range 0.50–5.0).

On reassessment, a focused thyroid examination revealed the presence of a goitre in our patient. There was diffuse enlargement of the thyroid gland. The patient did not experience pain or tenderness on palpation and the thyroid gland was firm and rubbery. There was no tracheal deviation with percussion demonstrating no retrosternal extension of the goitre. There were no bruits heard on auscultation. The patient displayed neither balding nor loss of eyebrows. His heart rate was within normal limits and reflexes were brisk.

He was also tested for thyroid antibodies over the next few days. His serum demonstrated high titres of both thyroglobulin antibodies (489 U/mL, normal value <60 U/mL) and thyroid peroxidase antibodies (123 U/mL, normal value <60 U/mL), which are associated with autoimmune thyroid diseases such as Hashimoto's thyroiditis.¹⁰ The patient's TSH receptor antibody titre was within normal range (0.6 IU/L, normal range <1 IU/L). He subsequently underwent an ultrasound of the thyroid gland, which revealed the presence of hypoechoic nodules within coarse and hypervascular thyroid gland parenchyma on the left side (figure 1A, B).

Owing to the patient's family history of thyroid cancer, he also underwent a fine-needle aspiration of his thyroid gland, which showed histological changes consistent with a diagnosis of Hashimoto's thyroiditis (figure 1C).

He was also tested for his urinary sodium levels, serum and urine osmolality levels as part of his work up because the team was particularly concerned about his hyponatraemia. His serum osmolality was 243 mOsmol/kg (normal range 278–298) and urine osmolality was 355 mOsmol/kg. Furthermore, the patient also had low urinary sodium levels (under 20 mmol/L) indicating appropriate renal conservation of sodium.

The patient's early morning cortisol levels were also tested and noted to be 111 nmol/L (normal range 110–550).

DIFFERENTIAL DIAGNOSIS

One of the main differentials considered in this patient was that he was experiencing glucocorticoid insufficiency (figure 2). Cortisol is the main glucocorticoid produced by the adrenal glands.¹¹ The team was concerned about the patient's cortisol levels, which were on the lower end of normal. Adrenal insufficiency, especially Addison's disease (primary adrenal insufficiency), was one of the differentials that the team tried to rule out vigorously.

The patient underwent a repeat early morning cortisol level test and was examined for his adrenocorticotrophic hormone (ACTH) levels. While his ACTH was 10 pg/mL (normal <46 pg/mL), however, his repeat early morning cortisol level was 118 nmol/L (lower end of normal) and, as a result, the patient underwent a short synacthen test (ACTH stimulation test), which showed appropriate elevations in serum cortisol levels after 30 and 60 min postsynacthen.

Furthermore, owing to the patient's overseas migrant background and work up for Addison's disease, he also underwent a quantiFERON gold test for tuberculosis. Tuberculosis is estimated to be responsible for between 7–30% of Addison's disease cases worldwide.^{12 13} Fortunately, the quantiFERON gold test did not reveal latent tuberculosis in our patient. CT of the abdomen and pelvis revealed neither enlarged nor calcified adrenal glands, which are, if enlarged or calcified, would be

Figure 1 (A and B) Transverse and longitudinal ultrasound images of the patient's left thyroid gland, respectively, showing a 2 cm mass (white arrows) with surrounding hypoechoic and heterogeneous thyroid parenchyma. (C) Histopathology of the patient's thyroid gland showing gross distortion and loss of normal thyroid microscopic architecture. There is also diffuse reactive lymphoid infiltrate of the normal thyroid mucosa. The arrow is pointing to a germinal centre with lymphoid cell follicles.

Figure 2 Flow diagram showing the differential diagnosis of hyponatraemia based on the patient's fluid status, urine sodium levels and urine osmolality. SIADH, syndrome of inappropriate antidiuretic hormone.

associated with metastatic, infectious or haemorrhagic causes for the development of Addison's disease.^{14–17} Our patient's chest X-ray also was clear.

There was an additional concern that this patient might be hyponatraemic due to the syndrome of inappropriate antidiuretic hormone secretion. In particular, the treating team was concerned about the possibility of a germ cell tumour affecting the patient's thyroid hormones and sodium levels. However, the patient's malignancy screen was negative with normal β -1 the hormone human chorionic gonadotropin, β -2 microglobulin, lactate dehydrogenase and α -fetoprotein levels. Last but not least, he also underwent a brain CT as part of his hypothalamic—pituitary—adrenal axis work up, which did not show any abnormality (including that of the pituitary gland) and revealed a normal intracranial study.

With regard to gastrointestinal symptoms, the patient underwent an ultrasound of the abdomen to investigate his abnormal LFTs, which did not reveal any gallbladder pathology. He also had a CT of the abdomen and pelvis, which showed evidence of focal thickening of the mucosa of the hepatic flexure and adjacent transverse colon (figure 3). There was also some mild para-aortic and mesenteric lymphadenopathy noted on the CT images. He was also meant to undergo a colonoscopy, however, this was deferred due to concerns about the patient being placed under a general anaesthetic while hyponatraemic.

One of the main differentials considered for the patient's gastrointestinal symptoms was that he may have infective diarrhoea. He had a stool microscopy, culture and sensitivity test and a faecal PCR performed and was also tested for faecal calprotectin. His stools did not show any presence of common

Figure 3 CT of the abdomen and pelvis, showing evidence of focal thickening of the mucosa of the hepatic flexure and adjacent transverse colon.

infective agents such as *Cryptosporidium*, *Giardia*, *Salmonella*, *Shigella* or *Campylobacter*. Furthermore, there were neither ova, cysts nor parasites detected in the stools and the faecal PCR tested negative for common viral species such as *Norovirus*, *Rotavirus*, among others. The stools also tested negative for *Clostridium difficile* toxin gene.

The patient's faecal calprotectin showed evidence of active inflammation with a level of 452 µg/g (normal <50). His serum also showed normal levels of deamidated gliadin IgG and tissue transglutaminase IgA. These antibodies made the diagnosis of coeliac disease unlikely in our patient.^{18 19}

TREATMENT

The patient was started on 100 mcg of thyroxine daily to treat his Hashimoto's thyroiditis. He was initially put on a daily fluid restriction of 1.5 L, which was decreased to 750 mL when his hyponatraemia was not improving as desired. During the course of the patient's admission, his sodium levels eventually improved day by day and normalised after 4 days of thyroxine and fluid restriction. He eventually underwent a colonoscopy. The endoscope was inserted to the terminal ileum and showed mild-to-moderate ulcerative colitis from the rectum to the hepatic flexure, in keeping with pancolitis distribution (figure 4A, B). The terminal ileum was noted to be normal.

Biopsies were taken during the colonoscopy and showed evidence of inflammatory cells in the mucosa and submucosa, distortion of the normal crypt architecture, crypt abscesses and submucosal fibrosis (figure 4C).^{20 21} The pattern and distribution of endoscopic inflammation and the histological appearance of the biopsies were consistent with a diagnosis of ulcerative colitis.

OUTCOME AND FOLLOW-UP

The patient was discharged home on 100 mcg of thyroxine daily with a repeat TFT and outpatient clinic appointment made for 6 weeks postdischarge.

During the inpatient admission, the patient had also been started on mesalazine and prednisolone daily. He had been discharged home to be reviewed in 2 weeks at the hospital's outpatient gastroenterology clinic, however, he suffered a flare of his ulcerative colitis and was readmitted to hospital. On readmission, his prednisolone was titrated to an increased dose and his diarrhoea and inflammation settled down.

On his second discharge, the patient was again referred to the outpatient gastroenterology clinic for regular clinic appointments and surveillance colonoscopies for his ulcerative colitis.

DISCUSSION

Hashimoto's thyroiditis is an autoimmune mediated condition leading to hypothyroidism (ref 11, pp 984–90). While the aetiology of Hashimoto's thyroiditis has not been fully elucidated, environmental factors and genetic susceptibility are believed to play a key role in the pathogenesis (ref 11, pp 984–90).^{22 23} There are several mechanisms by which it has been suggested that Hashimoto's thyroiditis develops and it is believed to be a combination of both an antibody and a cell-mediated immune process.^{22 23}

Patients afflicted with Hashimoto's thyroiditis have elevated antibody titres to various thyroid antigens including thyroid peroxidase (TPO), thyroglobulin (Tg) and thyroid-stimulating hormone (TSH) receptor. Nearly all patients with Hashimoto's thyroiditis have high serum concentrations of antibodies against one or more of these antigens (mainly TPO and Tg antibodies). These antibodies to TPO, Tg and the TSH receptors cause thyroid cell damage and lead to a decrease in thyroid function.^{22 23}

Furthermore, in Hashimoto's thyroiditis, lymphocytic (B and T) cells infiltrate the parenchyma of the thyroid leading to gradual thyroid failure.^{24 25} B cells are activated in the thyroid gland and are able to secrete antibodies to the thyroid autonomously whereas T cells contribute to disease progression

Figure 4 (A and B) Endoscopy of our patient, showing mild-to-moderate ulcerative colitis from the rectum to the hepatic flexure, in keeping with a pancolitis distribution. There is superficial ulceration and loss of normal mucosal architecture. (A) Depicting ulcerative proctitis (inflammation of the anus and rectum) in our patient. (B) Similarly, the sigmoid colon was also affected. (C) Histopathology of biopsies from the patient's sigmoid colon showing inflammatory cells in the mucosa, distortion of the normal crypt architecture and crypt abscesses (arrow).

through the apoptosis of thyroid cells by activating cytotoxic T cells (Th1 function) and further thyroid antibody production (Th2 function).^{24–28}

The formal diagnosis of Hashimoto's thyroiditis in patients with clinical features of hypothyroidism requires the demonstration of high serum concentrations of antibodies against one or more thyroid antigens (10–15% of patients with Hashimoto's thyroiditis may be antibody negative), diffuse lymphocytic infiltration of the thyroid on histology and follicular destruction of the thyroid, which manifests as a reduced echogenicity on ultrasound.¹⁰

Ulcerative colitis is a form of inflammatory bowel disease characterised by a relapsing and remitting pattern of inflammation that is limited to the mucosal layer of the colon. While the aetiology and pathophysiology of ulcerative colitis is not fully understood, it is believed that immunological, genetic and environmental factors all play a part in the development of ulcerative colitis (ref 23, pp 272–3). Ulcerative colitis commonly only affects the rectum of affected individuals, however, it may extend proximally and affect part of the distal colon (left-sided colitis) or the entire colon (pancolitis) (ref 23, pp 272–3). Similarly to Hashimoto's thyroiditis, a T cell driven inflammatory process leads to the distortion of normal cell architecture. In ulcerative colitis, T cells cause inflammation in the colon mucosa leading to the classic histological findings of crypt distortion and abscesses.^{29–30}

Ulcerative colitis has numerous extraintestinal manifestations including dermatological, rheumatological, metabolic, ophthalmological, haematological and hepatobiliary complications, among others.⁹ While an association between ulcerative colitis and Hashimoto's thyroiditis was first noted in 1962, no definitive link has been demonstrated between the two diseases and, as such, Hashimoto's thyroiditis is not considered to be an extraintestinal manifestation of ulcerative colitis despite studies suggesting that these diseases share a common dysregulated immunological background.^{1–9–30–32} The prevalence of Hashimoto's thyroiditis has been estimated to be between 0.9% and 3.5% in patients with ulcerative colitis.^{32–34}

In summary, the previous literature has demonstrated that an imbalance of type 1T helper (Th1) and type 2T helper (Th2) cells leads to the development of both Hashimoto's thyroiditis and ulcerative colitis. A larger case series may be warranted

Learning points

- ▶ Ulcerative colitis is a relapsing and remitting inflammatory disorder of the colonic mucosa whereby a T cell driven inflammatory process leads to the distortion of normal cell architecture and inflammation in the colon mucosa.
- ▶ Hashimoto's thyroiditis is an autoimmune cause of primary hypothyroidism characterised by antibodies to thyroid antigens and lymphocytic infiltration of thyroid parenchyma.
- ▶ The pathogenesis of both diseases involves an imbalance of type 1T helper (Th1) and type 2T helper (Th2) cells leading to immune dysregulation.
- ▶ Hashimoto's thyroiditis is prevalent in 0.9–3.5% of patients with ulcerative colitis and can influence the treatment course and disease progression of ulcerative colitis.
- ▶ A larger case series may be warranted to demonstrate a definitive link between Hashimoto's thyroiditis and ulcerative colitis so as to ensure optimal patient management.

owing to the fact that abnormalities of the thyroid gland can induce gastrointestinal symptoms that may mask or worsen the symptoms and disease progression of ulcerative colitis.

Contributors GS and SB together wrote the case report. SB also assisted with the literature review and proposed the idea of publishing the case report. ET prepared and analysed the histology samples of the thyroid and sigmoid colon that were obtained from the patient.

Competing interests None declared.

Patient consent Obtained.

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Patient's perspective

- ▶ I consent to my medical information being used for the purpose of this research article. I was a patient at Northern Hospital for 10 days in total. At the time of my admission, I was quite scared and terrified about what was going on with my body. My wife and the rest of my family were also quite concerned about my blood loss and my safety and it was a difficult ordeal for us on the whole. I also have a 6-month year old son who I missed quite badly during my hospital admission and, hence, I might have pressured to be discharged early that led to my coming back to hospital again within a short period of my initial discharge.
- ▶ I wish for my ordeal to help other medical practitioners in their research so that I can make a difference to other patients and families who are in situations similar to mine. I am grateful for the care that I received from the medical and nursing staff at Northern Hospital.

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