

Case Report

An intriguing autopsy case of gangrene intestine

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INTRODUCTION

Hypothyroidism, or myxedema, is the clinical syndrome that results from decreased production of T4 and T3. Most patients have primary hypothyroidism. The etiology of adult primary hypothyroidism includes autoimmune hypothyroidism (Hashimoto's thyroiditis), post – ablation after radio-active iodine, thyroid surgery and drugs such as Amiodarone and Lithium. Manifestations are variable and proportionate to the degree and duration of thyroid hormone deficiency as well as age of onset. The presence of goiter is common in younger patients (Hashimoto's) but often absent in the elderly. Clinical features of hypothyroidism are insidious and often missed, particularly in the elderly.

Hashimoto's thyroiditis is one of the most common causes of hypothyroidism. Hypothyroidism is often associated with hyperlipidemia, which is a known risk factor for atherosclerosis. Atherosclerosis can cause ischemia, which in turn can lead to hemorrhagic infarction of intestine [1].

Hypothyroidism as a cause of hemorrhagic infarction of intestine is not described in the literature. We present a case of intestinal gangrene with previously undetected hypothyroidism

CASE HISTORY

A 35-year-old male presented with chief complaint of episodic colicky abdominal pain of 2 years duration, which was associated with vomiting and constipation, exertional dyspnoea of 6 months and swelling of feet of 2 months.

Routine investigations like peripheral blood examination showed normocytic, normochromic anemia with macrocytes with total WBC count of $8.8 \times 10^9/L$ with differentials of neutrophils 69%, lymphocytes 26%, monocytes 2%, eosinophils 3 % and thrombocytosis. Urine examination showed 1+ albumin and 2-3 pus cells per high power field. Stool for occult blood was negative. Liver function test and renal function tests were within normal limit. Biochemical analysis showed hyperkalemia.

Upper GI endoscopy had evidence of healed duodenal ulcer. Ultrasound abdomen was apparently normal. Chest radiograph was normal. Echocardiogram finding was suggestive of constrictive pericarditis / restrictive

cardiomyopathy. ECG findings revealed incomplete right bundle branch block and 0.5 mm ST depression and T inversion in V4- V6.

Patient had no past history of chest pain, diabetes or hypertension. On general examination the patient was well built and moderately nourished with mild pallor and bilateral pedal edema. Systemic examination of central nervous system and cardio-vascular system was within normal limits. Per abdomen finding did not reveal any organomegaly.

There was no obvious thyromegaly. However T3 was decreased and TSH level was elevated as proved by hormonal level of T3 – 0.5 nmol/L (1.3-3.0 nmol/L), T4 – 6.2µg/dL (5.0-14.0µg/dL), TSH – 5.3 µIU/ml (0.2-4.0µIU/ml). Lipid profile was not done due to short hospital stay.

HOSPITAL COURSE

Patient stayed in hospital for four days with chief complaint of progressive abdominal pain associated with distension and vomiting.

Per rectal examination revealed rectum loaded with faeces. Patient symptomatically improved with enema but the symptoms recurred. Subsequently he developed sudden onset of poor respiratory effort with bilateral extensive ronchi and severe metabolic acidosis.

Patient was intubated, put on ventilator and given inotropic support. Repeat Echocardiogram revealed dilated right atrium, right ventricle with pulmonary arterial hypertension. He developed refractory hypotension and later on had cardio respiratory arrest.

AUTOPSY FINDINGS

Complete body autopsy was performed and *in situ* examination revealed grossly dilated and discolored intestine (Figure – 1) with no evidence of free fluid in any of the body cavity.

The small intestine was markedly dilated and discolored. There was no adhesion or growth to substantiate sub-acute intestinal obstruction and histologically it showed patchy

hemorrhagic infarction (Figure – 2,3). However multiple sections from mesenteric vessels did not reveal any evidence of thrombus or atheroma.

Thyroid histologically showed features of Hashimoto’s thyroiditis in the form of follicular destruction by lymphocytes and presence of Hurthle cells. (Figure – 4) Heart revealed presence of left ventricular hypertrophy and microscopically perivascular myxoid change with coronaries and aorta revealing presence of Grade IV atherosclerotic change. There was no evidence of constrictive pericarditis / restrictive cardiomyopathy.

Grossly the brain showed normal gyral and sulcal pattern and the vessels forming the Circle of Wills were unremarkable. Coronal sections revealed normal grey and white matter distinction. Histological examination revealed features of hypoxia manifesting as loss of Purkinje cells with few showing bright eosinophilic cytoplasm in cerebellum.

Organs like lungs, liver, and kidney showed features of hypoxia. Multiple sections from other endocrine organs like pituitary, pancreas and adrenal did not reveal any abnormality.

DISCUSSION

Hashimoto’s thyroiditis is one of the most common cause of hypothyroidism .It is most commonly seen in females of 45-65 year age group. Hashimoto’s thyroiditis is an autoimmune thyroiditis where important mechanisms are CD8+ cytotoxic Tcell mediated cell death and anti thyroid antibodies[.2, 3].

This patient was a male with previously undetected hypothyroidism, which was proven biochemically as T3 was decreased and TSH level was elevated however T4 level was within normal limits. The probable sequence of events proposed in the present case is hypothyroidism leading to hyperlipidemia. The histological features are consistent with Hashimoto’s thyroiditis. Clinically also this fact was substantiated as the patient had constipation of long duration, distension of abdomen and edema.

The coronaries and aorta showed evidence of Grade IV atherosclerosis though we did not have biochemically proven values of hyperlipidemia.

Hypothyroidism is a known cause of hyperlipidemia, due to defect in LDL receptors.4 Necropsy data suggest that the hypocholesterolemia of hypothyroidism predisposes to coronary atherosclerosis only in the presence of hypertension; in normotensive hypothyroid patients the degree of coronary atherosclerosis appears to be similar to that in age- and sex- matched normotensive control subjects.[5]

However, in some studies, no association was found between hypertension and hypothyroidism,[6, 7 5] and this patient was normotensive.

The most striking finding in this case was gangrene of intestine, which was patchy in nature. This can be explained by probable mesenteric artery atherosclerosis. Though multiple sections from mesenteric vessels did not reveal any evidence of thrombus or atheroma.

The gangrene in turn can explain features of shock in organs like liver, lungs and kidney. There are studies showing correlation between coronary and mesenteric vessel atherosclerosis.[8]

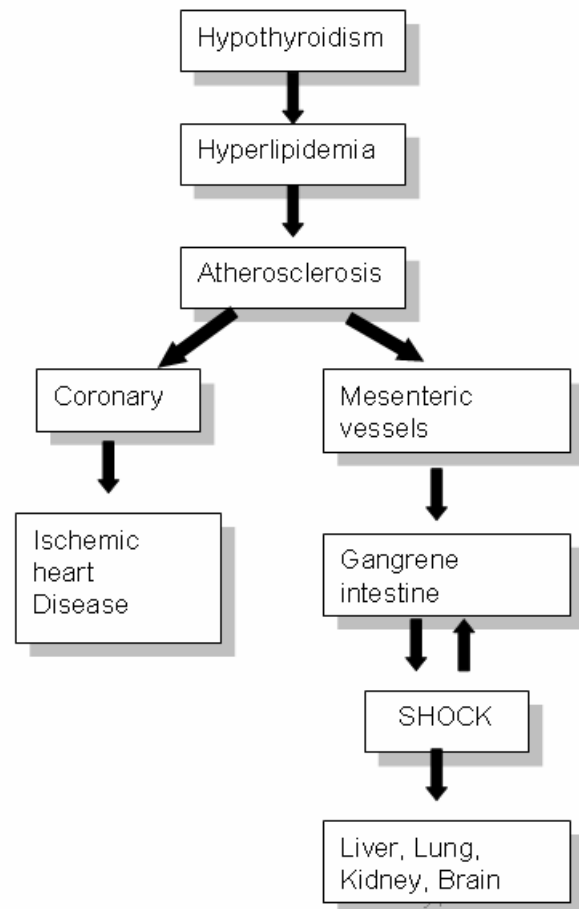


Figure – 1 Hypothyroidism causing intestinal gangrene, proposed hypothesis

Superior mesenteric vessels supply intestine by a unique blood supply known as collaterals or by parallel blood supply. So incidence of gangrene intestine is rare. However there are several causes of intestinal ischemia. In the present

case the important causes are shock and atherosclerosis. Others are acute vascular obstruction by thrombus, embolus, necrotising enterocolitis, vasculitides and vasculopathies, hypercoagulable state, drug effect, vascular compression, volvulus, celiac axis compression, infection associated with DIC or vasculitis, amyloidosis, diabetes and radiation damage [1].

Acute mesenteric ischemia is a cause of intestinal hemorrhagic infarction. It is mostly seen in elderly patients with severe cardio-vascular disease, [9] however this case was only 35 years old.

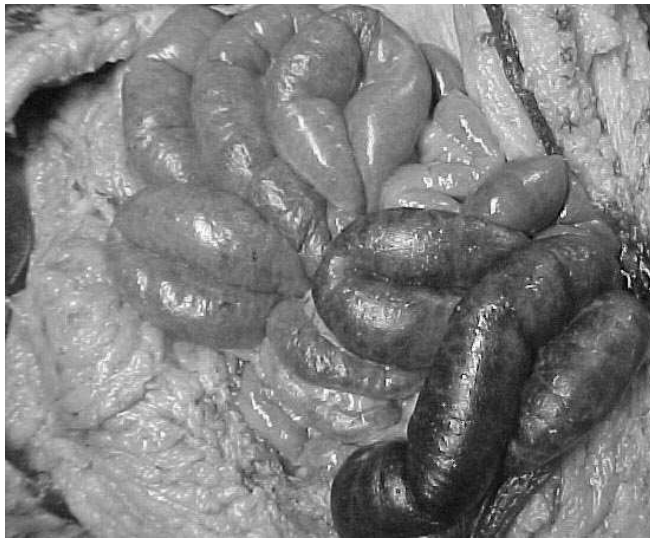


Figure – 2 Dilated and congested intestine

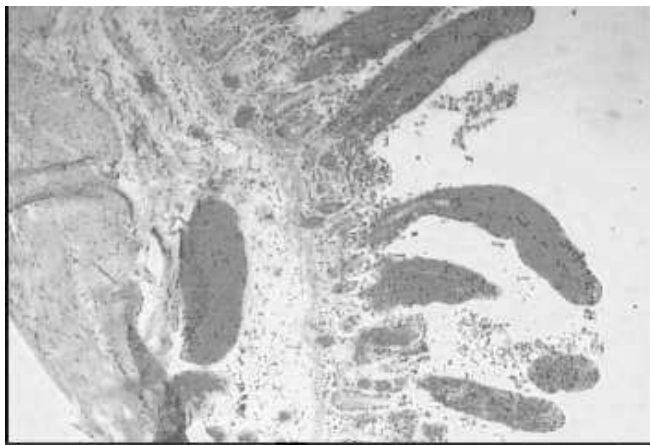


Figure – 3 Hemorrhagic infarction of intestine (H&E; X100)

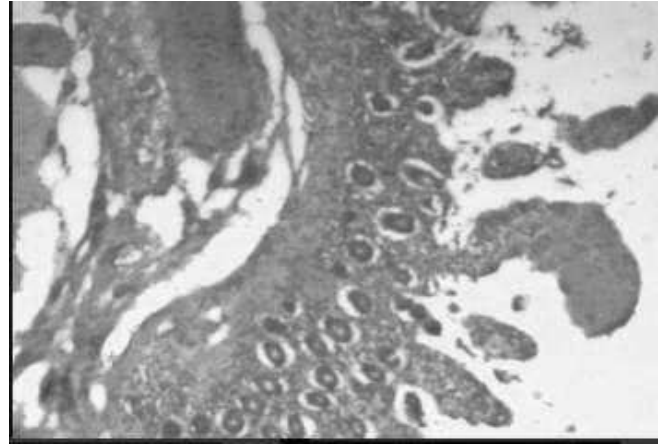


Figure – 4 Hemorrhagic infarction of intestine (H&E; X100)

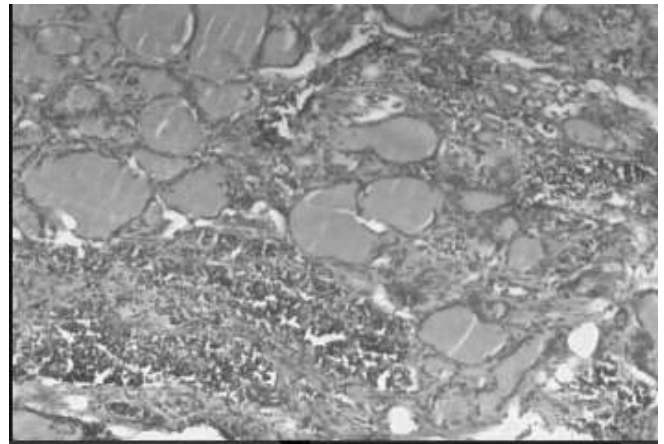


Figure – 5 Hashimoto's thyroiditis (H&E; X100)

CONCLUSION

Hypothyroidism as a cause of hemorrhagic infarction of intestine is not described in the literature. Hence hypothyroidism associated with hyperlipidemia can produce fatal intestinal gangrene as evidenced by the present case.

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