PERICARDIAL EFFUSION AND TAMPONADE COMPLICATING TREATED GRAVES’ THYROTOXOCOSIS

Editor,

Pericardial effusion has been reported in Graves disease. We report a case where symptoms of cardiac failure and the development of a symptomatic pericardial effusion were the predominant manifestations of thyrotoxicosis undergoing treatment.

Case Report: A 42 year old lady was admitted with a six-week history of increasing shortness of breath. She described pleuritic chest pain and had recently noticed ankle oedema. She had no symptoms typical of thyrotoxicosis. There was no relevant past medical history. One week before hospitalisation she was found to have markedly elevated thyroid function tests with Free T4 >100 pmol/L (NR 11-21) and TSH <0.02 mU/L (NR 0.3-4.5) and had commenced treatment with Carbimazole (40 mgs od) and Propanolol (80 mgs b.d.).

On admission she was dyspnoeic at rest with a sinus tachycardia. BP was 118/62. Bilateral pitting leg oedema, a small goitre and a right pleural effusion confirmed on chest X-Ray were noted. Biochemically there was continued evidence of hyperthyroidism - FT4 34.9 and TSH <0.02 with positive anti-thyroid peroxidase antibodies; 215 IU/ml (NR 0-135). C-reactive protein (CRP) was raised at 109. An echocardiogram revealed normal left ventricular function and evidence of a small localised pericardial effusion. There was no pericardial tamponade at this time. Following continued treatment with beta-blockers and an increased dose of Carbimazole (60 mgs o.d.) and diuretics the patient’s clinical condition improved and she was discharged.

Two weeks later she presented with further respiratory distress and was noted to have a raised venous pressure and a BP of 84/60. Thyroid function tests showed continued improvement. Repeat CXR revealed cardiomegaly (Figure 1). Repeat echocardiogram demonstrated a large pericardial effusion (Figure 2). The patient was transferred to the regional cardiology centre where 275mls of blood stained fluid was drained from the pericardial space with immediate improvement in dyspnoea and blood pressure. Biochemically the fluid was an exudate; culture and cytology were negative. Following this she remained well.

Conclusions: This case shows that pericardial effusion resulting in tamponade can develop in Graves thyrotoxicosis even during anti-thyroid treatment and with improving thyroid function tests.

A Medline search over the last twenty years uncovered two other reports of similar cases in English journals with a further report in a Japanese journal1-4. Authors from Oxford described a series of four patients all presenting with chest pain and effusions as the predominant manifestation of otherwise occult Graves’ thyrotoxicosis.2 The most recent case report from Israel3 describes a patient who developed a pericardial effusion despite treatment of hyperthyroidism. In this case tamponade did not develop and pericardiocentesis was not required. The aetiology of these complications is unclear although the blood stained nature of the pericardial effusion and the preceding pain and raised CRP suggests an inflammatory pericarditis. We therefore suggest it would be prudent to exclude Graves’ thyrotoxicosis in any patient presenting with an unexplained pericardial effusion despite the absence of classical symptoms of thyrotoxicosis. Furthermore, in a patient with active Graves’ disease, symptoms such as chest pain and dyspnoea need to be considered as potentially heralding the development of cardiac tamponade. While this complication is rare it may be rapidly fatal and thus go unrecognised and unreported.

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PATCHY SMALL BOWEL ISCHAEMIA SECONDARY TO SEPSIS

Editor,

Insufficient blood perfusion to the small bowel may result from arterial occlusion by embolus or thrombosis, thrombosis of the venous system, or non-occlusive processes such as vasospasm or low cardiac output. Patterns of segmental, skipped, or patchy small bowel ischaemia have been reported post abdominal aortic aneurysm repair and is thought to imply that microembolisation has played an important role. Microvascular thrombi in various organs can result from disseminated intravascular coagulation. We present a case of patchy small bowel ischaemia in a septic patient who had developed evidence of disseminated intravascular coagulation.

Case presentation: A 50-year-old man prescribed four weeks of diclofenac for a muscular strain presented with frank haematemesis secondary to an eroding gastric ulcer measuring 7 cm in diameter. A partial gastrectomy and Roux-en-Y reconstruction was performed and a massive blood transfusion of 25 units was required peri-operatively. The patient was referred to a tertiary care centre for intensive care where on day six post-operatively, he developed peritonitis as a result of a biliary leak from his duodenal stump. His blood tests at that time were suggestive of non-overt disseminated intravascular coagulation secondary to sepsis with an elevated D-dimer of 7.36 μg/ml (0.01-0.5 μg/ml). An emergency exploratory laparotomy was performed. This revealed a duodenal stump blowout and a 2 inch segment of proximal jejunum containing multiple less than 1 cm blisters in a triangular arrangement (figure 1). These were found to be necrotic areas and were resected. Histology of the specimen confirmed areas of transmural haemorrhagic ischaemic necrosis associated with a marked serosal exudate and incipient perforation. Thrombi were histologically identified within the omentum. Later in the post-operative period he developed bleeding from his duodenal stump for which he had successful radio-embolisation of the gastroduodenal artery. After a complicated postoperative period, he was discharged home. Although, patchy bowel ischaemia has been reported in past, this pattern in a mid jejunal small bowel segment containing multiple less than 1 cm areas of necrosis in a septic patient has not been reported to our knowledge.

Discussion: Patchy small bowel ischaemia has been noted in patients following abdominal aortic aneurysm repair. Some of these patients have been found to have widespread microembolisation or to have pathological evidence of microemboli. As a result it has been postulated that this pattern of ischaemia is a direct consequence of microembolisation. Sepsis almost invariably leads to haemostatic abnormalities. These range from the insignificant to severe disseminated intravascular coagulation. Compelling evidence from clinical and experimental studies suggests that disseminated intravascular coagulation is involved in the pathogenesis of microvascular dysfunction and that deposition of microvascular thrombi can occur. Given the above patient had an elevated D-dimer and evidence of thrombi on histology it is our feeling that the likely mechanism behind this unusual intra-operative finding was microembolisation. With sepsis related disseminated intravascular coagulation being the most likely cause.

Conclusions: We conclude that in septic patients, the bowel should be carefully examined as missing such pathology will have serious implications for critically ill surgical patients.

The authors have no conflict of interest

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CLINICAL EXPERIENCE WITH INTRAVENOUS IMMUNOGLOBULIN AND TNF-A INHIBITOR THERAPIES FOR RECURRENT PREGNANCY LOSS

Editor,

We report on a 22 year-old non-smoking nulligravida who presented with her husband for in vitro fertilisation (IVF). She was in good general health and had five prior unsuccessful IVF treatments, all with implantation failure. While her TSH and T4 were normal, a strongly positive (1:25,600) thyroid peroxidase antibody (ATA) titre was noted. Their sixth IVF cycle included IVIG infusion x3 as had been used in the immediately preceding cycle. However, etanercept (Enbrel®;