



## Case Report

## Myxoedema crisis as a cause for reversible complete heart block



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## ABSTRACT

An 85 year old female was brought in to hospital confused, dyspnoeic and pre-syncopal and was found to be bradycardic down to 19bpm and hypothermic down to 34.5 °C. She was found to be hypothyroid which required insertion of a temporary pacing system. Thyroid function resolved and she was no long pacing dependent.

This case highlights the sinister outcomes that can arise from thyroid dysfunction, which can be successfully treated if identified early enough.

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## 1. Case Report

An 85 year old lady presented to hospital by ambulance in extremis, bradycardic down to 19bpm, hypothermic down to 34.5 °C whilst maintaining adequate blood pressure. She was found by family to be confused, lightheaded and dyspnoeic, following a 3 day prodromal period of gastroenteritis.

Her background included coronary bypass grafting 3 years prior, hypertension, hyperlipidaemia, a partial thyroidectomy, type 2 diabetes mellitus requiring insulin.

She was resuscitated with atropine in the initial stages of her admission with poor effect. She was commenced on an isoprenaline infusion with improved heart rate and blood pressure response. Her electrocardiograms showed variation in patterns, suggesting a systemic cause for her bradyarrhythmias, with the presence of complete heart block with varying left (Fig. 1) and right bundle branch escape rhythms, together with spontaneous conversion into atrial flutter (Fig. 2).

Her initial blood work showed a metabolic acidosis, a lactate of 3.9mmol/L, an acute renal injury with hyperkalaemia of 6.3mmol/L and negative serial troponins. Her white cell count was  $12 \times 10^9$ , initial blood cultures returned negative, and urine cultures grew a pan-sensitive Escherichia Coli. Thyroid functions showed a thyroid stimulating hormone (TSH) of 12.7 mU/L, a T<sub>4</sub> of 11.8 pmol/L and T<sub>3</sub> of 2.7 pmol/L.

She was treated with intravenous Hydrocortisone and Levothyroxine, whilst being monitored in the intensive care unit. Despite treatment of her metabolic acidosis, hypothermia, electrolyte imbalance, and acute renal failure, her isoprenaline requirements increased significantly and she received a temporary-permanent pacing system on the 4<sup>th</sup> day of her admission (Fig. 3). Pacing was well tolerated and she had gradual resolution of her haemodynamic instability. An electrophysiological study was not conducted as she was too unstable to be able to withstand such an intervention, which was thought would not alter management. Her thyroid function improved with intravenous therapy and sinus rhythm was restored within 8 days (Fig. 4). The pacing system was eventually removed and she continued on thyroid hormone replacement therapy.

Follow up surpassed 30 days, out of which she showed complete resolution of her conduction issues not, not as much as first degree heart block being demonstrated on daily electrocardiograms.

## 2. Discussion

Bradycardia has previously been associated as one of the clinical manifestations of hypothyroidism, where patients have been noted to present in third degree atrio-ventricular (AV) block<sup>1,2</sup>. Hypothyroidism is an important diagnosis to make in the cardiology setting, as it has previously been described to contribute to, in addition to bradyarrhythmias, diastolic hypertension<sup>3,4</sup>, hypercholesterolaemia<sup>4</sup>, and further contributions to atherosclerotic plaque build, leading to higher rates of coronary artery disease<sup>1,5</sup>. These processes are potentially reversible with thyroid supplementation<sup>2</sup>.

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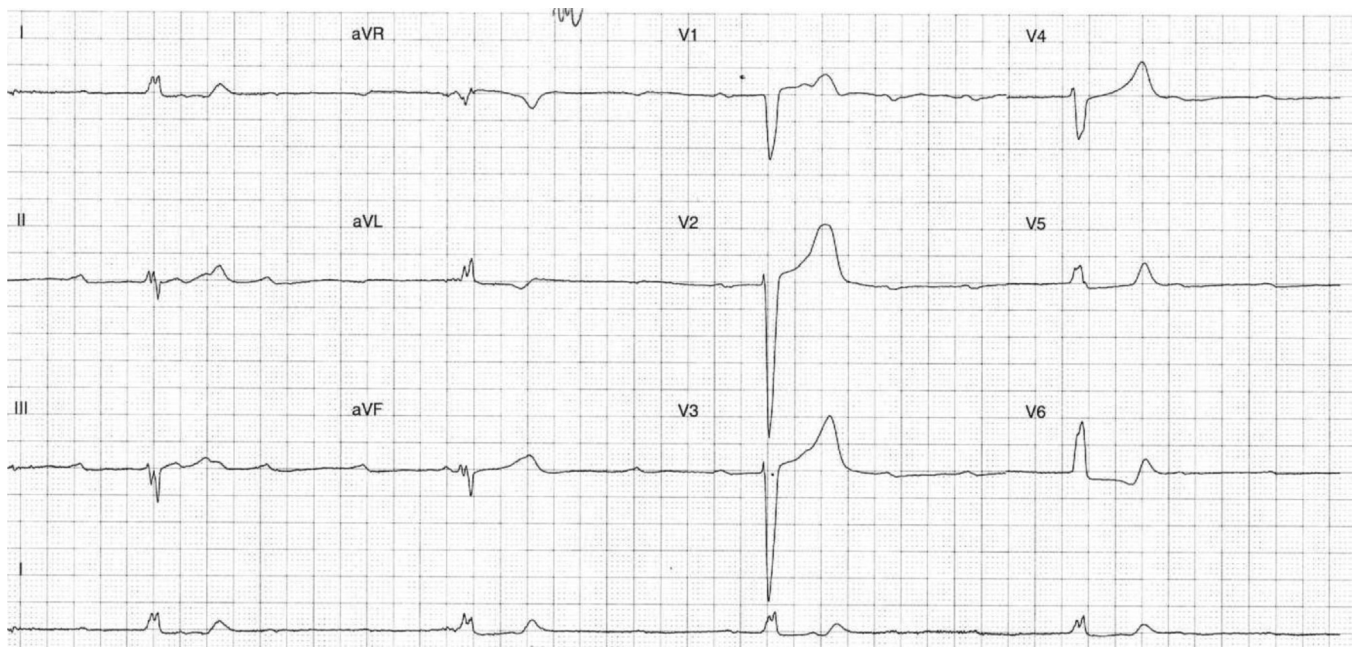


Fig. 1. Third Degree atrio-ventricular block with a left bundle branch escape rhythm.

Challenges in treating patients in this situation lie in the uncertainty in the clinical course following supplementation of thyroid hormone in the context of hypothyroidism. The overall resolution of the atrio-ventricular conduction block is not guaranteed<sup>1</sup>, and expected time course has not been published. Nevertheless, the expectation in this cohort of patients is that a pacemaker is not required<sup>6</sup> as systemic causes of bradycardia are often reversible.

A study conducted by Ozcan et.al, summarise previous reported cases of hypothyroidism in association with AV blocks which have identified cases of AV conduction delay in patients with severe as well as sub-clinical hypothyroidism<sup>1</sup>. Ozcan and colleagues themselves analysed their own cohort of patients with AV conduction delays, and identified hypothyroidism in 4% of their cases<sup>1</sup>. They found that 24% of the patients were found to have AV blocks due to the hypothyroidism itself, out of which they report

varying persistence of the AV block until resolution whilst on adequate supplementation<sup>1</sup>. Pacemaker implantation was still noted to be an adjunct to treatment in the majority of patients, despite treatment of the hypothyroidism, which questions whether hypothyroidism was the cause of the AV block, and if so, whether the block itself is reversible with hormone supplementation<sup>1</sup>.

This case highlights the importance of screening for reversible causes of bradycardia, such as hypothyroidism, which need to be excluded and treated prior to insertion of and commitment to a permanent pacing system. Pacemaker implantation may be required in patients found to have hypothyroidism despite adequate thyroid hormone replacement. Challenges lie in the uncertainty of the clinical course following thyroid hormone supplementation, and lack of guarantee for atrio-ventricular (AV) block resolution.

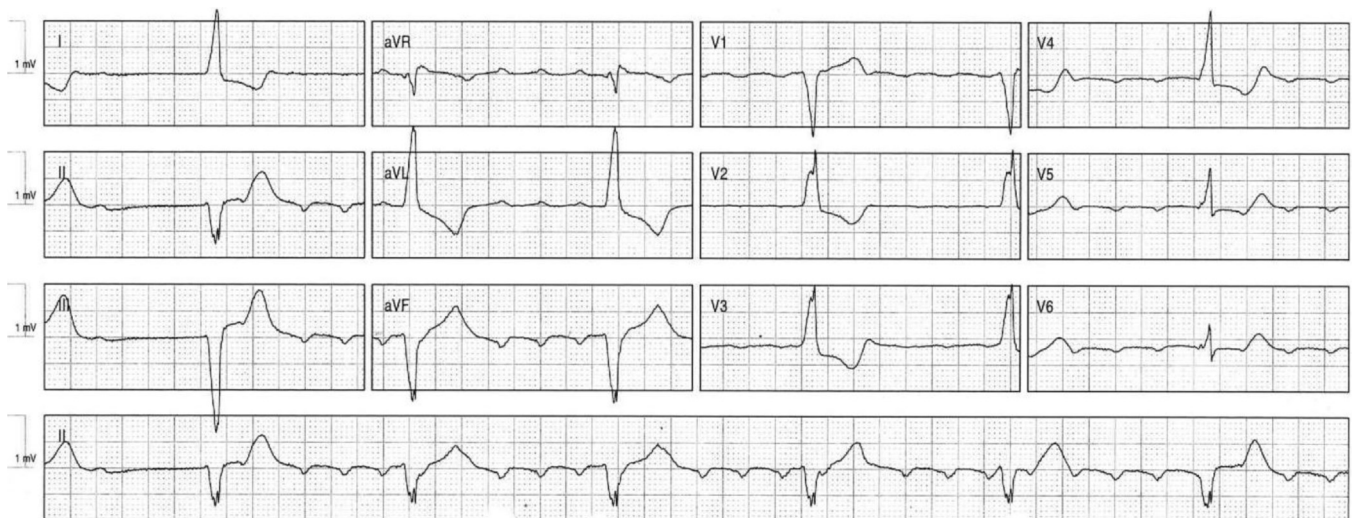


Fig. 2. Third degree atrio-ventricular conduction block converting into a 4:1 atrial flutter pattern with an left bundle branch escape rhythm.

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