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ABSTRACT

Thyroid storm (crisis) is uncommon but may be life threatening and is recognised by an exaggeration of the clinical features of thyrotoxicosis. Proximal myopathy is a well recognised presenting feature of Graves disease. Respiratory muscle weakness may also commonly occur in thyrotoxicosis but is often undiagnosed. We report a case of thyroid storm with rapid atrial fibrillation, severe agitation and extreme widespread muscle weakness. The respiratory muscles were so compromised that a respiratory arrest occurred. Ventilation was required for 7 weeks until full recovery occurred.

Keywords: Thyroid storm, thyrotoxicosis, ventilation, respiratory failure, myopathy

CASE HISTORY

A previously fit and well 58 year old shop worker presented as an emergency with a 3 month history of worsening breathlessness, agitation and palpitations. She was noted to have bilateral lid retraction and exopthalmos, (more so on the right than the left). She was pyrexial (38.5C), with a rapid irregular pulse of 200 beats per minute, confirmed on ECG as atrial fibrillation. She was markedly dyspnoeic at rest with arterial blood gases confirming type 1 respiratory failure (pO2 -7.9kPa, pCO2 -5.9kPa, O2 sats of 77% on air). Blood Pressure was 140/100mmHg. The clinical impression of a thyroid storm was confirmed by a markedly elevated free thyroxine (T4; 360pmol/L (ref range 9-19)) with an undetectable TSH. Later the TSH receptor stimulating antibodies (TRABS) were elevated at 11.2 u/L (normal range 0 – 1.5u/L) confirmed Graves disease. Urea and electrolytes were normal with no evidence of hypokalaemia. She was admitted initially to the coronary care unit for control of the atrial fibrillation with intravenous propranolol, digoxin, oral carbimazole and iodine aqueous solution.

Her condition deteriorated with the first hour, with hypoventilation, hypoxia (pO_2-6.9kPa, pCO_2-5.4kPa, pH-7.42; O2 stats 86% (on 70%O_2) and hypotension (BP- 80/40mmHg). An echocardiogram could not be interpreted because of the rapid ventricular rate. The patient was markedly hypotonic, unable to support herself upright in bed and she could not lift her head to look straight ahead. She was transferred to the intensive care unit for consideration of ventilation and on route suffered a respiratory arrest with a brief episode of asystole, which responded to a ‘thump’ on the sternum, returning a rhythm of fast atrial fibrillation. She was intubated within 3 minutes and ventilation started.

Seven weeks of ventilation, five weeks of which required inotropic support, followed by a five week period of rehabilitation led to a full recovery. Pulmonary function tests were not performed at presentation but this non-smoker had a FEV1/FVC of 78% predicted and a total lung capacity was 4.05 litres (predicted 4.58 litres) on recovery. The echocardiogram showed normal left ventricular function at this stage. The patient was discharged on carbimazole 20mg twice daily, propranolol 40mg twice daily with atrial fibrillation controlled at 80 beats per minute. Dalteparin was used for anticoagulation whilst ventilated and warfarin commenced prior to discharge. Some 18 months later, she was well, back at work and her euthyroid status maintained on carbimazole 20mg daily.

girdle muscles and muscles of respiration may be reduced by approx 20 and 40% respectively (6). Hypoventilation may occur, associated with diaphragmatic and intercostal muscle weakness, which is reversible with treatment (7,8). We can find no other reports in the literature of prolonged ventilation being necessary for full recovery from type 1 respiratory failure associated with thyroid storm. The exact pathophysiology is unclear, but reduction in muscle mass, dysynergia of the thoracic diaphragm and changes in adrenergic muscle stimulation may contribute.

The cardiovascular system is also directly and indirectly affected by thyrotoxicosis (9), with dysrhythmias (notably atrial fibrillation) or worsening of ischaemic heart disease symptoms. Acutely, thyrotoxicosis increases cardiac output through increased contractility and improved diastolic function. Over time however left ventricular hypertrophy and cardiac failure may occur. Acute cardiomyopathies have also been described in thyrotoxic states. These may present with sudden pulmonary oedema and are reversible upon treatment of thyrotoxicosis (10,11). Milder abnormalities of left ventricular function with no cardiac symptoms have been reported which took up to six weeks to reverse following the establishment of the euthyroid state (12). The patient described had rapid atrial fibrillation and the hypotension associated with the respiratory arrest may have been due to left ventricular dysfunction, but echocardiogram was inconclusive at diagnosis, and at six weeks showed normal findings.

In conclusion, cardiac symptoms e.g. palpitations are well recognised in thyrotoxic patients. Dyspneoa occurs in about 80% of such patients and is usually attributed to cardiac causes e.g. uncontrolled atrial fibrillation, but may be due to weakness of diaphragmatic and intercostal muscles. This should be kept in mind when assessing all thyrotoxic patients, not just those presenting in extremis with hypoventilation and type 1 respiratory failure as described. If ventilation is required in such exceptional circumstances, then it may have to be prolonged. In the case described it was seven weeks before the ventilation could be safely discontinued.

The patient has given full consent for her case details and images to be used in this publication.

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