Complete Heart Block Complicating Thyrotoxicosis

L. M. KERNOFF, F.C.P. (S.A.), PH.D., J. E. ROSSOUW, F.C.P. (S.A.) AND B. M. KENNELLY, M.R.C.P. EDIN., PH.D., Department of Medicine, Groote Schuur Hospital, Cape Town

SUMMARY

Two cases of complete heart block complicating thyrotoxicosis are recorded. In both cases intercurrent febrile illnesses were related to the development of this complication. It appears that while first and second degree atrioventricular block may occur not uncommonly in uncomplicated thyrotoxicosis, the presence of an additional factor which may further compromise atrioventricular conduction, is required for the development of complete heart block.


Whereas the association of first and second degree atrioventricular (A V) block with thyrotoxicosis is well recognized,1,6 complete (third degree) AV block is an infrequently reported association. Before 1937, 11 cases were reported in which the diagnosis of the thyrotoxicosis was based on clinical grounds, in some instances supported by basal metabolic measurements.5,6 Recently the first 2 cases of complete heart block in association with biochemically proved thyrotoxicosis were reported,7,8 and in this paper we document the case histories of a further 2 patients in whom this association was noted.

Case 1

A 40-year-old White female first presented in November 1968, with a 2-year history of irritability, tiredness, weakness, palpitations, heat intolerance, diarrhoea, and an 18-kg loss of weight, without loss of appetite. On examination, she was thin, and displayed a fine tremor of the hands. The thyroid gland was diffusely enlarged, with an asystolic bruit, and the eyes had a staring appearance.

The pulse was 120 beats per minute at rest and there was evidence of mild biventricular heart failure. A proximal myopathy was present. Urinalysis, blood count and electrocardiogram were all normal. Thyroid I31I uptake studies confirmed the thyrotoxic state, 80% of the dose being retained at 6 hours, and 85% at 24 hours (normal range: 6-hour 15 - 45%, 24-hour 20 - 50%). Red cell uptake of I131 was 34.4% (normal range: 12 - 20%). Treatment with propranolol 80 mg every 8 hours resulted in the abolition of all symptoms, and over the next year there was regression in the size of the gland, restoration of normal body weight and pulse rate. However, despite her euthyroid appearance clinically, she remained thyrotoxic on testing (6-hour thyroid I131 uptake 61%, 24-hour uptake 72%; red cell I131 uptake 34.6%). At this stage surgery was contemplated, but the patient refused on religious grounds. She discontinued her propranolol and was lost to follow-up.

One year later during a 'flu-like' illness that had lasted one week, she lost consciousness and when seen at home by her practitioner, was found in a shocked state with an unrecordable blood pressure, a pulse rate of approximately 6 beats per minute and Cheyne-Stokes respiration. She responded satisfactorily to atropine and isoprenaline and an ECG showed complete heart block (Fig. 1). Six hours later when admitted to hospital, she had reverted to sinus rhythm and was maintaining her blood pressure at 130/60 mmHg. The thyroid gland was barely palpable and signs of thyrotoxicosis were lacking. Her ECG showed first degree AV block and extensive T-wave inversion (Fig. 2(a)). Despite the euthyroid appearance, thyroid I131 uptake studies were markedly elevated: 6-hour 77%, 24-hour 81% and charcoal T: absorption (Charcoate) 0.6 (normal range: 0.8 - 12). Urinalysis and blood count were normal. No bacteria were cultured from throat swabs, and no viruses were isolated from throat swabs or faeces in either cell culture (monkey kidney and He La cells) or in suckling mice. Serum enzyme levels for the first three days after admission were SGOT 1640, 290, 118 mU/litre (normal range: 5 - 19) LDH 1080, 284, 300 mU/litre (normal range: 58 - 144) HBD 860, 204, 220 mU/litre (normal range: 53 - 127).

The remainder of her course was uneventful. Four days after admission the first degree AV block had disappeared, but left ventricular T-wave flattening persisted (Fig. 2(b)). Serum enzyme levels returned to normal at the end of a week. Definitive therapy was 6 mCi of

nodular goitre was present, her hands were warm and sweaty with a fine tremor, and there was marked generalized weakness. The pulse rate was 80 beats per minute, collapsing, and the blood pressure was 120/50 mmHg. Jugular venous pressure was 6 cm and the heart was moderately enlarged and hyperdynamic. The first sound varied in intensity and a third sound was present. A grade 2/6 ejection systolic murmur was heard maximally at the base, and in addition a scratchy systolic sound (Means-Lerman scratch), was audible over the pulmonary area and down the left sternal border. A few rhonchi and crepitations were heard at the lung bases. The liver was enlarged to 5 cm below the costal margin and a 2 cm splenomegaly was palpated.

Urinalysis was strongly positive for bilirubin and urobilinogen, with a trace of protein. The total white cell count was 12 200 with a normal differential count and the ESR was 105 mm in the first hour (Westergren). Chest X-ray showed biventricular cardiac enlargement and pulmonary venous congestion. Admission ECG (Fig. 3(a)) showed complete AV block with an atrial rate of 120 and ventricular rate of 80 per minute.

The total bilirubin was 3.9 mg/100 ml; conjugated bilirubin 2.6 mg/100 ml; alkaline phosphatase 128 mU/litre; SGOT 36 mU/litre. Quantitative serum protein electrophoresis was normal. Antistreptolysin O titre 1 250 Todd units. LE cells, hepatitis-associated antigen, antinuclear factor and latex fixation tests were all negative. Paul-Bunnell, Weil-Felix, Widal, brucella and leptospira agglutination tests were also negative. Blood cultures were negative on 14 occasions. Liver biopsy was normal, apart from a sparse infiltrate of lymphocytes and eosinophils in the portal tracts. Thyrotoxicosis was confirmed by a 6-hour thyroid 1 uptake of 78.2% and a 24-hour uptake of 74.2%. Resin T3 uptake was 53.2% (normal: 25 - 37%), and serum T3 was 13.1 μg/100 ml (normal: 5.4 - 13 μg/100 ml).

Treatment with penicillin and streptomycin was started 2 days after admission, and Lugol's iodine 4 days after admission, followed one week later by Neo-Mercazole and practolol. She became afebrile within 3 days of starting antibiotic therapy, but the jaundice took 2 weeks to clear. The ECG reverted to sinus rhythm with first degree AV block 3 days after admission (Fig. 3(b)), and normal AV conduction was established within 8 days (Fig. 3(c)). At follow-up one month later, she was still clinically thyrotoxic, the Means-Lerman scratch persisted, but the liver and spleen were no longer palpable and the ECG was normal.

**DISCUSSION**

From a review of the literature it appears that complete heart block in thyrotoxicosis is an extremely unusual occurrence, only 13 cases being described to date. The majority of these reported cases were complicated by the presence of factors in addition to the thyrotoxicosis which may slow AV conduction. Thus many acute infections may slow AV conduction and in 4 of 6 cases reported by Davis and Smith, the development of com-
Infiltration of the AV node and bundle of His by polymorphs and Gram-positive cocci was demonstrated in the single case which came to postmortem examination.

In case 1 the presence of associated infection, although likely, was not proved. While she gave a history suggestive of a 'flu-like' illness, in hospital she was apyrexial, had a normal blood count, and no pathogens were isolated from biological samples. It could be argued that the elevated enzyme levels indicated a myocarditis, but such levels are probably compatible with a prolonged period of hypoxia. Similarly there were no typical electrocardiographic changes of myocardial infarction.

In case 2 the nature of the febrile illness and jaundice was not fully elucidated. The high antistreptolysin O titre suggested either infectious hepatitis or streptococcal infection, with the response to antibiotics favouring the latter. Septicaemia appears unlikely in view of the numerous negative blood cultures. The sore throat, arthralgia, carditis, high ESR and ASO titre are compatible with a diagnosis of acute rheumatic fever, but the murmur and subsequent course were not typical. Part of the febrile illness may have been due to thyroid storm. Jaundice, though it occurs in 20% of patients with thyroid storm, is uncommon in thyrotoxicosis unless another factor such as cardiac failure, as in this instance, is present.

In both our cases, therefore, the development of complete heart block was intimately related to an intercurrent febrile illness, and in this respect bears similarity to most previous reports. Stern et al. have suggested that complete heart block in such cases may be due to the summation of delayed conduction induced by thyrotoxicosis and infection. Since only first and second degree AV block is generally associated with uncomplicated thyrotoxicosis, this view would appear to be correct. It is further supported by the fact that in both our cases the third degree block disappeared before the thyrotoxicosis was treated.

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REFERENCES