HYPERTHYROIDISM WITH GYNAECOMASTIA , GALACTORRHOEA AND PERIODIC PARALYSIS

E Muthusamy

ABSTRACT

A 32 year old male thyrotoxic presenting with gynaecomastia, galactorrhoea and later complicated with hypokalaemic periodic paralysis is presented. The gynaecomastia and galactorrhoea resolved with treatment. To the best of the author's knowledge this combination of association in one patient has not been reported previously.

Keywords : Hyperthyroidism, gynaecomastia, galactorrhoea, periodic paralysis.

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INTRODUCTION

Thyrotoxicosis with its many varied manifestations is well documented in the literature. Gynaecomastia, periodic paralysis, pretibial myxoedema, atrial fibrillation, thyroid acropachy, myopathy and pernicious anaemia have all been reported separately or in combinations in association with thyrotoxicosis. It is very rare to find a patient with many of the varied manifestations. Locally, gynaecomastia is a rare presentation in thyrotoxicosis but periodic paralysis is commoner (5%). Galactorrhoea in male thyrotoxics per se has not been reported locally.

CASE REPORT

A 37 year old male Malay clerk presented with one month history of bilateral breast enlargement and clear breast secretion. He also had tremors of the fingers, weight loss of 11 kg and increased appetite. He had no other features of thyrotoxicosis. There was neither sexual nor visual dysfunction. There was no symptom of raised intracranial pressure. He denied taking any drugs or alcohol. There was neither any relevant past history nor family history of thyroid disease or periodic paralysis. Both his parents and all his grandparents are of Malay descent.

Physical examination showed a thin, anxious man with tachycardia, fine finger tremors and hyper-reflexia. The thyroid gland was diffusely enlarged with bruit. There was mildly tender bilateral gynaecomastia (Fig 1) with clear nipple secretion. Other clinical signs of thyrotoxicosis were absent. There was no sign of chronic liver disease, pituitary tumour or hypogonadism.

Investigation confirmed thyrotoxicosis with a serum T4 of 260 nmol/L (normal range 64 - 154 nmol/L). His Hb, total white count, urine microscopy, blood urea, serum electrolytes, blood sugar, liver function test, serum calcium, serum prolactin levels (thrice), anti-microsomal antibodies, anti-thyroglobulin antibodies were all normal. X-ray of the skull, pituitary fossa and both hands were normal. Electrocardiograph showed sinus tachycardia.

Fig 1 – Bilateral Gynaecomastia

He was treated with propranolol and carbimazole. He achieved euthyroid state over the next two months. The galactorrhoea stopped within six weeks of treatment. He regained 7 kg of weight in six weeks. He continued to do well until nine months later when he developed sudden onset of weakness about one hour after lunch. He was unable to move all four limbs. Examination revealed signs of thyrotoxicosis, flaccid quadriplegia, slightly depressed reflexes, no sensory abnormalities, flexor plantar responses and no

Department of Medicine
District Hospital
14000 Bukit Mertajam
Penang, Malaysia

E Muthusamy, MBBS(S'pore), MRCP(UK)
Consultant Physician

bulbar weakness. ECG showed hypokalaemic changes and serum potassium was 2.3 mmol/L. He was immediately given intravenous potassium chloride infusion and was able to get up and walk after ten hours of treatment. His serum T4 was elevated at 252 nmol/L. His serum potassium normalised. He was discharged well with carbimazole and oral potassium supplements.

He was again readmitted one month later for sudden onset of weakness at around midnight. He had a vigorous game of badminton that evening. Examination revealed grade three weakness of all the limbs with reflexes and sensation preserved. His serum potassium was 2.2 mmol/L and electrocardiograph showed features of hypokalaemia. Intravenous potassium infusion was started and he recovered in twelve hours. His serum T4 was elevated at 240 nmol/L. His drug compliance was found to be poor. He was discharged well with normal serum potassium and was continued with carbimazole and potassium replacement. In view of his poor drug compliance surgery was suggested but he was not willing.

He continued to fluctuate between euthyroid and hyperthyroid states because of his poor compliance. We strongly suggested either surgery or radio-iodine therapy and he finally agreed to surgery.

His gynaecomastia regressed and the galactorrhoea did not recur. He did not have any more recurrence of hypokalaemic periodic paralysis since the last episode some twenty two months ago. He did not develop any other features of thyrotoxicosis other than those mentioned above during the thirty two months of follow up.

DISCUSSION

Von Basedow mentioned gynaecomastia as a manifestation of Graves disease in 1840[1]. In a Malaysian study of 180 patients over a 5 year period, there was no record of any patient presenting with gynaecomastia[2]. In a Singapore study of 51 male patients with thyrotoxicosis over a 3 year period, only one case was documented[3]. Gynaecomastia appears to be an uncommon and unusual manifestation of thyrotoxicosis. However, Locke found that 6 of his 31 male thyrotoxic patients had gynaecomastia[1]. One of his patients (not included in the above series) presented with mastalgia and gynaecomastia as the chief complaint.

In our patient, the main complaint was his enlarging breasts. He did not have any features to suggest an alternative cause for his gynaecomastia. Like most reported cases, the gynaecomastia appeared during the hyperthyroid state and regressed on return to the euthyroid state, strongly indicating that the gynaecomastia is due to the hyperthyroid state and not merely coincidental[3]. High serum levels of oestrogen and progesterone have been suggested to contribute to gynaecomastia in thyrotoxic men[4].

Galactorrhoea is broadly defined as any unilateral or bilateral non-purulent, non-bloody breast secretion ranging in appearance from clear to milky[5]. Galactorrhoea in female thyrotoxic patients, though rare, has been documented[5]. Gynaecomastia with nipple discharge is rare in male thyrotoxicosis[3]. Most cases of galactorrhoea in man are reported in patients with prolactin-secreting pituitary tumour[6]. However galactorrhoea and gynaecomastia in a hypothyroid man have been reported[7]. In our patient, there was no clinical feature of a prolactin producing tumour. The serum prolactin level was repeatedly normal. The galactorrhoea stopped with treatment of thyrotoxicosis and there was no recurrence since then. This further favours that the galactorrhoea is secondary to thyrotoxicosis.

Hyperthyroidism is not usually considered in the differential diagnosis of the cause of galactorrhoea.Popular textbooks in internal medicine do not mention thyrotoxicosis as a possible cause of galactorrhoea[8,9]. Hypokalaemic periodic paralysis is a well known complication of thyrotoxicosis. The incidence has been reported between 5% and 6% in Malaysia and Singapore[2,3]. Its occurrence is reported in some 2% of all thyrotoxic patients[10]. There is probably a 6% incidence of periodic paralysis in Chinese thyrotoxics, the condition being more common in males than females[11]. Certain racial groups (Chinese, Japanese and other Mongoloid races) have higher predilection. Its occurrence may be familial[12]. In over 82% of cases, thyrotoxicosis coincided with or preceded the periodic paralysis, and almost all patients had resolution of their paralytic episodes after successful treatment of their thyrotoxicosis[13]. In our patient, during each episode of periodic paralysis he had overt clinical and biochemical evidence of thyrotoxicosis.

In conclusion, a case of thyrotoxicosis complicated with gynaecomastia, galactorrhoea and periodic paralysis is presented. This combination of features, all in one patient, to the best of the author's knowledge has not been reported previously.

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