A CASE OF UNCONTROLLED THYROTOXICOSIS AND CONGESTIVE HEART FAILURE DUE TO GRAVES' DISEASE

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Background

Graves' disease is the commonest cause of thyrotoxicosis: globally, the incidence is 20 to 50 cases per 100, 000 persons annually. Famuyiwa and Bella reported an incidence of 8 cases per year at the University College Hospital, Ibadan about 3 decades ago. It is an autoimmune disease in which usually activating autoantibodies directed against thyrotropin receptor leads to excessive production of thyroid hormone and usually manifest clinically with features of hyperthyroidism. If left untreated myriad of complications, chief among which are cardiac related morbidity and mortality might supervene. Congestive heart failure due to Graves' disease is rare in blacks but has been well documented in previous studies from Nigeria. Kolawole and colleagues, Danbauchi and co-workers and Anakwue et al in different studies, done at different times, some years back in different parts of Nigeria, reported prevalence of 14, 8 and 16% respectively in hospital-based studies. Increase in the number of specialist physicians in field of endocrinology in Nigeria might account for not too frequent cases of congestive heart failure complicating Graves' disease seen these days in our practice. The contrary is actually true for this patient because despite being seen earlier in the course of the disease by an endocrinologist he developed congestive heart failure owning to his non-compliance with antithyroid drugs (ATDs) and his failure to attend the endocrine clinic for follow-up care, hence, the need to highlight this case.

Case report

A 31-year-old man presented to the endocrine clinic of LAUTECH Teaching Hospital, Ogbomoso, Nigeria 19 months ago with features suggestive of hyperthyroidism and a diagnosis of thyrotoxicosis secondary to Graves' disease was made. He was commenced on carbimazole and propranolol tablets. He has defaulted follow-up care until 14 months later when he presented at the emergency unit with a 10 month history of progressive bilateral swelling of the legs and 2 week history of worsening dyspnoea accompanied by other features of congestive heart failure; and tell tail signs of unabated thyrotoxicosis. He has not been compliant with ATD. On examination he was conscious but in respiratory distress and afebrile with temperature of 36.4C. Other positive findings include: chemosis, exolphthalmos, lid lag, goitre and bilateral pitting pedal oedema. Cardiovascular system examination revealed a pulse of 108/min regular and of normal volume. His blood pressure was 160/100mmHg, has distended neck veins, apex beat was not displaced and heart sounds were S1S2S3 and no murmurs. Significant findings in other systems included a right pleural effusion and ascites. He was stabilized on supplemental Oxygen, intravenous Lasix, carbimazole and propanolol were recommenced. Antimicrosomal antibody titre was 615.65 IU\ml (reference range is 0-35). His latest TFT results of 12/08/16: free T3-5.29 nmol/l (reference range is 1.13-3.1), free T4-77.61 nmol/l (reference range is 66-181), and TSH-0.005 UI/ml (reference range is 0.27-4.2). He is presently clinically stable and has been continued on carbimazole to await definitive treatment once euthyroid.

Discussion

Thyroid disorders of which Graves' disease is the commonest cause are the second most common presentation after diabetes mellitus in most endocrine clinics in Nigeria. Although Graves' disease is relatively uncommon in blacks compared with whites the clinical signs and symptoms accompanying Graves' disease is not in any way different in blacks as compared to their Caucasian counterparts as highlighted above in this patient. However, despite the challenges associated with accessing health care in our clime, this present patient presented early to a specialist endocrine clinic where a diagnosis of thyrotoxicosis secondary to Graves' disease was made and he was commenced on ATD. The culprit here is the lack of courage on the patient's part to continue with his treatment at that time. Existing data suggest some patients on antithyroid drugs go into remission after 1-2 years although some might relapse after the initial remission which is best achieved at the first treatment with ATD. Thus this present patient was recommenced on carbimazole because he presented in congestive heart failure. He was not a candidate for surgery at the time of presentation. Also the patient might not benefit from radioiodine therapy because of the size of the goitre. He is presently out of heart failure and our plan is to refer him to the surgeon for definitive treatment (thyroidectomy) when his serum TSH, free T4 and T3 normalizes and he will probably be on L-thyroxine replacement for life afterwards.

Table showing serial thyroid function test results

TEST	REFERENCE RANGE	UNIT	23/10/2014	5/1/2016	4/3/2016	1/4/16	10/6/16	12/8/16
Free T3	1.31-3.10	nmol/L		6.89	9.14	6.75		5.29
Free T4	66-181	nmol/L		232.80	236.0	188.6		77.61
TSH	0.270- 4.20	IU/ml	0.01(0.37-3.50)miu/L	0.133	0.183	0.140	0.005	0.005
Free T3	3.1 -6.8	pmol/L	24.9(3.8-6.0)				18.83	
Free T4	12-22	pmol/L	47.9(7.2 -16.4)				22.03	

Picture showing a moderately enlarged goitre

Conclusion

Graves' disease can be complicated by congestive heart failure which is reversible with the use of ATDs, and diuretics at the acute phase.

References

Graves' Disease Terry J. Smith, M.D., and Laszlo Hegedüs, M.D., D.M.Sc. N Engl J Med 2016;375:1552-65. DOI: 10.1056/NEJMra1510030

Congestive heart failure in subjects with thyrotoxicosis in a black community. Anakwue RC1, Onwubere BJ, Anisiuba BC, Ikeh VO, Mbah A, Ike SO.

2016 American Thyroid Association Guidelines for Diagnosis and Management of Hyperthyroidism and Other Causes of Thyrotoxicosis. Ross DS(1), Burch HB(2), Cooper DS(3), Greenlee MC(4), Laurberg P(5), Maia AL(6), Rivkees SA(7), Samuels M(8), Sosa JA(9), Stan MN(10), Walter MA(11).

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