Pipestem Carotids as a Marker of Vascular Insult in Graves' Disease

P. R. K. Bhargav · V. Amar ·
C. H. Venkata Pavan Kumar · K. B. Gayathri

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Abstract We present a case of Graves' disease with a image of plain radiograph of neck showing bilateral carotid calcifications and briefly discuss the pathophysiology of vascular calcification in Graves' disease.

Keywords Carotid calcifications · Graves' disease

A 21 year old lady presented with history of goiter, palpitations, weight loss and tremors for 3 years. She was on treatment with carbimazole 30 mg/day and propranolol 30 mg/day since 1 year, with partial relief of symptoms. She is still hyperthyroid with pulse rate of 110/minute, blood pressure of 130/90 mmHg and moist skin. Clinical examination revealed diffuse large goiter with bruit and bilateral exophthalmos. Cardiac evaluation with revealed no serious abnormality like cardiomyopathy, heart failure or arrhythmias. Plain X-ray neck showed linear calcification of both common carotid arteries (Fig. 1). Thyroid profile (thyroxine, triiodothyronine, thyrotrophin levels) were suggestive of persistent thyrotoxicosis. She was rapidly prepared with 45 mg/day of carbimazole, 120 mg/day of propranolol, 2 mg/day of dexamethasone for 6 days and operated with total thyroidectomy. Post operative period was uneventful except for transient hypocalcemia for 48 hours.

P. R. K. Bhargav (✉) · V. Amar · C. H. V. P. Kumar ·
K. B. Gayathri
Mamata Medical College and Hospital (MMC/MGH),
Khammam 507002 Andhra Pradesh, India
e-mail: kingbhargav@gmail.com

V. Amar
e-mail: surgeon_amy@yahoo.com

C. H. V. P. Kumar
e-mail: chvpavan@yahoo.co.in

K. B. Gayathri
e-mail: dr.gayathri.bhargav@gmail.com

Fig. 1 Plain antero–posterior X-ray of neck showing linear calcification of both common carotid arteries

Carotid and other vascular calcifications most frequently occurs in atherosclerosis, diabetes, old age, chronic renal failure and vasculitis. They are mostly implicated in atheroma plaque, disturbed mineral metabolism, vasculopathy and Monckeberg's medial calcification [1].

In the present case, none of the above factors were operating except for hyperdynamic circulation due to uncontrolled hyperthyroidism. Still, bilateral linear calcifications massive enough to be visible on plain radiograph occurred in a young lady with hyperthyroidism of 3 years duration, in the absence of hyperlipidemia and hypertension. Though exact cause is intriguing, past studies show that elevated thyroid hormone levels can cause vascular smooth muscle calcification by direct triggering of Gla matrix protein related gene [2]. Few studies show increased vascular injury marker titers and arterial wall

stiffness of carotid arteries in Graves' disease [3]. Volzke H et al. showed increased average carotid intimal–medial thickness (IMT) in hyperthyroid compared to euthyroid subjects [4]. Dorr M et al. reported increased carotid artery plaque burden associated with decreased serum TSH levels [5]. Also, vascular calcifications are reported in skin biopsy specimen of hyperthyroid patients [6]. We conclude by proposing that bilateral pipestem carotid calcification acts as a marker of peripheral vascular insult in Graves' disease, though it's a rare finding.

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