

Pituitary apoplexy presenting as myocardial infarction

Vishal Gupta, Sudarshan Patil¹, Dhiren Raval¹, Pratik Gopani²

Departments of Endocrinology, ¹Resident Doctor in Endocrinology, and ²Medicine, Jaslok Hospital and Research Centre, Mumbai, India

ABSTRACT

We describe a male patient who presented with sudden onset severe headache and right sided ptosis that was diagnosed to be secondary to pituitary apoplexy on the background of diabetes mellitus. This was complicated by left ventricular failure and acute coronary syndrome. The case highlights the importance of considering hypocortisolism/hypopituitarism as an important and rare precipitant of an acute coronary event as occurred in the case.

Key words: Acute coronary syndrome, myocardial infarction, pituitary adenoma, pituitary apoplexy, pituitary infarction

INTRODUCTION

Panhypopituitarism has been shown to predispose to cardiovascular disease (CHD). Potential incriminators are growth hormone, thyroid hormone, and gonadal hormone deficiency. Growth hormone deficiency has been associated with endothelial dysfunction [as evidenced by higher levels of plasminogen activator inhibitor-I levels (pro-coagulant), loss of circulating CD34+ cells], increased total cholesterol, low-density lipoprotein (LDL) cholesterol, increased body fat (truncal and waist-hip ratio), decreased lean body mass and increased risk of hypertension.^[1,2] Both central hypothyroidism and hypogonadism has also been associated with increased (serum total and LDL cholesterol, homocysteine, (C-reactive protein) CRP) and impaired endothelial-dependent vasodilatation and insulin resistance.^[3-5] Type 2 diabetes mellitus (T2DM) is “CHD equivalent”. Haffner *et al.* showed that the incidence of fatal and nonfatal myocardial infarction to be 3.5/100 person-years

in nondiabetics as compared with 20.2/100 person-years in patients with T2DM.^[6,7]

A 62-year-old male patient coming from the interiors of Maharashtra (Kolhapur), India, a nonsmoker, nonethanolic, on the background history of diabetes mellitus since the past 10 years, presented with sudden onset severe headache, right sided ptosis, for which he got admitted in a local hospital. Imaging of the brain showed pituitary macroadenoma. Within a few hours of admission he developed features suggestive of acute left ventricular failure secondary to acute coronary syndrome (non-ST-elevation myocardial infarction associated with elevated serum troponin T, regional wall abnormalities of mid and basal septum and apical and lateral wall of left ventricle on 2D-echocardiography. The ejection fraction was reduced to 35%). He discharged himself and was referred to our endocrine team by the cardiologist.

On examination his observations read as follows: Blood pressure 120/80 mmHg in right arm without any postural fall, pulse 84/bpm, elevated jugular venous pressure. His neurology suggested pupillary involving right-sided 3rd cranial nerve palsy. A visual perimetry showed binasal arcuate visual field loss. He showed signs of compensated left ventricular failure.

Baseline biochemical tests were normal except for an elevated creatine phosphokinase-MB 115 (4-36 IU/L) and troponin-T 21 mcg/L (0-0.1).

Access this article online

Quick Response Code:



Website:
www.ijem.in

DOI:
10.4103/2230-8210.129119

Corresponding Author: Dr. Vishal Gupta, Jaslok Hospital and Research Centre, 15, Dr. Deshmukh Marg, Pedder Road, Mumbai - 400 026, India.
E-mail: enquiry@drvishalgupta.com

BASELINE PITUITARY FUNCTION TESTS SUGGESTED PANHYPOPITUITARISM

Follicular stimulating hormone (FSH) 0.79 m IU/ml (0.95-11.95), luteinizing hormone (LH) 0.43 mIU/ml (0.57-12.07), total testosterone 0.88 ng/ml (1.56-5.63), Prolactin 1.14 ng/ml (3.46-19.4), T3 53.49 pg/ml (58-159), T4 6.74 ng/ml (4.87-11.72), thyrotropin stimulating hormone (TSH) 0.08 mIU/ml (0.2-6), Adrenocorticotrophic hormone 68 pg/ml (10-100), Cortisol 17.4 mcg/dl (3.729.4) [tests done while on hydrocortisone treatment], insulin like growth factor- 1 (IGF)-1 55.5 ng/ml (75-212), with a serum sodium of 139 meq/L.

2D echo showed a reduced ejection fraction of 35% with septal and left ventricular apical and lateral regional wall abnormalities.

DEXA densitometry revealed severe osteoporosis at the level of both hip (T-score -3.3) and spine (T-score -3.7).

Magnetic resonance imaging (MRI) pituitary [Figure 1a and b] showed a sellar mass (2.2 × 1.3 cm in size) causing compression of optic chiasma.

A coronary angiogram showed totally occluded right coronary artery proximally, 90% proximal stenosis of left anterior descending artery with heavy calcification and obtuse marginal 1 and 2 showing >85% stenosis. We took the decision to stabilize his heart before removing the pituitary mass transphenoidally. A coronary artery bypass surgery was planned, which was undertaken successfully under appropriate steroid and thyroid hormone cover. After 4 weeks, he had complete resolution of his right sided 3rd cranial nerve palsy. Visual perimetry showed persistent binasal arcuate visual field loss. He underwent a transsphenoidal procedure with the aim of removing the pituitary mass. However, to our surprise we found an empty sella with scar tissue. Postoperatively the patient recovered well. He was discharged on appropriate hormone replacement therapy.

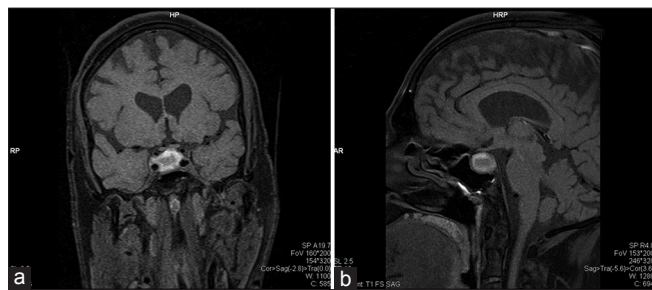


Figure 1: (a) MRI showing a sellar mass (2.2 × 1.3 cm in size) causing compression of optic chiasma, (b) MRI showing sellar mass compressing optic chiasma and invading cavernous sinus

DISCUSSION

Pituitary apoplexy refers to a clinical syndrome characterized by sudden onset headache, vomiting, visual impairment, and decreased consciousness caused by hemorrhage and/or infarction of the pituitary gland, which usually occurs in patients with preexisting pituitary adenomas and prolactinomas evolving over a period of hours or days.^[8] Patients with hypopituitarism are subject to a significant increase in all cause mortality (standardized mortality ratio being 2.06 for males and 2.8 for female) and mortality from vascular disease (standardized mortality ratio 1.4 for males and 1.7 for females).^[9] On the background of diabetes mellitus this cardiovascular mortality is amplified.

The case described brings to light the need for an appropriate cardiovascular evaluation in patients with hypopituitarism especially if they have a background history of T2DM. Current practice in patients presenting with hypopituitarism is to provide appropriate hormonal replacement and follow-up is focused only on the pituitary gland. There may be an argument for aggressive cardiac evaluation and treatment in patients with T2DM associated with hypopituitarism. This is the first case of pituitary apoplexy presenting as myocardial infarction to be reported from India.

REFERENCES

- Gupta V. Adult growth hormone deficiency. *Indian J Endocrinol Metab* 2011;15:S197-202.
- Shahi M, Beshyah SA, Hackett D, Sharp PS, Johnston DG, Foale RA. Myocardial dysfunction in treated adult hypopituitarism: A possible explanation for increased cardiovascular mortality. *Br Heart J* 1992;67:92-6.
- Mayer O Jr, Simon J, Filipovský J, Plásková M, Pikner R. Hypothyroidism in coronary heart disease and its relation to selected risk factors. *Vasc Health Risk Manag* 2006;2:499-506.
- Toft AD, Boon NA. Thyroid disease and the heart. *Heart* 2000;84:455-60.
- Makhsida N, Shah J, Yan G, Fisch H, Shabsigh R. Hypogonadism and metabolic syndrome: Implications for testosterone therapy. *J Urol* 2005;174:827-34.
- Haffner SM, Lehto S, Rönnemaa T, Pyörälä K, Laakso M. Mortality from coronary heart disease in subjects with type 2 diabetes and in nondiabetic subjects with and without prior myocardial infarction. *N Engl J Med* 1998;339:229-34.
- Kannel WB, McGee DL. Diabetes and glucose tolerance as risk factors for cardiovascular disease: The Framingham study. *Diabetes Care* 1979;2:120-6.
- Rajasekaran S, Vanderpump M, Baldeweg S, Drake W, Reddy N, Lanyon M, *et al.* UK guidelines for the management of pituitary apoplexy. *Clin Endocrinol (Oxf)* 2011;74:9-20.
- Sherlock M, Ayuk J, Tomlinson JW, Toogood AA, Aragon-Alonso A, Sheppard MC, *et al.* Mortality in patients with pituitary disease. *Endocr Rev* 2010;31:301-42.

Cite this article as: Gupta V, Patil S, Raval D, Gopani P. Pituitary apoplexy presenting as myocardial infarction. *Indian J Endocr Metab* 2014;18:232-3.

Source of Support: Nil, **Conflict of Interest:** None declared.