*Case Report*

# Management of a post hypophysectomy patient undergoing CABG - A case report

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**Abstract**

*A 56 yrs old male patient of 96 kg, with ASA physical status-III, a known case of Diabetes mellitus, hypertension & acromegaly (s/p hypophysectomy) was admitted in Ibrahim Cardiac Hospital Research Institute (ICHRI) with the history of severe chest pain (compressive) associated with nausea and sweating in cardiology ward. He was diagnosed as a case of NSTEMI. He was treated medically and after stabilization his CAG was done which revealed CAD (TVD) & transferred to cardiac surgery unit for CABG.*

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## Introduction

Acromegaly was first described by Saucerotte in 1801 and by Pierre Marie in 1886. It is seen in both men and women and occurs most frequently in the middle age. It is uncommon, with an estimated prevalence of 50-60 cases per million and an incidence of 3 to 4 cases per million per year[1]. And, acromegaly patient with history of hypophysectomy undergoing CABG is very rare. Acromegaly is characterized by thickening of the subcutaneous tissues of the scalp, lips, tongue, face, hands, feet, overgrowth of the frontal sinuses, jaw and distal phalanges. The soft tissue and bony changes develop slowly over decades. There is also overgrowth of hair and sebaceous glands. But, this disorder is more than cosmetically disfiguring and may involve nearly all the systems. The patients often complain of weakness and tiredness. Asthenia causes slackening of ligaments with kyphosis, scoliosis and occasionally lordosis, so that the enlarged hands hang below the knees. This combined with the atavistic appearance produced by the beetling brows, prognathous jaw, and overgrowth of hair on the chest, produces the 'ape man' of the circus[2]. The acromegalic involvement of the upper airway is the prime cause of concern for the anaesthesiologist. It occurs due to overgrowth of the upper airway, increased length of the mandible, epiglottis and cords[3]. Polypoid masses in pharynx makes them prone for sleep apnea, which can be central, obstructive or mixed. In addition, laryngeal stenosis and cricoid narrowing is often present. The basal metabolic rate is high. Most individuals have neurological and musculoskeletal symptoms, including headache, nerve entrapment and paraesthesia (often due to carpal tunnel syndrome), muscle weakness and arthralgia. The cartilage hypertrophy and osseous overgrowth often leads to degenerative arthritis, or even spinal stenosis[4].

## Case report

He was diabetic for last 23 yrs and was on oral hypoglycemic agent (dimerol) & was on oral anti-hypertensive drug. He was also diagnosed as a case of acromegaly secondary to pituitary adenoma on 1993 with bitemporal hemianopia & bilateral loss of olfactory function. His pituitary adenoma was operated on 30/09/1993 through transsphenoidal approach in the Aga Khan University Hospital, Karachi. After hypophysectomy, he regain his olfaction & vision. He had history of right ulnar fracture 2 yrs back (2005) and was known hypersensitive to penicillin, tetracycline, sulphonamide, β-lactum group cephalosporin & doxicycline. He was on long standing steroids &

thyroxine. On hospital he was on glycerine trinitrate, ACE inhibitor, atorvastatine, omeprazole & injectable short acting insulin. An endocrinologist was consulted pre-operatively and his advice was to check blood glucose, electrolyte, fasting lipid profile & serum cortisol & thyroxin level. The morning dose of thyroxin & steroid was increased on the day of surgery.

His Chest X-ray & other laboratory Investigations were within normal limit. ECG finding was anterolateral old MI. Echo revealed- Aortic sclerosis, asymmetrical septal hypertrophy, mid segment of anterior & inferior septum, apical segment of interventricular septum were hypokinetic, mild mitral regurgitation, LVEF 50%. Basal cortisol level 36.74nmol/L, FT4- 14.64 pmol/L, TSH- 0.34μIU/ml. Duplex study of arterial & venous system of lower limb were normal. Carotid duplex imazing showed- heterogenous calcified plaque situated in right carotid bifurcation (20-30% ICA, 10-15% ECA) & in left carotid bifurcation (10-15% ICA, 20-30% ECA).

The patient underwent CABG on 13/01/2007. Induction of anesthesia was done by thiopental sodium 200mg & fentanyl 500μgm. Orotracheal intubation was performed by pancuronium bromide 10mg & anesthesia was maintained with 50% oxygen in 50% air with halothane (0.5%). Morphine 9mg was given before skin incision. The patient went on cardiopulmonary bypass 2hrs after induction. During bypass fentanyl 50μgm & pancuronium 2mg was given on pump. The patient went back from pump smoothly 3hrs after induction. Time taken from off bypass to the transfer of patient to ICU was 55minutes. During this period 2 doses of fentanyl 50 μgm was given 30 minutes apart & propofol continued at 25-30 μgm/kg/min.

From induction of anesthesia upto the transfer to ICU it was a period of 4hrs. On arrival in ICU, patient was haemodynamically stable with moderate inotropic support. The patient was extubated two & half hours after shifting in ICU with mild to moderate inotropic support. Doses of inotropic support was tapered gradually. Pethedine 25mg IV was given for analgesia. ABG was corrected accordingly & tight glycemic control was maintained using short acting insulin. Patient was haemodynamically stable post-operatively. Normal diet was allowed from first post operative day & use of Spirometry was encouraged. Chest drain was removed & routine medication (thyroxine, hydrocortisone, digoxin & antibiotic) was started from 1st post operative day morning. For analgesia tramadol hydrochloride 50mg orally was given in 1st post operative day & paracetamol 1gm TDS continued for subsequent days. On 3rd POD he was shifted to general ward & on 5th POD was transferred to BIRDEM for endocrine evaluation. From there, he was discharged for home on 7th POD.

**Discussion**

Acromegaly is a chronic, insidious, debilitating disease, which occurs due to acidophillic, or chromophobe adenoma of the pituitary resulting in excess secretion of growth hormone in an adult[5]. These patients often have multi system involvement including respiratory, neuroendocrine, neuromuscular and skeletal systems. Anaesthetic implication of this disorder is particularly significant in terms of changes in the upper airway and increased chances of pulmonary and cardiovascular complications[6]. Acromegaly patient with history of hypophysectomy undergoing coronary artery bypass grafting (CABG) is very rare and seeks extra attention for complex multi system involvement of two different disease pathology.

Cardiac complication in acromegaly patients is well described[7-9]. Acromegaly usually involves cardiac tissue and can occur with coexisting hypertension. The incidence and severity of cardiac hypertrophy relates to the duration of the disease. Cardiomegaly was found to be disproportionateto the other organ hypertrophy. There is little evidence to support that there is accelerated atherosclerosis in this population. Oversecretion of GH with acromegaly produces resistence to the effects of insulin, which leads to glucose intolerance. This was noted in our patient, with blood sugar concentration being significantly higher. This is important, as hyperglycemia worsen some type of cerebral ischaemia[10]. Our patient was on short acting insulin and a tight glycemic control was ensured per & postoperatively. Once it is suspected, ideally, advance tests like basal or random growth hormone (GH) assay should be employed to confirm the diagnosis. GH concentration are

measured and failure of hormone concentration to decrease 1-2 hours after the ingestion of 75-100g of glucose is presumptive evidence of acromegaly. This patient was on thyroxine & steroid replacement therapy pre-operatively and continued after surgery. According to Agastas, systemic involvement should always be kept in mind while giving anaesthesia to these patients. Hypertension occurs in 1/3 cases, half of which have increased left ventricular mass or left ventricular wall thickness. Although, it is not established, whether cardiomyopathy occurs, acromegalics may develop congestive cardiac failure in the absence of another known underlying heart disease[11].

Management of anaesthesia for these patients is to be considered in the backdrop of safety and appropriateness as also available logistic and infrastructural support. Effort was to minimize mechanical trauma to the upper airway and vocal cords, as additional edema would have resulted in more postoperative edema. Etomidate inhibits the synthesis of cortisol transiently should be avoided. There is also no evidence that hemodynamic instability or alteration in pulmonary gas exchange accompany anesthesia in acromegalic patients.

Concluded, no specific anesthetic technique is recommended for acromegalic patient undergoing CABG. In acromegalic patients airway difficulty occur most frequently. Severe haemodynamic instability do not typically occur during surgery. Pulmonary gas exchange was not altered during surgery; glucose intolerance may be an intraoperative problem and fluid regulation may be altered. These patients should be managed aggressively with invasive monitoring , intravenous corticosteroids, and fluid electrolyte resuscitation. Minimum doses of anesthetic agents and drugs are recommended since myocardial depression and skeletal muscle weakness are frequently part of clinical scenario.

**References**


1. Barkan AL. Acromegaly : Diagnosis and treatment. Endocrinol Metab Clin North Am 1989;18(2):277-310
2. Hassan SZ, Matz G, Lawrence AM, Collins A. Laryngeal *MJAFI, 2003; 59: 3.* stenosis in acromegaly. Anesth Analgesia 1976; 55: 57-60
3. Stoelting R, Dierdorf SF. Anaesthesia and coexisting diseases, 3rd ed, Churchill Livingstone, Acromegaly. 1993;369-70
4. MKBiller B,Daniels GH. Neuromuscular regulations and diseases of the anterior pituitary and hypothalamus. Harrison's Principle of Internal Medicine 14th ed. McGraw Hill Publications, Edited by Anthony S Fauci, Eugene Braunwald et al. 1998; 2: 1980-83.
5. Rushman GB, Davies NJH, Cashman JN. Diseases of the pituitary gland. Lees Synopsis of anaesthesia 12th ed. Reed Education and Professional Publishing. 1999;339-40
6. Kitahata LM. Airway difficulties associated with anaesthesia in acromegaly. BJA 1971;43:1187-90
7. Rossi L, Thiene G, Caregaro L, Giordano R, Lauro S. Dysrhythmias and sudden death in acromegalic heart disease. Chest 1977; 72: 495-8
8. Lie JT. Pathology of the heart in acromegaly: Anatomic findings in 27 autopsied patient. *Am Heart J 1980;* 100: 41-52
9. McGuffin WL jr, Sherman BK, Roth J*, et al.* Acromegaly and cardiovascular disorders: a prospective study. *Ann Intern Med* 1974; 81: 11-18
10. Sieber FE, Traysman RJ. Special issues: Glucose and the Brain. CCM 1992; 20: 104-14
11. Agastas L, Muri M, Clark N. Heart disease in acromegalics. Endocrinol Metab Clin North Am 1991;20:619