

Anesthetic Management Of Resection Of A Cortisol Secreting Tumour: Cushing's Syndrome, Perioperative steroid replacement

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Citation

A Kulkarni, G Rastogi, A Bhargava. *Anesthetic Management Of Resection Of A Cortisol Secreting Tumour: Cushing's Syndrome, Perioperative steroid replacement*. The Internet Journal of Anesthesiology. 2006 Volume 14 Number 1.

Abstract

A young male presented with features suggestive of Cushing's syndrome. On investigation serum cortisol levels were raised. C T Scan of the Abdomen revealed a large mass in the region of left adrenal gland. He was posted for resection of the adrenal cortical tumour. Anaesthetic management and perioperative steroid replacement for unilateral adrenalectomy is discussed.

CASE REPORT

A 24 year old male presented with complaints of weight gain about 12 kg in 3 months headache and puffiness of face for 1 month as seen in Figure 1.

Figure 1

Figure 1: Moon shaped face suggestive of Cushing's syndrome.



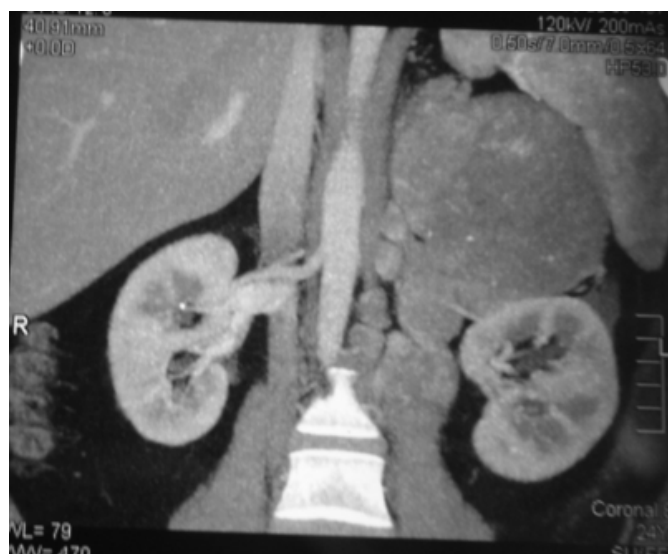
On G/E patient was obese, wt-93kg, BMI - 32, moon shaped

face, Pulse –88/min, BP-160/110mmHg. RS/ CVS-NAD, P/A striae were visible. Haematological and Biochemical investigations were normal, except decreased serum potassium –2.6mEq/L, X-Ray Chest and ECG was normal. 2D Echocardiography revealed LVEF 68%, No RWMA. CT Brain was normal. 24 hr urinary cortisol was 25 mg/day (Normal range 3 – 10 mg/day). Serum Cortisol levels were raised, morning– 49.64mcg/dl (Normal range 4.3-22.4) Evening -52.07mcg/dl(Normal range 3.1-16.6)

Computerised Tomography (C T) Scan of the Abdomen revealed large mass measuring 11.8 x 11.2 X 9.2 cms in Left Adrenal gland region as shown in Figure 2

Figure 2

Figure 2: C T Scan Abdomen, arrow shows Left adrenal adrenal mass.



Fine Needle Aspiration Cytology of the adrenal mass was positive for malignancy. Patient was accepted for Left Adrenalectomy as ASA Grade III. He was optimized preoperatively; hypertension was controlled with Tab Carloc 12.5mg BD, Tab Telistra RM 5mg BD and Tab Aldactone 1 OD. Hypokalemia was treated with Potassium Chloride 60mEq in Normal Saline to raise Serum Potassium to 3.8meq/L. Blood Glucose was controlled with sliding scale insulin. Patient received antibiotic prophylaxis and protien supplementation. Premedication included continuation of antihypertensive drugs till the morning of surgery. Tab Diazepam 5 mg HS and at 6 am. Tab Ranitidine 150 mg and Tab Granisetron 2 mg.

On shifting to the OT, the patient had HR 84/min, BP 160/108mmHg, and iv midazolam 1mg was given. Standarad monitoring, also including PNS, CVP and IBP was applied.

An epidural catheter 16 G was placed in T11 – T12 interspace, 3cms cephalad, 2.5ml 0.5% bupivacaine test dose was given.

Anaesthesia was induced with morphine 0.75-0.1mg/kg⁻¹, fentanyl 1.5 -2µg/kg⁻¹, propofol 2-3mg/kg⁻¹ and atracurium 0.5mg/kg⁻¹. Trachea was intubated with cuff portex ETT 8mm ID. Anaesthesia was maintained with Isoflurane 1-1.5% in 33% Oxygen and Nitrous Oxide, incremental doses of fentanyl and atracurium. During surgery, the patient also received epidural morphine 3mg and intermittent 0.25%

bupivacaine 8-12 ml.

At the initiation of resection of the adrenal tumour IV Hydrocortisone Succinate 100mg bolus was given and hydrocortisone infusion was started @ 100mg/24hrs.

Intraoperatively, the patient remained haemodynamically stable. Surgery performed was Left Adrenalectomy, left Nephrectomy and Para aortic lymphadenectomy. Duration of surgery was 4 hours 26 min.

At the end of surgery neuromuscular blockade was reversed with neostigmine 3.0mg and glycopyrolate 0.5 mg. Postoperatively the patient was electively ventilated with SIMV (12 breaths), PSV 20cms H₂O mode with Fio₂ -40% For sedation midazolam infusion 1mg/hr, morphine infusion 1mg/hr was commenced. For postoperative pain relief epidural bupivacaine 0.125% infusion 5 ml/hr was given. The trachea was extubated successfully 16 hours after surgery. Postoperatively the patient received steroid supplementation with hydrocortisone succinate infusion as shown in T able 1.

Figure 3

Table 1: Postoperative steroid supplementation with Hydrocortisone succinate infusion

Postoperative Day	Hydrocortisone Infusion dosage
Day 1	5mg/hr
Day 2	3mg/hr
Day 3 and 4	1.5mg/hr
Day 5	1 mg/hr
Day 6	Tab. Methyl Prednisolone 10 mg BD post meal

DISCUSSION

Glucocorticoids regulate protein, fat, carbohydrate and nucleic acid metabolism. Blood glucose is raised by antagonising the secretion and action of insulin. Glucocorticoids cause protein catabolism and fat mobilization. Anti-inflammatory action is due to effects on microvasculature and suppression of inflammatory cytokines.

Increased production of cortisol results in Cushing syndrome characterised by truncal obesity, hypertension, hyperglyceamia, hypokalemia, fatigability, myopathy, amenorrhea, hirsuitism, purpulish abdominal striae, odema

and osteoporosis.^{1,2}

Causes of Cushing's syndrome are adrenal hyperplasia usually secondary to pituitary ACTH overproduction, Adrenal neoplasia, and exogenous causes due to prolonged use of glucocorticoids. Approximately 20-25% of patients with Cushing's syndrome have an adrenal neoplasm, usually unilateral, and about half of these are malignant.² In our patient signs and symptoms suggestive of Cushing's Syndrome and investigations lead to the diagnosis of cortisol secreting adrenal tumour and the patient was posted for left adrenalectomy. Preoperative preparation included control of hypertension, diabetes, normalizing intravascular fluid volume and electrolyte concentrations. Prophylactic antibiotics are recommended as due to lympholytic and immunosuppressive action patients are prone to infection. Intraoperative considerations include careful positioning of the osteopenic patient for both regional and general anaesthesia to avoid fractures³, and control of blood glucose.

Intraoperative monitoring depends on patient's cardiac reserve and the extent of surgery.

Muscle relaxants should be used in titrated doses due to existing myopathy and hypokalemia. Controlled ventilation is advisable considering underlying respiratory muscle weakness.

GLUCOCORTICOID REPLACEMENT THERAPY

The normal adrenal gland can secrete up to 200mg of cortisol /day. During periods of extreme stress the gland may secrete up to 500mg /day of cortisol.² The pituitary – adrenal axis is considered to be intact if a plasma cortisol level of greater than 22mg/dl is measured during acute stress.

Mean maximum plasma cortisol level reached for major surgery (colectomy, hip osteotomy) is 47mg/dl and for minor surgical procedures (Herniorrhaphy) is 28mg/dl.

Endogenous cortisol levels rise up to 75-150mg in response to major surgery and the secretion parallels duration and extent of surgery.⁴ The main increase takes place immediately after anaesthesia is induced and cortisol levels return to base line within 24-48 hrs after surgery. This short term elevation prevents stress induced hypotension and shock. When unilateral or bilateral adrenalectomy is planned glucocorticoid replacement therapy is initiated at the start of resection of the tumour, at a dose equal to full replacement of adrenal output during periods of extreme stress.^{2, 5}

Popular regime for steroid replacement is administration of 200-300mg of hydrocortisone per 70kg body weight in divided doses on the day of surgery. In low dose cortisol replacement program iv cortisol 25mg is given before the induction of anaesthesia followed by continuous infusion of cortisol 100 mg in 24 hrs.²

Michael F. Roizen and Lee A. Fleisher, advocate 100mg of iv hydrocortisone every 24 hours ⁵. The total dosage is reduced by approximately 50% per day over a period of 3-6 days until a daily maintenance dose of steroids (20-30 mg.day-1) is achieved.

Hydrocortisone given in high doses exert mineralocorticoid activity hence exogenous replacement is not necessary during the perioperative period⁵. Functioning solitary adrenal cortical tumours are treated surgically, unilateral adrenalectomy may be followed by normalization of function in the contralateral gland over a period of several months.^{2,5}

It is recommended that these patients should receive physiological steroid replacement therapy to avoid perioperative haemodynamic instability⁴. Side effects of excessive steroids are hyperglycaemia, immunosuppression, protein catabolism, impaired wound healing, hypertension, fluid overload, and increased susceptibility to infection.

CONCLUSION

We conclude that when unilateral adrenalectomy is planned glucocorticoid replacement therapy should be initiated at the start of resection of the tumour, at a dose equal to full replacement of adrenal output during periods of extreme stress ^{2, 5} to avoid haemodynamic instability.

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