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Case Report

Management of a case of parathyroid adenoma- An anaesthetic challenge

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ABSTRACT

Calcium plays a major role in a number of physiological actions. Primary hyperparathyroidism is the most common cause of hypercalcaemia. Many patients with primary hyperparathyroidism are asymptomatic. Parathyroidectomy is the definitive treatment for primary hyperparathyroidism. We present the successful anaesthetic management of a patient with primary hyperparathyroidism and discuss the anaesthetic issues involved.

1. Introduction

Calcium plays a major role in a number of physiological actions. The effects of calcium on the myocardium, vascular smooth muscle and blood coagulation are of special importance to the anaesthetist. Primary hyperparathyroidism is the most common cause of hypercalcaemia, with most of the patients being asymptomatic.[1] The incidence of hyperparathyroidism ranges from 1 in every 500 women and 1 in every 2000 men older than 40 years.[2] Parathyroidectomy is the definitive treatment and multisystem involvement poses anaesthetic challenges.

2. Case Report

A 52-year-old female patient weighing 30kg presented with weight loss since 1 month; increased thirst and frequency of micturition since 3-4 days and abdominal pain since 2 days.

Her renal functions and liver function tests were within normal limits. Preoperative ECG and QTc interval was normal. Radiographs of knee and skull bones revealed reduced bone density. Ultrasound neck revealed right parathyroid adenoma which was confirmed by cytology. A diagnosis of primary hyperparathyroidism was made on the basis of hypercalcaemia with increased parathormone levels. Preoperative optimisation was done with intravenous fluids and Inj Furosemide. Subsequently her total Serum Calcium was normalised. Her hydration status and serum electrolytes were monitored during this forced saline diuresis therapy. After adequate preoperative evaluation and optimisation, patient was posted for right parathyroidectomy. Investigations on the day of surgery were within normal limits. Sr Mg was low, hence correction was given.

In the operating room, non invasive monitors were attached. Patient was premedicated with Inj Ondensetron, Inj Glycopyrollate, Inj Midazolam, Inj Fentanyl. Anaesthesia was induced with Inj Propofol and Inj Atracurium was administered to facilitate endotracheal intubation. Anaesthesia was maintained with O2:N2O and Isoflurane and Inj Atracurium. After intubation, Superficial Cervical Plexus Block was given under USG guidance with

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Table 1: Preoperative investigations [IU/L- International Units/Litre]

	Day 1	Day 2	Day 3	Day 4	On the day of surgery
Total Calcium (8.5-10.3 mg/dl)	20	17	14	9.51	9.1
Ionic Calcium (1.1-1.32mmol/l)	1.98	1.12	1.1	1.16	1.23
Serum Magnesium (1.8-2.6 mg/dl)	2.07				1.1
Serum Phosphorous (2.5-4.5 mg/dl)	3.9				3.8
Parathyroid Hormone (11-79 mg/dl)	841				
Vitamin D3 (25-80 ng/ml)	11.83			12.93	
Alkaline Phosphatase (44-147 IU/L)	411				

Inj Bupivacaine. Invasive BP monitoring was established by cannulating the right radial artery. Patient's ECG and QtC was continuously monitored. Patient's heart rate and blood pressure were stable throughout the surgery. End tidal CO2 was maintained between 32 and 36 mmHg. Intraoperatively arterial blood gases were monitored along with Serum Calcium and Ionised Calcium.

Input and output was monitored. After completion of surgery, neuromuscular blockade was reversed. Post extubation check laryngoscopy was suggestive of right vocal cord palsy.

Postoperatively, the patient was kept in post anaesthesia care unit. Arterial Blood Gas Analysis was repeated 12hrly and was within normal limits. Serum calcium levels were checked regularly. Patient developed features s/o carpopedal spasm on day 4 along with a decreasing trend seen in serum Calcium for which Inj Calcium Gluconate was given.

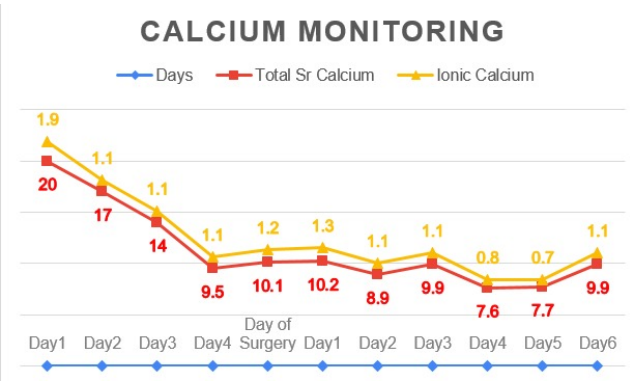

Fig. 1: Trend in serum calcium levels post operatively

Inj MgSo4 was given on Day 2 and Day 3. Indirect Laryngoscopy was done which was suggestive of right vocal cord palsy for which she was counselled. Histopathology report revealed parathyroid adenoma. She was discharged with normal serum calcium levels on oral medications and advised follow up in routine OPD.

3. Discussion

Calcium is important for various biological processes which include cardiac automaticity, excitation contraction coupling, blood coagulation, neuronal conduction, synaptic transmission, hormone secretion. Calcium exists intracellularly and extracellularly. Calcium in extracellular compartment is present in three forms. It is the ionized form of extracellular calcium that is responsible for all the physiological effects. Levels of extracellular calcium depend on three main calciotropic hormones: parathyroid hormone (PTH), vitamin D and calcitonin. Single parathyroid adenoma is the most commonly seen lesion among parathyroid disorders (80%). Multiple gland hyperplasia is seen in about 10-20% of patients whereas parathyroid carcinoma is rarely seen (1%).[1] Parathyroid hormone acts on bone causing resorption of calcium and on kidney causing reabsorption of calcium. Other actions of parathyroid hormone on kidney includes the conversion of 25 – hydroxycholecalciferol to 1,25 –dihydroxycholecalciferol. 1,25 –dihydroxycholecalciferol, being the active form of vitamin D acts on the intestine to increase absorption of calcium and causes calcium reabsorption from the kidneys. The parafollicular C-cells of the thyroid gland secrete calcitonin in response to a high plasma Calcium concentration. Calcitonin inhibits bone resorption and encourages calcium excretion in urine, thus counteracting the actions of parathyroid hormone.Increased secretion of parathyroid hormone causes hyperparathyroidism. Hyperparathyroidism may be primary or tertiary (due to intrinsic pathology of glands) or secondary (due to extrinsic pathology). Many patients with primary hyperparathyroidism are asymptomatic. Only 20–30% of patients are symptomatic at the time of diagnosis.[3]

In symptomatic patients common findings include renal calculi, bone pains, pathological fractures, skeletal muscle weakness or non-specific symptoms such as depression, lethargy, and pains, the classic "moans, stones and groans" presentation. The disease is more common in women and

Table 2: Effect of gland removal on serum calcium and magnesium

	Pre Gland Removal	Post Gland Removal
Serum Calcium	12.4 mg/dl	11.7 mg/dl
Serum Magnesium	2.3 mg/dl	2.1 mg/dl

elderly, therefore the peak incidence is in post-menopausal women.).[1] Renal manifestations include nephrolithiasis as the commonest symptom. Severe form of acute hypercalcaemia impairs renal tubular function, leading to nephrogenic diabetes insipidus. Such patients usually present with dehydration, polyuria and progressively worsening hypercalcaemia. Reduction in cortical bone density is seen in patients with primary hyperparathyroidism with an increased risk of distal fractures. Bone density seems to improve after surgical parathyroidectomy.

Patients can also present with gastrointestinal complaints which include constipation, anorexia and non-specific abdominal pain.[3] Prolonged PR interval, short QT interval and systemic hypertension are some of the cardiac related complaints.[1] Primary hyperparathyroidism can be demonstrated by persistent hypercalcaemia in the presence of normal to elevated parathyroid hormone levels and ultrasonography of neck.[1] Elevated markers of bone turnover, such as osteocalcin and bone-specific alkaline phosphatase, decreased 25- hydroxycholecalciferol levels and normal to mildly high levels of 1,25-dihydroxycholecalciferol may also be seen.[3]

In our case, the patient presented with bilateral lower limb pain along with nausea, vomiting and abdominal pain. The patient was investigated which subsequently demonstrated hypercalcaemia with primary hyperparathyroidism. The most sensitive technique for preoperative localization of hypersecreting parathyroid gland appears to be ultrasonography and technetium-99m sestamibi tomographic nuclear scanning. If medical management fails, Parathyroidectomy is the definitive treatment for primary hyperparathyroidism associated with 95% cure rate.[1]

Patients who present for parathyroidectomy are often elderly and are therefore more likely to have significant comorbid conditions. These conditions need to be evaluated and optimised preoperatively. Severe hypercalcaemia is a medical emergency. Patients usually present with polyuria, vomiting, dehydration, tachycardia, and even psychosis. If left untreated, it may lead to coma and collapse.

Initial therapy for severe hypercalcaemia includes administering IV fluids. Diuretics must be started after euvolemia is achieved. Loop diuretics increase the urinary calcium excretion by inhibiting the proximal tubular reabsorption of calcium. Thiazide diuretics cause renal tubular reabsorption of calcium and are hence avoided. Forced diuresis may cause hypophosphataemia, hypokalaemia and hypomagnesaemia. In our case, normocalcaemia was achieved with hydration and furosemide therapy.[1]

Multisystem involvement poses anaesthetic challenges. Preoperative correction of malnutrition and hypoalbuminemia is essential as these conditions may alter calcium levels. Neurological assessment and other endocrinopathies must be ruled out.

Due to increased vulnerability to fractures, careful positioning and laryngoscopy is essential. In the intraoperative period, special focus needs to be made on the acid base status.[4] Hypercalcaemia may be associated with disturbance in cardiac rhythm, hence continuous ECG monitoring is required. Warming devices may help prevent hypothermia. Blood loss is usually minimal. It is advisable to use a peripheral nerve stimulator for neuromuscular monitoring. It is important to maintain normocarbia as acidosis decreases calcium binding to albumin causing life threatening hypercalcaemia.

Fig. 2: Intraoperative picture of the monitor

Various complications may occur, including bleeding, metabolic abnormalities, recurrent laryngeal nerve trauma, oedema of the glottis and hypocalcaemic tetany, postoperative haematoma.Unilateral recurrent laryngeal nerve injury causes hoarseness of voice while Bilateral nerve injury may cause stridor. Hence position of the vocal cords must be checked during extubation. After parathyroidectomy magnesium or calcium may be redistributed internally into the bones causing hypomagnesaemia, hypocalcaemia or both.

Postoperative hypoparathyroidism needs to be monitored carefully. Hypocalcaemia due to removal of all four glands presents 6–24 hours postoperatively. The patients initially complain of perioral paraesthesia followed by restlessness and neuromuscular irritability (positive Chvostek's and Trousseau's signs and stridor). Prolongation of the ST and QT intervals may be evident on electrocardiogram. If left untreated, severe tetany may develop.[3]

Non-steroidal anti-inflammatory drugs should be avoided for postoperative analgesia if there is renal function impairment.[1] Parathyroid surgeries are usually done under general anaesthesia but in patients with severe cardiorespiratory dysfunction, it can be done under Peripheral nerve blocks which include cervical nerve blocks. Cervical nerve blocks may be deep, superficial or combined. Deep cervical block carries a risk of inadvertent injection into dural space or vertebral artery or phrenic nerve palsy. Superficial or combined superficial and deep cervical plexus block are comparatively safer. In our case we used general anaesthesia with superficial cervical plexus block for postoperative analgesia.[5]

4. Conclusion

It is worth emphasizing that successful anaesthetic management of a patient with hyperparathyroidism requires vigilance for factors that might potentiate adverse effects of hypo and hypercalcaemia. Adequate preoperative assessment and optimisation, close monitoring of the signs and symptoms of hypo-and hypercalcaemia, restoration and keeping ionized calcium within normal limits during perioperative period is must for the successful anaesthetic management of such patients.

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6. Conflict of Interest

None.

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