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HUNGRY BONE SYNDROME (HBS)- A CASE REPORT

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INTRODUCTION

Hungry bone syndrome is a rare but severe condition where the patients calcium levels drops persistently for a long time period after parathyroidectomy. Along with hypocalcaemia patient may also experience hypophosphatemia and hypomagnesaemia. It is a relatively uncommon but serious adverse effect of parathyroidectomy.

Parathyroid Hormone (PTH) produced by parathyroid gland helps to regulate and maintain the calcium levels in the body. There are four parathyroid gland and hyperactivity of these glands (Hyperparathyroidism) leads to increase in the PTH levels which further increase the serum calcium by forcing bones to release high amount of calcium in to the blood stream. ALP (Alkaline phosphatase) values will also be high in such cases. In patients with severe hyperparathyroidism post-operative period after the removal of one or more hyper active parathyroid glands the PTH levels in the body drops drastically which results in the drop in calcium levels which usually settles within four days. In patients with HBS the calcium levels along with phosphorus and magnesium drops even after the fourth day of surgery. The severe hypocalcaemia is mainly due to increased calcium influx into the bones. Patients with HBS will also have sever VIT d deficiency. HBS is characterised by long standing hypocalcaemia along with symptoms like Chvostek sign and Trousseau sign, perioral paraesthesia, carpopedal spasms, tingling extremities.

The treatment of HBS is aimed to restore the severe calcium deficiency. It is usually done with oral calcium supplementation but in patients with HBS the amount of calcium supplementation required is higher than the maximum dose which can be tolerated by the patient hence IV calcium supplementation is usually given along with the oral tablets. Further treatment with active vitamin D and phosphorus and magnesium supplements are also done.

CASE STUDY

A 46-year-old female patient during health check for complaints of asymptomatic swelling in front of neck, c/o pain in right knee, itchy lesions all over body with history of hyperkalaemia and vitamin D deficiency, she was evaluated for the same and found that PTH is high 1137 pg./mL, hyperkalaemia (10.3 mg/dL) and elevated serum ALP levels (602). Fibro scan, MRI and MIBI scan for parathyroid was conducted which revealed hyper functioning parathyroid adenoma in the lower pole of the right lobe of thyroid gland. She was diagnosed with Right Inferior Parathyroid Adenoma and Primary Hyperparathyroidism.

She was explained regarding the condition and surgical intervention was planned- RIGHT INFERIOR PARATHYROID ADENOMA EXCISION + FROZEN SECTION + LEFT SUPERIOR PARATHYROID NODULE EXCISION + LEVEL VI LYMPH NODE DISSECTION DONE UNDER GENERAL ANESTHESIA.

The day 1 to day 3 of postoperative period was uneventful with calcium being 7.6, 8.1, 8.4 and PTH 75.2, 34.9 and 49.7 with mild symptoms which was treated with oral supplements respectively. on the fourth day after surgery the patient started experiencing severe symptoms like contraction of fingers and toes, facial numbness, mental confusion, depression etc and the lab investigations reflected calcium was dipped to 6.5 along with the phosphorus being 1.9 and Vitamin D 4.04. she was started with IV calcium (1 mg/ mL elemental calcium in DNS at rate of 50 mL/hr) with oral calcium, phosphorus supplements and 6L units of active Vitamin D IM STAT. on day 5, day6 and day 7 the calcium had further dipped to 6.4,6.2 and 5.5 respectively along with vitamin D reducing to 3.17 and magnesium being 1.9 . she was continued treatment with IV calcium (1 mg/ mL elemental calcium in DNS at rate of 50 mL/hr) oral calcium, magnesium supplements and 6L units of active vitamin D IM STAT. on Day 7 the calcium started improving from 5.5 to 6.4 and vit D becoming 11.8 from 3.17. the symptoms started improving and the flow rate of IV calcium infusion was reduced to 20 ml/hr on Day 9 the Patient showed minimal symptoms with s calcium being 7 with support of IV calcium infusion 1mg/ml @ 20 ml/ hr rate.

DISCUSSION

Hungry bone syndrome is an unusual but serious condition where the calcium levels drop persistently for a long period of time after the parathyroidectomy. they also experience hypophosphatemia and hypomagnesaemia along with hypocalcaemia.

The prevalence of HBS is varied and has changed. In 1980, it was estimated that approximately 13% of cases post parathyroidectomy for primary hyperparathyroidism, but more recently, in case series, reports are as low as 4%. HBS can bring significant morbidity related consequences like hypocalcaemia, generalized seizures, congestive heart failure. Treatment option for preventing the hungry bone syndrome is preoperative treatment with bisphosphonates.

The management of hungry bone syndrome involves, monitoring of serum electrolytes such as calcium, phosphate, and magnesium. The treatment involves administration of elemental calcium. The route of administration depends on severity of signs and symptoms. Oral calcium supplements can be used in mild symptoms and serum calcium concentrations greater than 7.5 mg/dl. intravenous treatment is considered for the patients with below 7.5mg/dl or prolonged QTC interval.

The 46 years old patient discussed in this report was administered with oral supplements for initial three days where calcium levels were 7.6, 8.1, 8.4 with mild symptoms. On day four patient experienced severe symptoms and the lab parameters revealed that calcium drops down to 6.5, phosphorus 1.9 and vitamin D- 4.04. therefore, she was started with IV calcium (1mg/ml elemental calcium in DNS at rate of 50ml/hr) with oral supplements of magnesium, phosphorus and 6L units of VIT D IM STAT. on further days, calcium continuously dropped down to 6.4, 6.2 & 5.5 on respective days along with reduced VIT D – 3.17, she was continued with IV calcium. On day seven her lab parameters were improved, on day nine patient was showing mild symptoms.

CONCLUSION

As a result of parathyroidectomy or therapy for hyperparathyroidism, hungry bone syndrome (HBS) is an uncommon but significant condition that can happen. Hypocalcaemia and hypophosphatemia are the results, and they are characterised by a fast decline in blood calcium levels and an increase in bone absorption of calcium. Those who have severe

hyperparathyroidism, chronic renal illness, or who have seen significant bone resorption are more prone to experience this syndrome.

The treatment of HBS entails careful monitoring of blood calcium and phosphate levels as well as the administration of calcium and vitamin D supplements as necessary. Calcium and phosphate intravenously may be needed in extreme circumstances. Preoperative evaluation should be thorough, calcium and vitamin D levels should be optimised, and hyperparathyroidism should be gradually corrected if HBS is to be avoided.

Thus, HBS is an uncommon but potentially lethal complication that has to be promptly identified and managed. Healthcare professionals should be aware of this disease and take the appropriate precautions to avoid and treat it in patients receiving therapy for hyperparathyroidism.

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