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Case Report

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SYSTEMIC LUPUS ERYTHOMATOSIS: AS A RARE CAUSE OF SEVERE HYPERCALCEMIA

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ABSTRACT

Hypercalcemia is a relatively common electrolyte abnormality observed in clinical practice. Great majority of patients with hypercalcaemia will be found to have either primary hyperparathyroidism or malignancy as a cause of hypercalcemia. Nevertheless other rarer causes are important to consider in certain clinical situations, when underlying cause cannot be attributed to hyperparathyroidism or malignancy. Systemic Lupus Erythomatosis[SLE] is a very rare cause of hypercerlcemia. Only a handful cases of hypercalcemia secondary to SLE are reported in medical literature. We describe a case of pregnant lady who was diagnosed with SLE previously presenting with concomitant severe hypercalcemia and discuss the path physiological mechanisms contributing to hypercalcemia in SLE and its management with review of literature.

KEYWORDS: Systemic lupus erythomatosis, severe hypercalcemia, rare cause of hypercalcemia.

INTRODUCTION

Systemic lupus erythomatosis [SLE] is an autoimmune disease with protean manifestation in multiple organs. The clinical manifestations include arthritis, dermatitis, involvement of cardiovascular, respiratory, central nervous and hematological systems.[1] Hypercalcemia is usually caused by primary hyperthyroidism, and malignancy. SLE is unusual and rare cause of severe hypercalcemia reported in about 12 cases in medical literature. Several mechanisms were attributed to explain the hypercalcemia in SLE. We report a 36 year-lady with 36 week pregnancy and known case of SLE presenting with severe hypercalcemia.We discuss the path physiological mechanisms involved in development of hypercacemia in SLE and its management.

CASE REPORT

A 36-year- woman at 36week pregnant presented to the emergency room[ER] complaining of nausea, vomitings, polyuria,constipation of 4 days duration. There was history of joint pains, myalgias, worsening of facial rash, and fatigue 3 weeks prior to the presentation to ER. The patient was diagnosed to have SLE fulfilling 7 of 17 criteria of SLICC[systemic lupus international collaboration clinic] classification criteria for classification of SLE[2], four years before and being treated and followed up in Rheumatology clinic. She was taking prednisolone 10 mg once a day, along with Enoxheparin, hydroxychloroquine. On examination she was conscious and oriented. Pulse: 105 BPM, BP: 100/70 mm/Hg,Temparature:37C. There was rash on the malar area of face. Cardiovascular, respiratory, central nervous system examination were normal. Abdominal exam revealed gravid uterus around 36 weeks with normal fetal heart sounds. Laboratory investigations shown in table no:1.

Table no: 1 Laboratory Results.

Variable	Normal range (SI Units)	3 weeks Before admission	On admission	1 month after admission	4 months after admission
White blood count	3.3-10x10^9	5	8.4	___	___
Hemoglobin	12-16gm/dl	10.9	11.4	___	___
Platelets	150-500x10^9	205	326	___	___
Serum creatinine	40-80 µmol/l	66	125	81	69
Serum Albumin	38-48 gm/l	36	27	31	34.5
Serum calcium	2.15-2.55 mmol/l	2.46	4.52	2.52	2.33
Serum phosphate	0.87-1.47mmol/l	0.95	1.28	1.01	1.35
Thyroid stimulating hormone	0.27-4.2µ	1.86	0.772	___	___
Serum thyroxin	12-22pmol/l	13.9	14.69	___	___
ESR	10-14 mm/hr	72	102	___	54
C-reactive protein	0.425-5 mg/l	11	80.9	___	2.7
Random blood sugar	3.9-8.3 mmol/l	5.4	6.3	___	___
C3 compliment	0.9-1.8gm/l	1.2	0.0852	___	1.1
C4 compliment	0.16-0.47gm/l	0.186	0.126	___	0.224
i PTH	1.6 -6.9pmol/l	___	1.6	___	___
Angiotensin converting enzyme	8-52 u/l	___	41	___	___
1-25 dihydroxy vitamin D	53-267 pmol/l	___	30	___	___
25 hydroxy vitamin D	75 -250 pmol/l	___	71.09	___	___
ANA	< 1:10	>1:160	___	___	___
Anti DNA antibodies	<1:10	>1:80	___	___	___
Anti cardiolipin antibodies IGG	-	Reactive	___	___	___
Anti Sm antibodies	<10.0 u/l	<10.0	___	___	___
PTHrP	<1.3 pmol/l	___	<0.8	___	___

Patient was diagnosed to have severe hypercalcemia, dehydration and acute kidney injury[AKI], and was started on intravenous fluids initially. Several sessions of hemo dialysis were performed as hypercalcemia was severe and not adequately responding to intravenous fluids. Patient underwent cesarean section to deliver the baby. One dose of Zolendronic acid 4 mg was given after the patient delivered,. With rehydration and Zolendronic acid treatment serum calcium became normal and AKI resolved. As iPTH and PTH rP were normal and other causes of severe hypercalcemia were excluded by preliminary investigations, hypercalcemia secondary to SLE was considered. Clinical and biochemical flare up of SLE was diagnosed by rheumatologist during the admission and prednosolone dose was increased to 40 mg once a day. Patient was delivered by caesarean section. Subsequent days Patients serum calcium normalized and patient was discharged on prednisone 40 mg once a day. Follow up visits to the out-patient clinic over a period of four months revealed normal serum calcium.[Fig:1] Steroids were planned to be tapered to her usual maintenance dose of prednisolone :10mg once daily.

Legend

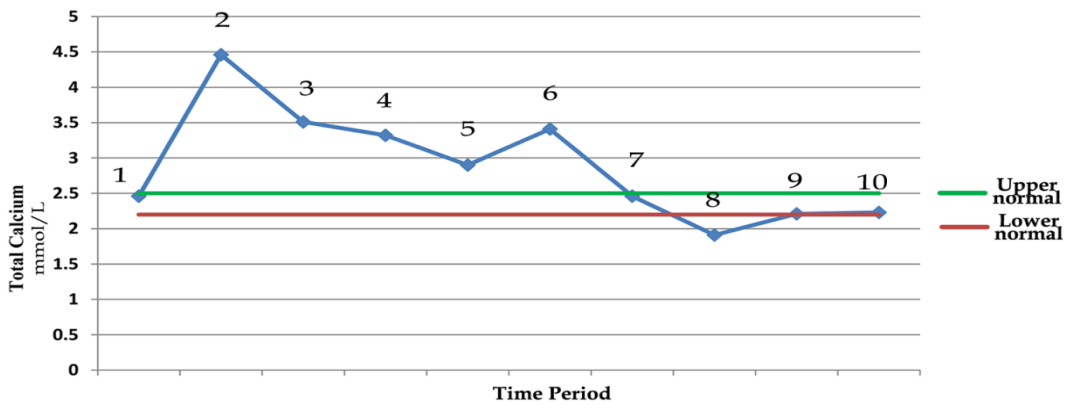

Fig no 1: Figure showing serum calcium levels at different periods during management of the case.1: Before admission, 2:On admission, 3,4,5: On IV fluids and hemodialysis, 6: At start of steroids and zolendronic acid,7,8: At discharge,9: one month after discharge on steroids, 10: On 4 months after discharge on steroids.

DISCUSSION

Hypercalcemia in majority of cases usually caused by primary hyperparathyroidism and malignancy. In less than 10% of cases other less common causes like vitamin D intoxication, Grannulomatous diseases, thyrotoxicosis, milk-alkali syndrome, immobilization were recognized.[3] Nevertheless it's important to consider in certain clinical situations when underlying cause of hypercalcemia cannot attributed to potential causes listed above. Rare etiologies and diseases reported contributing to hypercalcemia reported in single or in small groups of patients should be explored.SLE is such a rare cause of severe hypercalcemia. To our knowledge only handful of cases SLE associated hypercalcemia were reported in English medical literature.[4-10] The path physiology of hypercalcemia in SLE is not completely understood. High PTHrP and immunochemically identifiable PTHrP in excised lymph nodes of SLE patients with hypercalcemia were reported in 2 cases.[11] In one case low level of iPTH and FGF23 which were observed during onset of hypercalcemia and acute exacerbation of SLE. Both iPTH and FGF3 reverted back to normal levels after treatment with prednisolone suggesting FGF23 might be involved in pathogenesis of hypercalcemia in SLE.[9] Polyclonal activation of B lymphocytes and elevation of certain cytokines in active SLE could stimulate PTH receptors leading to hypercalcemia and suppress the levels of PTH.[6] Several factors described in pathogenesis of hypercalcemia in SLE believed to work together to cause severe hypercalcemia. It has been observed that hypercalcemia in SLE occurs with acute relapses and remits with remissions. The case presented is a well established case of SLE presented with severe hypercalcemia. Patient had evidence of clinical flare-up during the admission time with hypercalcemia. The potential list of causes of severe hypercalcemia were excluded by appropriate investigations. As SLE was documented as a rare cause of hypercalcemia, it was considered as cause for the hypercalcemia in our case.Patient was given higher doses were steroids were given to the patient, because of flare-up of SLE and hypercalcemia. The excellent and persistent response with serum calcium levels remaining in normal range strongly support the diagnosis of SLE related hypercalcemia. The low IPTH which was low at the time of admission became normal after 3 months period on steroid therapy may indicate that low FGF23 might have played a role in suppressing the IPTH as described in one of the reported cases of SLE related hypercalcemia.

CONCLUSION

SLE is a very rare cause of severe hypercalcemia.SLE should be considered in unusual cases of hypercalcemia where the common potential causes were ruled out. Hypercalcemia in such cases responds to steroids.

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