

Primary Hypoparathyroidism : Psychosis in Postpartum Period

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Abstract

We report a 30 yr old lady who was being treated as postpartum psychosis. Patient had generalized tonic clonic convulsions and was found to have bilateral cataract. Her serum calcium, serum magnesium and serum parathyroid were low. On correction of serum magnesium her PTH did not increase. She was hence diagnosed as primary hypoparathyroidism. On correction of her serum calcium her psychosis improved.

Introduction

Patients with postpartum psychosis present with severe depression, mania, high suicidal drive, schizophrenia symptoms, persisting crying, lack of appetite, self harm and harm to child within three months of delivery.¹ It is diagnosis of exclusion. All known treatable causes of psychosis like metabolic, endocrine, connective tissue disorder, space occupying lesion of brain, nutritional deficiencies should be ruled out before labeling patient as postpartum psychosis. Presence of rash, pallor, goiter, arthritis, convulsion, raised ESR, anemia, active urinary sediments point toward systemic cause of psychosis.



Fig. 1 : Transverse leuconychia



Fig. 2 : Bilateral cataracts

Case Report

A thirty year old female G₁P₁A₀ who had delivered two months ago (LSCS) was admitted in psychiatry ward with history of poor verbal output, altered behavior, poor self care, decreased appetite, withdrawal behavior, not caring for and not feeding her baby, decreased communication, self neglect, stool incontinence, apathy, somnolence over the past two months. She was diagnosed as post-partum psychosis and treated with antipsychotics. She developed one episode of generalized tonic clonic convulsions in sleep. She had two similar episodes of

Table 1 : Investigation

Date	3/5/08	7/5/08	3/6/08
Serum calcium (mg/dl)	3.6	8.5	8.4
Serum magnesium (meq/L)	0.5	2.5	2.0
Serum phosphorus (mg/dl)	3.8	3.9	4.0
Parathyroid hormone (pg/ml)	<3	10.5	5.93

calcium 500 mg tid. She followed up after one month with the following reports serum Ca⁺⁺ = 8.4mg/dl, serum Mg⁺⁺ = 2 meq/l, serum Po₄⁻² = 4mg/dl and serum PTH = 5.93 pg/ml. In spite of correction of magnesium to normal levels parathormone did not increase significantly. Patient was therefore diagnosed as primary hypoparathyroidism in view of low serum PTH, low serum Ca⁺⁺ levels. Low serum phosphorus is seen in hypomagnesaemia.² Anemia, low total protein and low serum albumin were probably due to malnutrition.

Discussion

Postpartum psychosis was discovered in 1850. It occurs in 1-2/1000 child birth.¹ It is also referred as puerperal psychosis or post natal psychosis. It is a serious mental illness which involves rapid onset of psychotic symptoms within 3 months of postpartum period.¹ It commonly occurs 2-3 weeks after delivery in four fifth of the cases.³ It is a psychiatric emergency. Etiology of post partum psychosis is radical hormonal changes, dormant bipolar disorders which get activated in post partum period, delivery stress and lack of social and family support. It is treated with antipsychotic medicines, psychotherapy and requires hospitalization. Infanticide rates 4% and suicide rates are 5%.¹ Patients developing post partum psychosis have 50% increased risk of similar episode in subsequent pregnancy. Differential diagnosis includes electrolyte imbalance, hypothyroidism, Cushing syndrome, infection, neoplasm, toxemia of pregnancy. Our patient developed convulsions; she had alopecia, bilateral cataract, and had transverse leuconychia which are pointers of chronic calcium deficiency. Primary hypoparathyroidism

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convulsions in past. In view of convulsions, she was transferred to medical ward. She had no history of fever, tetany, and connective tissue disorder. She was non-diabetic and did not have any major illness in the past. On examination her pulse was 90 beats per minute, blood pressure of 100/60 mmHg, pallor was present. There was no icterus, cyanosis, clubbing, edema feet. She had hair loss. Her hair was sparse, thin and brittle. Nails had transverse leukonychia (Fig. 1). Bilateral cataracts were present (Fig. 2). On central nervous system examination patient was drowsy, not obeying oral commands, rigid in all four limbs, no focal neurological deficit and no neck stiffness. Cranial nerves were normal, deep tendon reflexes were normal, plantars were flexor bilaterally. Patient was diagnosed neuroleptic malignant syndrome which gradually improved on tab bromocriptine 2.5 three times a day for 10 days. But sensorium of the patient did not improve and patient remained drowsy. Her investigation showed Hb =7.3 gm%, T.C =14300 cells/cmm, neutrophil =85%, lymphocytes =15%, platelet count =3.83 lac/cmm, MCV = 79fL, MCHC = 34gm/dl, MCH = 26 pg, NA^+ = 135meq/L, K^+ = 3.3meq/L, ESR=123 mm at the end of one hr, total protein= 4.1gm%, serum albumin= 2.1gm%, serum Ca^{++} = 3.6mg/dl, serum PO_4^{-2} = 3.8mg/dl, corrected Ca^{++} was 5.2 mg/dl, serum Mg^{++} = 0.5meq/l, serum PTH <3pg/ml (N =9.5 to 75 pg/ml), ECG showed prolonged Qt interval = 0.51. The blood glucose, serum cortisol and thyroid function test were normal. 24hour urinary magnesium = 40 mg/24hr (N = 24 to 255mg/24hr), 24hr urinary Ca^{++} = 6.9mg/24hr (N <40mg/24hr). Other investigation like renal function test, liver function test and arterial blood gas were within normal limits. VDRL and HIV test were negative. MRI brain was normal. Cerebrospinal fluid examination showed protein =33mg/dl, sugar =41mg/dl, lymphocytes =4 cells, CSF ADA =normal. CSF culture = negative. CSF did not show any capsulated yeast. In view of low serum calcium, low serum magnesium, she was treated with 5 ampoules of (10%) calcium gluconate in one pint of 5% dextrose over 5hrs for 5 days, IV magnesium sulphate (50%) 4gm per day for 3 days, IM inj vit D 6 lac units following which her serum Ca^{++} improved to 8.5 mg/dl and serum magnesium improved to 2.5 meq/l. Serum parathyroid hormone was 10.5pg/ml on 4th day. This mild and insignificant rise in serum PTH was attributed to correction of magnesium, as magnesium is necessary for formation and secretion of parathormone. Patient improved clinically. Her sensorium improved and she did not get any further convulsions in follow up. Patient was discharged on tab

is a condition in which there are low circulating levels of parathormone due to decreased production of PTH. Etiology of hypoparathyroidism includes neck surgery, radiation, autoimmune process, infective, infiltrative disorder. It can present as a part of autoimmune Polyglandular insufficiency type 1 in which antibodies to parathyroid and antibodies against calcium sensor receptors are present. Polyglandular autoimmune syndrome type 1 starts in childhood and is characterized by mucocutaneous candidiasis, hypoparathyroidism and adrenal insufficiency.⁴ Hypoparathyroidism associated with defective development of both the thymus and parathyroid gland is termed as Digeorge syndrome.⁵ Hypoparathyroidism presents with features of hypocalcaemia like muscle spasm, tetany, paresthesia, seizures, laryngospasm, psychosis, confusion, intestinal cramps, prolong QT on ECG and bradyarrhythmia. Calcium ion acts as second messenger and helps in release of neurotransmitter at pre synaptic terminal. Hypocalcaemia results in increased central and peripheral neuronal excitability because of increase neuronal membrane permeability to sodium ions, also it causes changes in cellular enzyme activities. Chronic hypoparathyroidism presents with cataract, mental retardation, tooth malformation, heart failure, and intracranial calcification.⁶ Diagnosis of primary hypoparathyroidism is supported by low serum calcium, raised serum phosphorus, low serum PTH in absence of renal failure and intestinal malabsorption. Treatment is calcium and vitamin D supplementation.⁵ Secondary hyperparathyroidism is common in pregnancy, our patient presented with primary hypoparathyroidism in pregnancy.

References

1. Sadock BT, Sadock V A : Psychiatry and Reproductive medicine. Synopsis of psychiatry 10th edition 2007;30:865-866.
2. R Singh, MH Bhatt, A Bhansali : Hypomagnesemia masquerading as hypoparathyroidism. *J Assoc Phys Ind* 2006;54:411-412.
3. PL Rossman, RM Vock: Postpartum tetany and psychosis due to hypocalcemia. *California Medicine* 1956;85:109-193.
4. SV Trivedi, Chitra Joshi, K Amichandwala : Polyglandular autoimmune syndrome type 1. *J Assoc Phys Ind* 2004;52:681-682.
5. John T Potts Jr : Diseases of parathyroid gland and other hyper and hypocalcemic disorders. Harrison principle of internal medicine 14th edition. 354:2227-2246.
6. RP Gupta, RA Krishan, S Kumar : Rare cause of heart failure – Primary hypoparathyroidism. *J Assoc Phys Ind* 2007;55:522-524.