

## Case Report

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# Acute acalculous cholecystitis: an unusual presentation of vitamin B12 deficiency

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## ABSTRACT

Paediatric cases of acute acalculous cholecystitis (AAC) are due to infectious diseases or immune mediated disorders. Hereby, we present a case of 6 months old infant who presented with excessive crying with irritability and tenderness over right hypochondrium region with a palpable liver. Investigations revealed AAC and anaemia. In the work-up of anaemia, vitamin B12 deficiency was diagnosed and after giving treatment with vitamin B12, anaemia resolved as well as AAC. Through this case report we intent to emphasize the possibility of vitamin B12 deficiency presenting as AAC.

**Keywords:** Acute acalculous cholecystitis, Vitamin B12 deficiency, Immune mediated disorders

## INTRODUCTION

Vitamin B12 deficiency is considered rare in infants. It is only seen in infants of breastfeeding mothers who themselves are vitamin B12 deficient. Vitamin B12 deficiency leads to accumulation of homocysteine and methylmalonic acid and causes the onset of haematological, neurological and psychiatric manifestations.<sup>1</sup> Vitamin B12 is found mainly in animal products such as meat, egg, fish and milk. Inadequate dietary intake of animal foods is the important cause of Vitamin B12 deficiency in infants and children.<sup>2,3</sup>

Data suggests an incidence of vitamin B12 deficiency as 32%, folate deficiency as 6.8%, and combined deficiency as 20% in north Indian mothers.<sup>4</sup> Clinical features of vitamin B12 are hyperpigmentation of skin on knuckles, hepatomegaly and splenomegaly (30-40%). Neurological signs include developmental delay and regression of milestones.

Unusual presentations of Vit B12 are cerebral atrophy and encephalopathy presenting as infantile spasms<sup>6</sup> or life-

threatening conditions like haemolytic anaemia, pancytopenia or bleeding in approximately 10% of cases.<sup>5,7</sup>

## CASE REPORT

A 6-month old infant presented with excessive crying, irritability and poor oral intake for 2-3 days. The child was full term baby with uneventful neonatal period. He was exclusively breastfed, developmentally normal, and immunized for age.

On examination, he was afebrile and hemodynamically stable. He had tenderness over right hypochondriac region with palpable liver and mild hypotonia. Investigations revealed haemoglobin (Hb) of 9.8 gm/dl, total leucocyte count (TLC) of 10,060 with normal liver function test, amylase and lipase. Ultrasound abdomen showed mild hepatomegaly, grossly distended gall bladder (GB), increased GB wall thickness & echogenic biliary sludge. Findings were consistent with AAC. Computed tomography (CT) scan confirmed AAC.

As we noticed a gradual drop in Hb from 9.8 to 8.9 gm/dl over a period of 4 days during hospital stay, work-up for haemolytic anaemia was done along with work up for AAC. Thyroid function test, Hb electrophoresis and G6PD was normal. Peripheral smear was showing hypochromic anisocytic RBC's with mainly microcytosis with target cells and elliptocytes. No evidence of sickle cell, spherocytes, acanthocytes or burr cells. No evidence of nucleated RBC's polychromasia or schistocytes in these smears.

The most common causes of AAC are either infection, immunological or haemolytic. Except for gradual fall in Hb, work up for haemolytic anaemia was negative. During evaluation of anaemia, child was found to be deficient in Vitamin B12 (< 83.0 pg/ml) with elevated serum homocysteine (49.56 micromol/L). Further workup was done for Vitamin B12 deficiency like gas chromatography-mass spectrometry (GCMS) and tandem mass spectrometry (TMS) to rule out any inborn error of metabolism which was inconclusive.

The cause of vitamin B12 deficiency in our infant was due to low level of maternal B12. Maternal B12 was 198 pg/ml and folic acid was 1.94 ng/ml.

Baby was started on Methylcobalamin as per standard regime. Child improved with treatment with decrease in irritability, improvement in hypotonia, resolution of abdominal symptoms and normalization of USG findings. Hence, we conclude that AAC was a manifestation of vitamin B12 deficiency which was diagnosis of exclusion.

## DISCUSSION

Deficiency of vitamin B12 can lead to megaloblast formation, intramedullary destruction or haemolysis of fragile and abnormal red blood cell precursors as the result of ineffective erythropoiesis secondary to defective DNA and cell maturation. Intramedullary destruction of red blood cells secondary to vitamin B12 deficiency is a well-recognized phenomenon but not very well understood.<sup>8</sup> It is very well demonstrated in vitro that elevated homocysteine levels could be a possible etiology for both intravascular and intramedullary haemolysis in vitamin B12 deficiency.<sup>8</sup> This intramedullary haemolysis in our infant could have caused the gall bladder sludge and the pain of cholecystitis. Hepatosplenomegaly could be due to extramedullary erythropoiesis.<sup>9</sup> AAC is an inflammation of gall bladder, which does not appear to be associated with the presence of gallstones. AAC is estimated to represent 50% to 70% of all cases of acute cholecystitis in children.<sup>10</sup> The co-existent iron deficiency can mask the typical megaloblastic picture of vitamin B12 deficiency in blood count and peripheral smear which happened in our case. Given the low incidence of haemolysis due to vitamin B12 deficiency, early suspicion and diagnosis in

the appropriate clinical setting is suggested. Counselling done regarding the dietary habits to improve the vitamin B12 sources to the parents.

## CONCLUSION

Vitamin B12 deficiency should be considered in differential diagnosis of some haematological disturbances of the infants. Physical examination and haematological tests always play a crucial role in diagnosing Vitamin B12 deficiency. Unusual finding like AAC can be attributed to vitamin B12 deficiency especially if infant presents with anaemia with acute abdomen.

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