Pyrexia due to Megaloblastic Anemia

Dr. G. Ramanujam¹, Dr. S. Prasanth²

¹Postgraduate, Department of General Medicine, Vinayaka Mission’s Research Foundation – Deemed to be University, Vinayaka Mission’s Medical College & Hospital, Karaikal, India
²Senior Resident, Department of General Medicine, Annapoorana Medical College and Hospital, Salem, India

Abstract: Deficiency of vitamin B12 and/or folic acid as a cause of pyrexia, though known, is rarely reported in literature. We report a case in a 39 year old male, alcoholic who presented with fever for 6 month and was diagnosed to have megaloblastic anaemia secondary to vitamin B12 and folate deficiency. The pyrexia subsided following the intramuscular injection of vitamin B12 and oral folic acid administration.

Keywords: Megaloblastic anemia, Pyrexia, Vitamin B12, Folic acid

1. Introduction

Megaloblastic anaemia are a group of disorders which are most commonly caused by nutritional deficiencies of either vitamin B12 or folate or both, inherited disorders of DNA synthesis or following certain drug therapy. Megaloblastic anaemia rarely may be a cause of pyrexia which may be difficult to differentiate from pyrexia of unknown origin even after exhaustive laboratory investigations.

2. Case Report

A 39 year old male presented with complaints of fever, which is on and off for about 6 months duration. It is associated with generalized weakness, easy fatigability and loss of appetite. There was no history of burning micturition, arthralgia or skin rash. There was no history of recent travel to malarial endemic zone or exposure to any patient suffering from communicable diseases, eg. TB, etc...Clinical examination revealed a pulse rate of 110 per minute, blood pressure of 120/70 mmHg and oral temperature of 101° F. The patient was a known alcoholic and smoker for past 4 years. He had moderate pallor and mild icterus. There was no cyanosis, clubbing, pedal edema, generalized lymphadenopathy, dyspnoea or skin rashes. Examination of cardiovascular, respiratory, abdomen and nervous system examination were within normal limits. X-ray chest was within normal limits and ultrasound abdomen revealed no significant abnormalities. Routine hematological evaluation revealed low hemoglobin 6gm%. Low hematocrit 18%. Low total leucocyte count 4000cells/cumm. Low total platelet count 100,000 cells/mm. high reticulocyte count 3.5%. high mean corpuscular volume 115fl. Peripheral smear showed pancytopenia with a moderate degree of anisopoikilocytosis and hyper segmented neutrophils. Megaloblastic anemia was confirmed biochemically by low levels of serum vitamin B12 59.6pg/ml (reference : 180-900), low folic acid 3.9 ng/ml (reference 4-24) and markedly elevated serum lactate dehydrogenase 7500 IU/l (reference 225-420) In view of the positive laboratory investigations pointing towards megaloblastic anemia, the patient was started on injection vitamin B12 1000 ug IM and folic acid 5 mg oral daily for about 6 months, the patient improved symptomatically. After being prescribed vitamin B12 and folic acid supplements, following which the patient was discharged in a stable condition. Routine followup two months showed normalization of vitamin B12 and folate levels as well as improvement in hematological parameters (hemoglobin 12gm% : mcv : 87 fl ) without any febrile episode.

3. Discussion

Dramatic response to nutritional supplements in our case suggests that the pyrexia was attributable directly to megaloblastic anaemia secondary to vitamin B12 and folate deficiency rather than anything else, as was ruled out by appropriate available diagnostic modalities. The exact cause of fever in megaloblastic anaemia is unknown and at present, seems more hypothetical rather than conclusive. An association of pyrexia and megaloblastic anaemia appears to be caused, whereas in other type of anemias, it seems more coincidental. Megaloblastic anaemia is a pannymelosis characterized by hypercellular marrow and ineffective haemopoiesis, premature destruction of haemopoietic precursors possibly releases intracellular substances which might function as systemic pyrogens, as was suggested by the researches, dramatic response to B12 and/or folate supplementation (within 72 hrs) strongly supports the above said hypothesis alternatively. The defective oxygenation at the thermoregulatory centre of the hypothalamus might be the explanation for pyrexia. However, lack of correlation between neurological manifestation and pyrexia in megaloblastic disease does not support this theory. Moreover studies have also shown that a rise in temperature might cause depletion of folate stores, both in red blood cells and serum, leading to disturbance of folate metabolism. So whether pyrexia is the cause of folate deficiency or vice versa is yet to be fully understood.

4. Conclusion

All patients presenting with pyrexia, megaloblastic anemia and cytopenia should be carefully evaluated for possible vitamin B12 and folate deficiency in order to prevent delay in diagnosis, initiate appropriate curative treatment and unnecessary use of antibiotics and other empirical medication.

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References