Correspondence

Vitamin B<sub>12</sub> Deficiency Presenting as Pyrexia

Sir,

Negi and colleagues, in their case report “Vitamin B<sub>12</sub> Deficiency presenting as pyrexia” (JAPI, June 2011; 59:379-360), have brought out a very important but often forgotten, cause of pyrexia. We had a very similar case, a 32 year old male, with high grade fever and anorexia since 15 days. There was pallor and bald tongue but no knuckle hyperpigmentation. His investigations were also very similar, except for MCV which was 80 fl. There was no evidence of blood loss, gastroscopy was normal and serum iron/TIBC studies were within normal limits. Fever did not respond to antibiotics and antimalarials. Serum B<sub>12</sub> levels were 92 pg/ml (normal 140-180 pg/ml). Bone marrow aspiration and trephine biopsy showed a cellular marrow with megaloblastic picture. However, after 2 days of intramuscular cyanocobalamin, he became afebrile, and remained so, thereafter.

The incidence of pyrexia in megaloblastic anaemia has been reported about 40-65%.<sup>1,2</sup> The authors have mentioned certain hypotheses for the causes of pyrexia in megaloblastic anaemia – defect in oxygenation to the temperature regulatory centre and increased activity within the bone marrow. We would like to highlight the fact that the commonest cause of fever in megaloblastic anaemia is still infection, to which the individual is reverted after specific therapy.<sup>3</sup> Hence, while awareness of deficiency (but not so with folate deficiency). So, bacterial killing is reduced and abnormal; it is reverted after specific therapy.<sup>3</sup> Hence, while awareness of this cause of pyrexia and the testing for folate and B<sub>12</sub> should be emphasized, the search for infection is equally imperative and it cannot obviate the need for further investigations and antibiotic use.

References


Kavita Krishna

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Reply from Author

Sir,

In response to comments by Dr. K. Krishna on our article “Vitamin B12 Deficiency presenting as Pyrexia”. We would like to clarify the point; the commonest cause of fever in megaloblastic anaemia is still infection. We fully agree with this statement, in our case also we have ruled out infective as well as inflammatory cause of pyrexia.

Non-ST Elevation Myocardial Infarction in Organophosphorous Compound (DEMETHOATE) Poisoning

Sir,

Organophosphorous compounds are possibly the most widely used insecticides world-wide and are cause of more than 75% of acute poisoning in hospital practice in developing countries.<sup>1</sup> Cardiac complications that often accompany poisoning with these compound may be serious and fatal<sup>2</sup>.

Here we report a 23 years old male who ingested organophosphorous compound (DEMETHOATE) (confirmed by original container brought)and was seen within an hour after ingestion. At admission he had in altered sensorium. On examination frothing secretions were present in mouth and nostrils, pupil were bilaterally contracted and fasciculations were present but there was no cyanosis. His pulse rate was 68/Min, blood pressure was 118/80 mm of Hg and RR was 14/Min. There were basal crepts on both sides. After admission patient was investigated and treated with atropine and PAM in usual doses and other supportive measures like-Antibiotics, Bronchodilators and High flow oxygen but no assisted ventilation. Cholinesterase level detection facility was not available. All investigation including ECG on 1st day were normal.

On the second day ECG showed ST segment coving with T wave inversion in inferior leads (II, III avF) and chest leads V<sub>1</sub> – V<sub>4</sub> showed deep T wave inversion, 2D echo showed hypokinetic anterio septal wall of Left Ventricle, LVEF 57% and Right Ventricle diastolic dysfunction stage I. On biochemistry,cardiac markers CPK MB was 212 u/l and Trop I-0.26 was ng/ml. On 7th day ECG reverted to normal and patient was discharged.

Soon after exposure to organophosphorous compound signs and symptoms develop in patient with significant poisoning and are muscarinic and nicotinic. Major feature of muscarinic crisis include vomiting 97%, pin point pupil 66%, salivation 62%, increased bronchial secretion 21% and nicotinic effect are characterized by Tachycardia 24%, hypertension 10%, muscles fasciculation 1.8% and mental confusion 43%.<sup>1</sup>

The cardiovascular effect of organophosphorous compound reflect net result of excitatory and inhibitory actions of accumulated acetylcholine (ACh) at ganglionic, medullary, vasomotor and cardiac centres. These effects are further compounded by hypoxia, local release of catecholamine and disturbances on ion transport. Anand S. et al explained that in organophosphorous compound poisoning there are 3 phases of cardiac toxicity (1) a brief increased sympathetic tone (2) prolonged period of parasympathetic activity (3) QT prolongation followed by VT.Both sympathetic and parasympathetic overactivity, hypoxia, acidosis, dys电解trolytemia and direct toxic effect of compound damaged myocardium.<sup>2</sup> Due to myocardial muscular dysfunction the activity of Biochemical Marker’s is increased<sup>1</sup> and ECG changes appear, which have been reported in past.<sup>1,2</sup>

References

2. Saadeh AM, Farsakh NA, AL-Ali MK. Cardiac Manifestation of


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**Resurgence of Complicated Malaria Associated with Severe Thrombocytopenia in a Tertiary Care Hospital in Delhi**

Sir,

The last few months have seen a resurgence of complicated malaria caused by *P. vivax* infection. This is amidst the dengue epidemic which engulfs Delhi in months of Aug-Nov each year and has become a major public health problem. There have been earlier case reports especially by Kochar et al² wherein complications associated with Vivax malaria have been reported. Last published case series were way back in JAPI in year 2004.³

We hereby submit a preliminary report of 30 cases of *P. vivax* malaria with various systemic complications especially severe thrombocytopenia, admitted in Medicine ward at Lady Hardinge Medical College and associated hospitals in the month of August and September 2011.

30 patients of acute febrile illness averaging 3-7 days were diagnosed to have *P. vivax* malaria based on peripheral blood smear and rapid blood tests to differentiate it from *P. falciparum*. Serological tests to rule out other common causes of thrombocytopenia with acute febrile illness specially dengue fever and enteric fever (serology) were carried out.

*Plasmodium vivax* is widely believed to be incapable of cytoadherence and microvascular sequestration and thus not non-sequestration related complications are known to occur in *P. vivax* i.e. anemia and thrombocytopenia. The severe manifestations include cerebral malaria, hepatic and renal dysfunction, ARDS, severe anemia, pulmonary edema and hemoglobinuria. These observations are based on a number of single case reports from India and South asian countries. Recently two large series of severe *P. vivax* malaria were reported from Indonesia and Papua New Guinea.³

In this prospective observational ongoing study, we report the clinical, hematological and biochemical profile of 30 consecutive patients of complicated *P. vivax* were studied at LHMHC.

The most common complication observed were hematological. All the 30 patients presented with fever and 100% (n=30) had thrombocytopenia of which 73.3% had a platelet count of less than 50000. Anemia was seen in 10 patients but only 2 had severe anemia i.e. Hb<5 gm%.

Although the patients had thrombocytopenia but none of them had clinically significant bleeding from any site, none requiring transfusion.

83.3% of all the patients had organomegaly in the form of hepatomegaly, splenomegaly or both. Jaundice was present in 40% (n=12) of patients while hepatic dysfunction (transaminits) was present in 33.3% of the total cases. In the earlier case reports from India, hepatic dysfunction and jaundice were the commonest presentations. One patient each had ascites and ARDS. Leucopenia was present in 20 patients i.e. 66.6% and only 2 patients had leucocytosis. Anemia viz. Hb<10 gm% was present in 10 patients but severe anemia was seen only in 2 patients. Renal dysfunction was noted in 10 patients i.e. 36.3% but none had hemoglobinuria.

The gametocytes and trophozoites of *P. vivax* were seen in a thick and thin peripheral blood smear of 25 patients. The rapid blood test was negative for falciparum in these patients but positive for pan malarial antigen. All patients were treated with artesunate or quinine according to WHO guidelines 2010 and the hematological parameters started improving by day 3 and recovered in more than 90% cases by day 7. Rest recovered by day 10 and there were no mortalities.

**Discussion**

The recent few years have seen outbreaks of fever with thrombocytopenia in Delhi and NCR region aptly highlighted by the Media.

This case report serves to highlight the following points:

1. To differentiate cases of severe thrombocytopenia due to malaria from those attributed to viral fever esp. Dengue since the former is a treatable cause and easily diagnosed by bedside kits freely available in Delhi and NCR.

2. The etiology of thrombocytopenia in all i.e. 100% of cases may be due to emergence of new strains of *P. vivax* targeting other blood cells (platelets and WBCs) besides RBCs. This needs further investigation.

3. To follow the loco-regional patterns and epidemiological trends of complicated vivax malaria in the coming years data from which may be of public health importance.

**References**


2. UM Jadhav, VS Patkar, NN Kadam. Thrombocytopenia in Malaria - Correlation with Type and Severity of Malaria. *JAPI* 2004;52:615-618.


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