Vitamin B12 deficiency presenting with hyperpigmentation and pancytopenia

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ABSTRACT

Hyperpigmentation with generalized weakness may be the initial presentation of several systemic conditions. Vitamin B12 deficiency can present very rarely as hyperpigmentation. Association of pancytopenia at this stage is even rare. Early identification and management can prevent various neurological complications. Here, we report a rare case of Vitamin B12 deficiency presenting as hyperpigmentation and pancytopenia.

Keywords: Hyperpigmentation, pancytopenia, Vitamin B12 deficiency

Introduction

Hyperpigmentation with generalized weakness may be the initial presentation of several systemic conditions. Vitamin B12 deficiency can present very rarely as hyperpigmentation. Association of pancytopenia at this stage is even rare. Early identification and management can prevent various neurological complications. Here, we report a rare case of Vitamin B12 deficiency presenting as hyperpigmentation and pancytopenia.

Case Report

A 52-year-old male patient presented with fever, weakness, weight loss, breathlessness, and progressive pigmentation. There was no history of diabetes mellitus, hypertension, recent malaria, or chronic drug intake. Family history was not significant. The patient was a vegetarian, not a smoker, and not an alcoholic.

On examination, the tongue was pale [Figure 1]. Hyperpigmentation in the form of macules present over the palm [Figure 2] and axilla [Figure 3]. Hemogram showed hemoglobin of 4.8 g%, total cholesterol 2600, platelets were 56,000, red blood cell (RBC) 1.6 million, packed cell volume 13.1, mean corpuscular volume (MCV) 11.2, mean corpuscular hemoglobin 42.8, mean corpuscular hemoglobin concentration 36.6, and erythrocyte sedimentation rate was 120/1st h. Urine showed albumin 1+, pus cells 7–8/HPF, epithelial cells 2–3/HPF, and RBC positive. Urine culture sterile. Peripheral smear showed RBC mild anisocytosis, poikilocytosis, and macrocytosis with hypochromia, tear drop cells, macroovalocytes with decreased white blood cell. Bone marrow was hypercellular, increased erythropoiesis with micronormoblastic, and mild megaloblastic change. Dengue antibodies were negative. Widal test negative. Plasmodium vivax and Plasmodium falciparum were negative. HIV and HCV were nonreactive. Thyroid-stimulating hormone was 2.93. Chest X-ray was normal. Ultrasound abdomen showed hepatomegaly and calcified granuloma of the liver.

Discussion

Vitamin B12 deficiency is defined as a plasma concentration of <148 pmol/L (200 pg/ml). The prevalence of Vitamin B12 deficiency varies from 3% to 5% in the general population and 5% to 20% among people older than 65 years. Inadequate intake, pernicious anemia (low intrinsic factor), and food-bound cobalamin malabsorption in part due to gastric atrophy are

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the main causes of low serum Vitamin B12, especially in poor populations. In the present case, the cause of Vitamin B12 deficiency was decrease intake with low intrinsic factor which presented with fever, weakness, weight loss, breathlessness, and progressive pigmentation. The common systemic features reported were fatigue, glossitis, weight loss, and anorexia that were found in 34%, 31%, 27%, and 22% of cases, respectively. The cutaneous manifestation of Vitamin B12 deficiency is skin hyperpigmentation, vitiligo, hair changes, and recurrent angular stomatitis. Hyperpigmentation of the extremities, especially over the dorsum of the hands and feet, with accentuation over the interphalangeal joints and terminal phalanges associated with pigmentation of the oral mucosa is characteristic of Vitamin B12 deficiency. It is suggested that deficiency of Vitamin B12 causes decrease in intracellular reduction potential that leads to oxidation of the reduced glutathione and decrease in GSH/GSSG ratio. The epidermal melanocytes are then stimulated to produce melanin as the tyrosinase inhibiting effect of GSH has been diminished. Hyperpigmentation of the skin has been reported only rarely as the presenting manifestation of Vitamin B12 deficiency as found in this patient. There are only three such Indian reports found in Indian literature. In this case, patient presented with hyperpigmentation over the palms and the axilla, which is again a rare site of presentation. The patient also had pancytopenia, which is even rarer association with hyperpigmentation as presentation of Vitamin B12 deficiency. So far, only one case has been reported.

The patient was treated with antibiotics (amoxicillin clavulanic acid and metronidazole) for the fever, started on intravenous fluids with multivitamin (MVI), transfused with 2 units of packed cells one on day 1 and other on day 5. Starting from day 1, he was treated with intramuscular injection of Vitamin B12 (1000 mg) daily for 10 days, then weekly for 1 month, and then monthly for 2 months. Subsequently, the patient had been receiving a MVI tablet daily containing Vitamin B12 (1 mg) and showed improvement in his presentation [Figure 4]. Usually, hyperpigmentation resolves within 3 months of initiation of treatment, in about 87% of such patients. After the treatment, hematologic response began after several days, and the final hematologic landmark was improvement in blood count, including MCV that was normalized by 8th week. Hyperpigmentation also resolved completely by 8 week.
Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest
There are no conflicts of interest.

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