Periodontal findings in a patient with Dimorphic anaemia

Sushama R Galgali,[1] Rajiv Nidasale Puttaswamaiah[2]

ABOUT THE AUTHORS

1. **Sushama R Galgali**

Professor & H.O.D.
Department of Periodontics
V.S.Dental College & Hospital
Bangalore.

2. **Rajiv Nidasale Puttaswamaiah**

Senior Lecturer
Department of Periodontics
V.S.Dental College & Hospital
Bangalore, Karnataka, India.

Corresponding Author:

Rajiv Nidasale Puttaswamaiah

Senior Lecturer
Department of Periodontics
V.S.Dental College & Hospital
Bangalore, Karnataka, India.

Email:
rajivnidasale@yahoo.com

Abstract

Dimorphic anaemia can present itself with deficiency of more than one nutrient. The blood picture shows the presence of morphologically different red blood cells. The cause of dimorphic anaemia has to be established in order to systematically manage the patient. Combined deficiency of iron and folic acid more commonly lead to dimorphic anaemia with clinical features of both forms of anaemia. The aim of this paper is to report a rare finding of periodontal involvement in a patient with dimorphic anaemia.

Key words: anaemia, iron deficiency, folic acid deficiency, red blood cells, periodontal abscess

Introduction

Anaemia is functionally defined as an insufficient RBC mass to adequately deliver oxygen to peripheral tissues. For practical purposes, any of the three concentration measurements (i,e; hemoglobin concentration, hematocrit or RBC concentration) performed on whole blood can be used to establish the presence of anaemia. The RBC concentration is least commonly used in the definition of anaemia[1]

Dimorphic anaemia is the anaemia in which there are two distinct red cell populations. A dimorphic blood film can be seen in several circumstances. It can occur when iron deficiency anaemia responds to iron therapy, after the transfusion of normal blood to a patient with a hypochromic anaemia and in sideroblastic anaemia.[2] Dimorphic anaemia can show not only signs of folic acid deficiency, such as megaloblastic dysplasia, but also signs of severe iron deficiency anaemia. [3]

Iron deficiency is a common nutritional disorder in adult and pediatric patients. The most obvious consequence of iron deficiency is anaemia.[4] Lack of iron in the diet, loss of blood (which can be either slow or rapid loss) or poor absorption from the diet is the causative factor for iron deficiency anaemia. Iron is involved in hemoglobin, myoglobin & cytochrome systems. Its role in the movement of oxygen from the environment to cells such as those of the immune system and its role in cytochrome enzymes impact both innate & adaptive immunity. The neutrophils bactericidal activity is impaired by iron deficiency. In addition, the proliferation of lymphocytes reduced, along with their response to antigens, increasing the risk & severity of infection.[5]

Folic acid deficiency, the most common vitamin deficiency among the B-complex vitamins, may occur secondary to excessive alcohol consumption, pregnancy, lactation, kidney dialysis, liver disease, inadequate dietary intake, gastrointestinal disease or medications that interfere with folate absorption and/or metabolism.[6]

Folate deficiency symptoms first appear in rapidly dividing cells, such as GIT, RBCs & WBCs. RBCs do not develop normally; they become pale and extremely large (megaloblastic) a condition known as megaloblastic anaemia.[6]

Glossitis is usually present in persons with folic acid deficiency. The tongue becomes fiery red and papillae are absent. Marked chronic periodontitis with loosening of the teeth may occur. Folic acid deficiency impairs immune responses and resistance of the oral mucosa to penetration by pathogenic organisms such as candida.[6] We report a case of Dimorphic anaemia showing signs of both Iron deficiency and folic acid deficiency who presented with periodontal findings.

Case Report:

A 21 year old female patient presented with a complaint of multiple swelling in the gums since 3 months. She also complained of fatigue and lethargy. She gave a history of oral prophylaxis that was done 4 months back. Her past medical history revealed visits to gynaecologist with complaint of increased menstrual bleeding. Definite pallor of the skin was noticed indicating some form of anaemia (Fig-1). Conjunctiva, lips and the nail beds also showed paleness. Oral examination revealed pallor associated with the oral mucosa of the mouth & pharynx and the presence of periodontal abscess with respect to upper left first premolar and lower left first molar (Fig-2,3 &4). A draining sinus was also noticed in relation to lower left first molar. Oral hygiene status was good. Gingival inflammation with enlargement was also noticed which did not correlate with the amount of local factors. OPG showed the presence of severe bone loss with respect to 17,24 & 36 (Fig-5). Routine blood investigations were advised. A diagnosis of anaemia was established based on the laboratory findings.

As part of the emergency phase the area was anaesthetized and abscess drainage was carried out. The patient was then referred to the physician for management of her anaemic status. In view of her low haemoglobin concentration the patient was admitted to the hospital and further investigations were carried out. Her blood investigation report was as follows: RBCs 4.02million/mm3, Hb 7gm%, PCV 23.6%, MCV 59 fl, MCH 17.5 pg, MCHC 29.8%, TC 5,800 cells/mm3, DC P70 L30, Blood Urea 13mg/dl, Serum Creatinine 0.9mg/dl. RBS 123mg/dl and Urine sample did not show any presence of Albumin or Sugar. Stool for occult blood and for Ova & cyst gave negative results. Peripheral smear showed the presence of moderate to severe degree of hypochromia with both microcytes and macrocytes giving a picture of Dimorphic anemia. Tear drop shaped cells characteristic of Iron deficiency were also present. Thrombocytosis was also noticed. Ultrasound investigation of the abdomen revealed borderline splenomegaly. Serum Vitamin B12 level was 241pg/ml and Serum Folate level was 4ng/ml. Serum ferritin was measured at 15ng/mL. Results showed a combined Iron deficiency and folic acid deficiency state.

Patient was transfused with 2 units of packed cells and was put on Inj Vitcofol 2mg – 5 injections every alternative days and 5 injections once a week and Cap Vitcofol once a day for 3 months. Patient was re-evaluated 4weeks after the abscess drainage was carried out (Fig-6). Gingival inflammation had reduced, but periodontal pockets were present in relation to 17,23,24,26,36 and 37 which were more than 5 mm. Patient was recalled after 3months for further evaluation & treatment. Repeat laboratory investigations showed that the haemoglobin percentage was 12.9gm%. Periodontal management of the patient included GTR for 24 (Fig-7), Hemisection for 36 (Fig-8) and access flap procedure for first quadrant. Postoperative healing was satisfactory at 3 months recall evaluation (Fig-9).

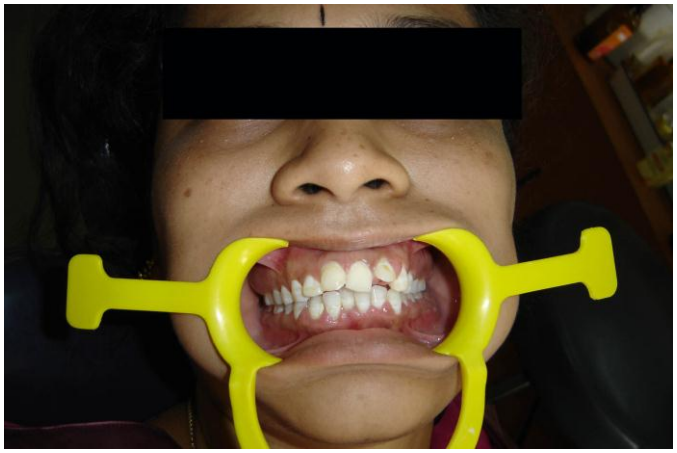

Figure -1 Face showing definite pallor

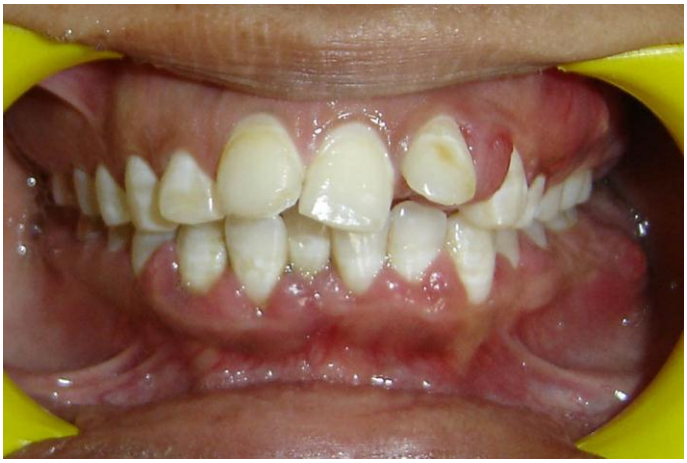

Figure -2 Appearance of the gingiva (front view)

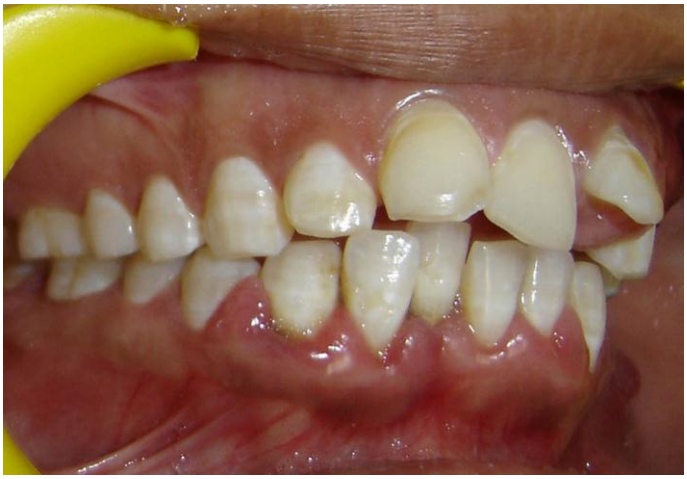

Figure -3 Right lateral view of the teeth and the gingiva

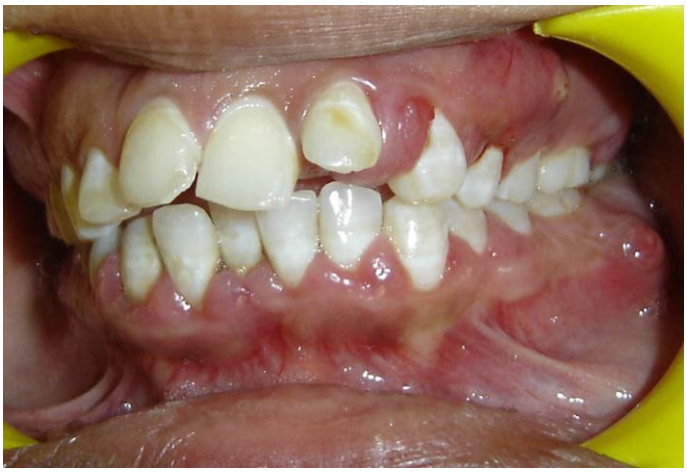

Figure -4 Left lateral view showing the presence of abscess in relation to 24 & 36.

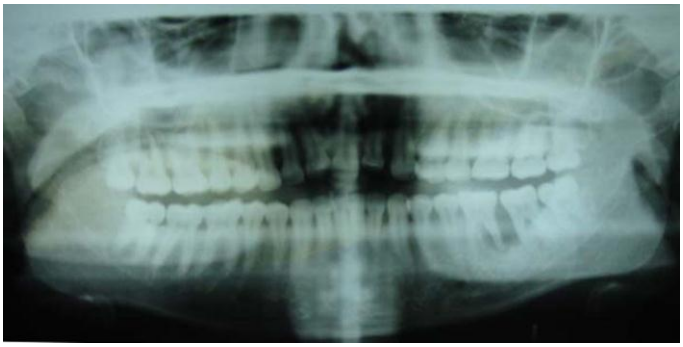

Figure -5 OPG showing severe bone loss in relation to 16. 24 & 36

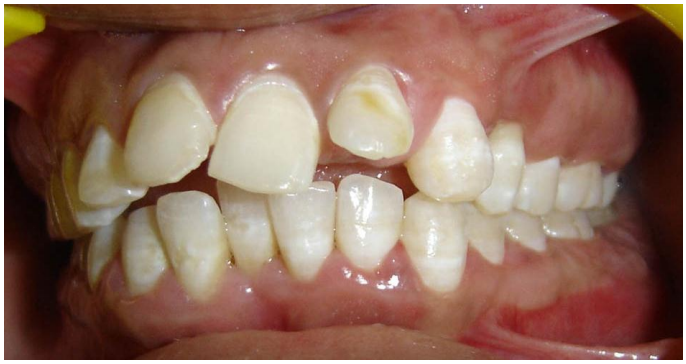

Figure -6 Changes seen at the site 1month post abscess drainage

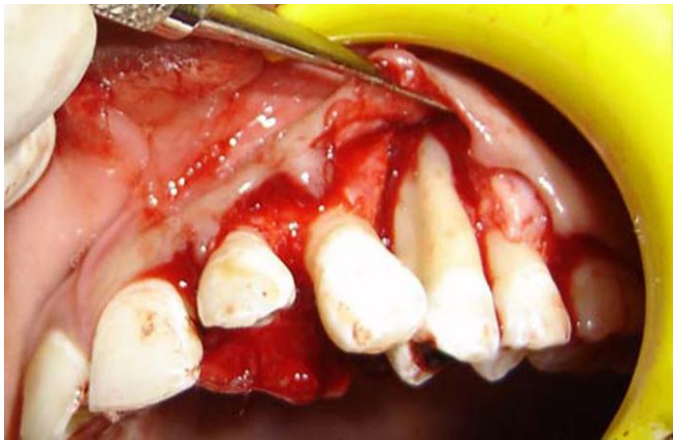

Figure -7 Defect site- after degranulation- where GTR was performed

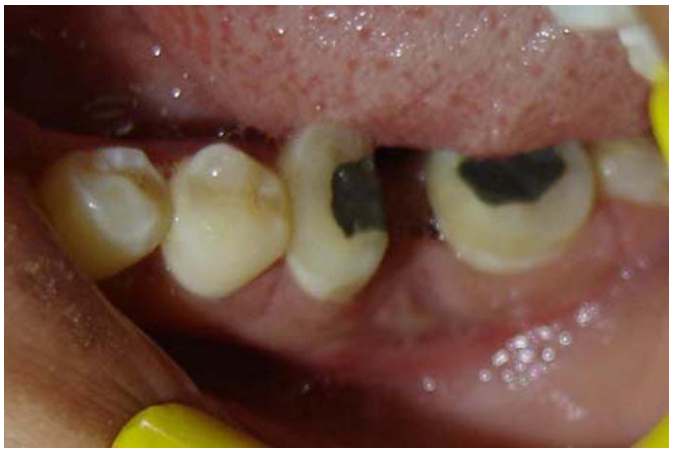

Figure -8 Hemisection performed in relation to 36

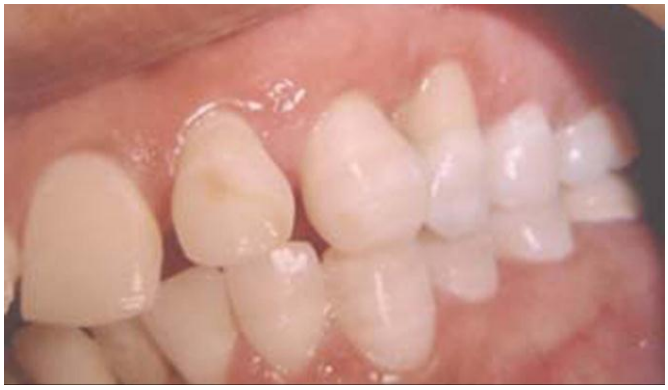

Figure -9 Post-operative healing at the site of periodontal abscess- 3 months

Discussion:

Comprehensive dental care requires dental professionals to assess the general health of their patients and to understand the implications of underlying factors that may impact oral health. One of these underlying factors is the interaction between nutritional status and the immune response to the bacterial challenge to periodontal disease. It is important that dental professionals be able to identify patients at risk for poor nutrition, which may compromise their immune response and place them at higher risk for infection.[5] The signs of paleness of the skin, conjunctiva, nail bed and oral mucosa was characteristic of anaemia.

Haemoglobin concentration of 7gm%, PCV 23.6%, MCV 59fl, MCH 17.5Pg, MCHC 29.8% were suggestive of anaemia. Peripheral smear showed the presence of dimorphic blood picture with both microcytosis and macrocytosis also associated with hypochromia. It also showed tear drop cells which is characteristic of iron deficiency anaemia.

Serum Ferritin level of 15ng/ml, confirmed the presence of iron deficiency. Serum folic acid level of 4ng/ml showed an intermediate folic acid deficiency state. Serum Vitamin B12 level was 241 pg/ml was within the normal limits. This ruled out the possibility of Vitamin B12 deficiency. Thrombocytosis was relative to the anaemic status. Borderline splenomegaly was also present which is commonly seen in anaemia. Stool

for occult blood gave negative result and ruled out the possibility of blood loss through the gastrointestinal system.With all the signs and symptoms and haematological investigations it was diagnosed as Dimorphic anaemia due to Iron and folic acid deficiency.

Studies have indicated that iron deficiency does in fact result in biochemical and morphologic signs of folic acid deficiency, notwithstanding that dietary intake of folic acid is perhaps more than adequate.[3] Chanarin et al. (1965) concluded that the addition of iron to the diet of pregnant women reduced the frequency of megaloblastic anemia and suggested that iron deficiency may in the first instance produce folic acid deficiency signs. Iron deficiency might produce folic acid deficiency signs if an enzyme concerned in the metabolism of folic acid was iron-dependent. Vitale et al.(1965) have demonstrated that the activity of the enzyme glutamate formimino transferase is markedly reduced in livers of iron-deficient rats. A diminution in the activity of this enzyme would explain in part the increase in urinary excretion of FIGLU in rats as well as in humans.[3] Gross et al.(1964) have shown that four of twenty eight children with iron deficiency had signs of folic acid deficiency, both biochemical and morphologic, which could be attributed solely to iron deficiency.[3]

Omer and co-workers (1970) found an abnormality in the distribution of folate in iron deficient patients and postulated that, in iron deficiency, red cells fail to utilize folate. Chanarin et al. (1965) found that the highest rate of megaloblastic anemia in pregnant patients occurred in those who were markedly iron deficient. They suggested that iron deficiency may help to produce folate deficiency. It may be that iron deficiency adversely affects cellular immunity through interference with folate metabolism.[7]

Iron deficiency anemia can be seen as a result of poor dietary intake, loss of blood or impaired absorption. Loss of blood can be slow (bleeding peptic ulcer, colon cancer, hook worm infection, urinary tract bleeding, menstrual bleeding etc.,) or rapid (blood loss during surgeries or severe injuries).

In the case described above the patient gave a history of increased menstrual bleeding, which could be the primary cause for the iron deficiency anaemia.

Though paleness of the oral mucosa was seen changes in the tongue were not observed. Patient presented with periodontal abscess at multiple sites in the oral cavity. Periodontal abscess could have developed because of the decreased immune response seen in both Iron deficiency and Folic acid deficiency. Axelrod (1971) showed a severe defect in the antibody response of white rats made folate deficient.[7]

The cell-mediated immunological system is generally acknowledged to be responsible for resistance to viral, fungal, parasitic, gram-negative bacterial, and mycobacterial infections.[7] Gross et al, showed that both in vivo and in vitro measures of cell-mediated immunity are depressed in megaloblastic anaemia due to folate deficiency. The prompt return of lymphocyte function to normal (as determined by [3 H] thymidine uptake) and the conversion of the great majority of DNCB skin tests to positive after folate treatment strongly suggest that folic acid deficiency is responsible for the observed depression of cell-mediated immunity.[7]

Patient was transfused 2units of packed cells to take care of the emergency situation. She was put on Oral and Systemic folic acid supplements in order to replenish the folic acid level in the body. Since there was presence of active infection in the form of periodontal abscess iron supplements were not prescribed as it is believed that pathogens can sequester iron from the host tissues, which acts as a virulence factor for these organisms.[5] Periodontal treatment was carried out after 3 months of folic acid supplementation, once the Hb% reached 12.9%. Since periodontal treatment consisted of periodontal surgery which leads to blood loss, patient was asked to modify her dietary habits with inclusion of diet rich in iron and folic acid. Blood parameters were evaluated every month till all the haematological parameters had stabilized.

Conclusion:

Dimorphic anaemia has a complex pathogenesis with involvement of more than one deficiency state. Oral lesions are more commonly associated with the deficiency of Iron and folic acid. Dentists should identify these findings and should aid in the diagnosis of the conditions which might have more serious systemic effect.

References:

1. Means RT Jr, Glader B. Anemia: General considerations. In, Greer JP, Foerster J, Rodgers GM, Paraskevas F, Glader B, Arber DA et al., (ed). Wintrobe's Clinical Hematology, 12th edition. Philadelphia, Lippincott Williams & Wilkins ,2009;779-809.
2. Bain BJ. Blood cell morphology in health & disease. In, Lewis SM, Bain BJ, Bates I(ed). Dacie & Lewis Practical haematology, 10th edition. Philadelphia, Churchill Livingstone, 2006; 79-113.
3. Velez H, Restrepo A, Vitale JJ, Hellerstein EE. Folic Acid Deficiency Secondary to Iron Deficiency in Man. Remission with Iron therapy and a Diet Low in Folic acid. Am J Clin Nutr 1966;19: 27-36.
4. Yager JY, Hartfield DS. Neurologic Manifestations of Iron Deficiency in Childhood. Pediatr Neurol 2002; 27: 85-92.
5. Boyd LD, Madden TE. Nutrition, infection and periodontal disease; Dent Clin N Am 2003;47: 337-354.
6. Stegeman CA, Davis JR. Vitamins Required for oral soft tissues & salivary glands. In: "The Dental Hygienist's Guide to Nutritional Care," 2nd ed. St. Louis Elsevier Saunders; 2005: 216-230.
7. Gross RL, Reid JVO, Newberne PM, Burgess B, Marston R, Hift W.Depressed cell mediated immunity in megaloblastic anemia due to folic acid deficiency. Am J Clin Nutr 1975;28:225-232.