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Idiopathic thrombocytopenic purpura presenting as gingival bleeding in a Periodontal Clinic in Benin City, Nigeria

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ABSTRACT

This is a case of Idiopathic Thrombocytopenic Purpura (ITP) which led to continuous intermittent bleeding from the gingival sulcus of 11,21, 32, 34, 35, 44, 45, 36. Intraoral examination showed petechiae and ecchymosis on the lips, buccal mucosa, hard palate and on the ventral surface of the tongue. Petechiae and black patches on the upper and lower limbs led to the suspicion of altered coagulation or bleeding disorder. A full blood count revealed pancytopenia (RBC 2.87X 10^6/ul; WBC 2.5 x10^3/ul and platelet 9x10^3/ul). This emphasizes the significance of a good clinical examination, early diagnosis and referral of such patients to specialists for prompt treatment. This case report will serve as a guide to dentists that not all gingival bleeding are due to plaque accumulation that result in gingival inflammation and that some systemic diseases present with oral manifestations.

Keywords: gingival bleeding, Idiopathic Thrombocytopenic Purpura, pancytopenia

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INTRODUCTION

Abnormal bleeding from the gingiva may indicate a wide range of health conditions and diseases. These health conditions and diseases may be of oral or systemic origin. It is commonly caused by gingivitis and periodontitis but may also be due to dermatoses, erythema multiforme, lupus erythematosus, leukaemia, blood dyscrasias and thrombocytopenia.[1] It is necessary for dentist to properly evaluate any reported gingival bleeding to rule out health conditions and diseases that can become serious, if left undiagnosed

Thrombocytopenia are occur through decreased bone marrow production, increased peripheral destruction and increased splenic sequestration of platelets. Idiopathic Thrombocytopenic Purpura (ITP) also known as primary immune thrombocytopenic purpura and autoimmune thrombocytopenic purpura, is primarily a disease of increased peripheral destruction of platelets as majority of sufferers have antibodies which opsonizes specific platelet membrane glycoproteins resulting in reduced platelet survival by the reticuloendothelial system.

ITP is a haematologic disorder characterized as isolated thrombocytopenia with normal bone marrow without a clinically apparent cause.[2] It is a rare disease with incidence of 66 in 1,000,000 per year in USA. The clinical presentation may be acute with severe bleeding, insidious (chronic) with slow development with mild or no symptoms or recurrent.[3] It is diagnosed by a low platelet count in full blood count after exclusion of other causes of a low platelet count, additional investigations such as a bone marrow biopsy may be necessary in some cases.[4]

ITP causes a characteristic purpuric rash, an increased tendency to bleed and affects both children and adults differently. The peak age of prevalence of ITP in childhood is 2 to 4 years. Children often develop acute form of idiopathic thrombocytopenic purpura after a viral infection, such as mumps, measles or a respiratory infection and this usually has spontaneous resolution within two months in 70%- 80% of children even without treatment. ITP distribution in children is almost equal in both gender [females (48%) and males (52%)]. In adults, the peak age prevalence is 20 to 50 years and often presents as a chronic condition usually persisting for longer than six months and therefore require therapy. This form of ITP is more common in females than males with a ratio of 2.6:1. Recurrent form of the disease has limited data with one study revealing a 6% prevalence with most patients (69%) having just a recurrence[5].

The assessment of the patient includes detailed history to elicit risk factors for HIV and systemic symptoms linked to other illnesses or to medications like heparin, alcohol, quinidine, quinine, sulfonamides) that may cause thrombocytopenia.[6] Admission is usually

necessary for most patients with undiagnosed thrombocytopenic purpura for further evaluation and treatment since ITP is a diagnosis of exclusion.[4] Treatment is not required in adult with platelets counts of >50,000/mm^3 and in many cases of ITP in children as spontaneous remission is common in childhood.[7]

Treatment is indicated in children who have platelet counts < 20,000/mm^3 and minor purpura, and adults with counts <50,000/mm^3 with symptoms such as mucous membrane bleeding, purpura, nose bleeding etc. Treatment is also indicated for those adults with risk factors for bleeding such as hypertension, peptic ulcer disease and vigorous lifestyle and in patients with platelets count < 20,000—30,000/mm^3

Glucocorticoids and intravenous immunoglobulin (IVIg) are the mainstay of medical therapy.[7] Indications for use, dosage and route of administration are based on the patient's clinical condition, the absolute platelet count and the degree symptoms. Consultation with a haematologist will be needed prior to starting therapy. Life-threatening bleeding in known ITP patient requires conventional critical care interventions, high-dose parenteral glucocorticoids and IVIg, with or without platelet transfusion are appropriate. Platelet transfusion is indicated for controlling severe haemorrhage, done after screening, grouping and cross-matching. Platelet survival is said to increase if the platelets are transfused immediately after IVIg infusion. Prednisolone is said to be the drug of choice for all adult patients with platelet counts < 50,000/mm^3 while methylprednisolone is used as an alternative glucocorticoids of choice for all patients with severe, life threatening bleeding or children with platelet counts < 30,000/mm^3. Administration of IVIg may temporarily increase platelet counts in some children and adults with ITP, and therefore may be considered if situation requires a rapid temporary rise in platelet count. However IVIg is a drug of choice for severe life threatening bleeding or for children with platelet counts < 20,000/mm^3 with minor purpura. It can be used alone or in combination with glucocorticoid therapy.

Clinical trials have shown promise for agents that directly stimulate platelet production, such as thrombopoietin (TPO) receptor- binding agents like Elthrobopag. They are usually indicated for thrombocytopenia associated with chronic ITP in patients experiencing inadequate response to corticosteroids, Immunoglobulins or splenectomy.

The objective of this study was to present a case of undiagnosed Idiopathic Thrombocytopenic Purpura that presented to Periodontology Clinic of University of Benin Teaching Hospital, Benin City, Nigeria with complaint of non-painful spontaneous gingival bleeding is as to increase index the of suspicion of systemic condition on gingival bleeding complaints among dentists.

CASE DESCRIPTION

A 41 year old lady presented to the Periodontology Clinic of University of Benin Teaching Hospital on account of gingival bleeding of 4 weeks duration. The bleeding was spontaneous, generalized gingival oozing with no associated pain. She also had intermittent nose bleeding during the same period, but menstrual flow was normal of 2 days duration. Prior to the bleeding episode she claimed to have taken some medication for dermatological problems, the names and indication of the drugs was not known. There was no history of fever, weakness or night sweats. On examination there were petechiae spots on the palate, gingival, cheek and the ventral surface of the tongue. Dark spots were also noticed on the skin of the upper and lower limbs. Impression of gingival bleeding secondary to drug induced thrombocytopenia was queried. The free gingival margin was cleaned with sterilized gauze using normal saline and patient was investigated for full blood count (FBC) and differentials. The FBC revealed pancytopenia with WBC reading 2.5 x 10^3/ ul; RBC was 2.87 x 10^6/ ul and platelet count was just 9 x 10^3/ul. Patient was placed on haematinics and referred to the haematologist. Following the patient's presenting complaints and clinical examination, the haematologist arrived at a provisional diagnosis of Idiopathic Thrombocytopenic purpura (ITP). A repeat FBC by the haematologist revealed WBC to be 3.7 X10^3/ul; platelet count of 12 x 10^3/ul; while RBC came up to 4.11 x 10^3/ul and diagnosis of bicytopenia was made and ITP queried. Patient was to be admitted for further investigations, but she refused and was therefore sent home on oral prednisolone 15mg thrice daily and haematinics, with a week recall visit. Patient was later admitted into the ward and the following investigations were carried out, FBC and peripheral blood film, bone marrow aspiration, retroviral screening and fasting blood sugar and Coomb's test. All the test came out negative while FBS was within normal range. The patient was transfused with 2 pints of fresh whole blood while still on oral prednisolone, Tranexamic acid and haematinics. Following the blood transfusion the platelet count increased to 34,000 x 10^3/ul and WBC was 5,400 x 10^3/ul. The prednisolone was tapered down to 5mg twice daily, and the patient was discharged home. The RBC and WBC were normal on discharge. The

platelet count continued to increased to 78 x 10^3/ul a week post discharge when reviewed in the out-patient visit and subsequently rose to 112 x 10^3/ul on recall visit the following week. No further bleeding has been reported since then. The patient had since remained asymptomatic more than 12 months ago.

DISCUSSION

Gingival bleeding which can either be provoked or spontaneous depending on the severity and progression of the periodontal disease is a common presenting complaint in Periodontology Clinic. Systemic causes of gingival bleeding are rarely seen in Periodontology Clinic. This made this case report very interesting and unique and is expected to increase the index of suspicion of systemic condition on gingival bleeding presentation to dentists. Gastro-intestinal bleeding, menorrhagia, intracranial bleeding which are uncommon findings were also not found in this patient, however gingival bleeding and epistaxis which are said to be less common manifestations were the presenting complaints of this case report.[8]

Clinically severe bleeding does not seem to occur in ITP patients unless the platelet count is less than 10,000^3/mm and physicians generally start treatment when the platelet count is below 30,000^3/mm, the baseline FBC check of our case showed platelet count of 9,000^3/mm which must have been the reason why the patient experienced the gingival bleeding and epistaxis.[3] The diagnosis was based on exclusion of other likely causes of thrombocytopenia such as HIV/AIDS, diabetis mellitus, for which the patient was screened for. Other tests included Coomb's for HbAg antibodies, Hepatiitis B and C screening. Bone marrow aspiration was also carried out, all the investigations came out negative. The investigations carried out complied with the accepted protocol of diagnosis of exclusion in the management of ITP.

The patient under review did well on the combination of steroid therapy, anti fibrinolytic drugs and transfusion of whole blood. The platelet count on discharge was 34,000^3/mm, with no further symptoms. At recall visit a week after discharge the platelet count was 78,000^3/mm and it rose to 112,000^3/mm two weeks after discharge from the hospital. Though, there was room for splenectomy, IVIg, IVRhIg and chemotherapy, this patient did not require them since she steadily improved on the steroids and other treatment given to her. Usually it is when the first line of management does not improve the platelet count that intravenous Immunoglobulins are used.

CONCLUSION

A case of ITP that first presented with gingival bleeding to the Periodontology Clinic and did well on steroids and blood transfusion. The dentist can be the first to diagnose certain systemic diseases of grave importance. This also shows the need for interdisciplinary management of patient to provide maximum service delivery to our patients and the community at large.

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