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The role of oral hygiene in a patient with idiopathic thrombocytopenic purpura

Abstract: Objective: Idiopathic thrombocytopenic purpura (ITP) is an acquired disease of children and adults defined as isolated thrombocytopenia with no clinically apparent associated conditions or other causes of thrombocytopenia. Oral manifestations are gingival bleeding, petechiae, mucocutaneous bleeding and haemorrhage into tissues. Case description and results: An 11-year-old Turkish female was referred to the periodontology clinic by the paediatric haematologist for the treatment of spontaneous gingival bleeding, gingival oedematous enlargement and hyperaemia. She was diagnosed as chronic ITP 6 months ago and she was told not to brush her teeth. She was also complaining with dull pain and oral malodor. She was motivated for oral hygiene and after consulting with paediatric haematologist, under the proper circumstances, dental plaque and calculus were removed. Healing was uneventful. Conclusion: Oral hygiene motivation and dental plaque control is crucial in order to prevent gingival bleeding, inflammation and severe periodontal diseases in patients with haematologic disorders. Understanding of the oral findings is essential in the management of patients and close cooperation between patients’ dentist and haematologist is needed. Dental consultation is essential for diagnosis and improvement of medical conditions. It is possible to obtain adequate oral hygiene with limited performance and haematologic disorders are not handicaps for dental/periodontal procedures under the proper circumstances.

Key words: gingival bleeding; idiopathic thrombocytopenic purpura; oral hygiene
Introduction

Idiopathic thrombocytopenic purpura (ITP), also known as primary immune thrombocytopenic purpura, is a haemorrhagic disorder of the blood and an acquired disease of children and adults, defined as isolated thrombocytopenia with no clinically apparent associated conditions or other causes of thrombocytopenia (1). ITP is a disorder in which antibodies are generated against platelet antigens, resulting in accelerated platelet destruction (2). It is characterized by severe acquired thrombocytopenia and mucocutaneous bleeding due to opsonization and destruction of platelets by circulating antibody (3).

No specific criteria establish the diagnosis of ITP; however, while the diagnosis is based primarily on the history, according to American Society of Hematology Guidelines (4), and in British Guidelines, it relies on exclusion of other diagnoses (2, 5). The diagnosis of ITP is one of exclusion and is based on the history, clinical examination, a full blood count and inspection of the peripheral blood film/smears (2, 3).

Idiopathic thrombocytopenic purpura develops due to severe bacterial or fungal sepsis, hypersplenism, disseminated intravascular coagulation and frequent use of heparin in the ICU setting. Kocak et al. found serological evidence for a recent viral infection with common viruses including rubella, Epstein–Barr virus (6), cytomegalovirus and hepatitis A in 17% of patients at diagnosis (7). However, in adults it is rarely associated with infectious diseases (8). Symptoms usually appear over a period of 24–48 h in children with acute ITP. Recommended tests for establishing the diagnosis of ITP in children include full blood count with blood film examination. Platelet counts are usually <10–20 $\times 10^9/l$ (9). Thrombocytopenia is expected to resolve within 6 months of diagnosis in 80–85% of cases, while 15–20% of children diagnosed with ITP are estimated to develop chronic disease (4, 5, 10). Special attention should be given to children with a longer period of purpura leading up to the discovery of the low platelet count. Other diagnostic tests, including other laboratory tests, radiographic or sonographic imaging tests, and bone marrow examination, are reserved for patients that present atypical clinical or laboratory findings. Examples would include an elevated WBC count or suspiciously immature WBCs on the peripheral blood smear or organomegaly (11).

The peak incidence of childhood ITP occurs between ages 2 and 4 years with equal incidence in boys and girls. Children with ITP are usually otherwise well and do not have symptoms of bone pain, recurrent fevers or infections, weight loss, excessive fatigue or skin rash (2). Children present a history of acute purpura, most often with symptoms of <1 or 2 weeks’ duration. Typically bruises, petechiae and mucous membrane bleeding are the nearly universal presenting clinical symptoms, and other bleeding manifestations characteristic of thrombocytopenia, epistaxis and gastrointestinal bleeding are uncommon (1, 9). The incidence of life-threatening haemorrhage is rare (0.2–0.9%) but can be fatal when presenting in vital organs (12). In a study, bleeding from mouth, gums and tongue was found in 68 (15.9%) of 427 children (9). For a patient with poor or inadequate oral hygiene, bleeding of the gingiva is frequent (13).

Below, we would like to report the case of 11-year-old Turkish female patient who was told to stop brushing after she was diagnosed as ITP.

Case description and results

In 20 February 2006, an 11-year-old Turkish female (45 kg, 1.42 m) was referred to the periodontology clinic at Baskent University by the paediatric haematologist for the treatment of spontaneous gingival bleeding, gingival oedematous enlargement and hyperaemia. She was diagnosed as chronic ITP 6 months ago in paediatric haematology department with biopsy obtained from bone marrow.

During physical examination, petechiae on her lower extremities diffuse ecchymoses and purpura on her skin were noted. Thrombocyte count was 31.20 $K/mm^3$ (normal range: 130.00–400.00). She was hospitalized in paediatric haematology clinic. Cyclosporin A (5 mg $\times 3$ capsules daily) and deltacortil (5 mg daily) were prescribed.

The patient’s intraoral examinations revealed gingival inflammation and spontaneous gingival bleeding at the initial examination (Fig. 1). She was told not to brush her teeth after she had gingival bleeding in a rural hospital. She had hyperaemic, oedematous and swollen gingiva and abundant supra and

Fig. 1. A 11-year-old female patient with ITP at initial examination on 20 February 2006. Spontaneous gingival bleeding, gingival haemorrhage and supragingival plaque accumulation were seen.
subgingival plaque deposits all around the teeth. She was also complaining of bad breath and dull pain.

While oral hygiene is important in minimizing the gingival haemorrhage, our first goal was to improve and maintain her oral hygiene. The treatment consisted of oral hygiene motivation, debridement, and scaling and root planning under local anaesthesia. To obtain proper medical condition, she consulted a paediatric haematologist.

At the conclusion of her initial examination, she was encouraged to practice regular dental brushing with a soft-bristle toothbrush and a 0.2% chlorhexidine digluconate oral rinse was prescribed and scheduled for debridement. Figure 2 shows 4 days after the initial examination. Her oral hygiene improved and supra gingival plaque and calculus were removed at this appointment.

On 6 March 2006, her thrombocyte count was 144.00 K/mm³ and scaling and root planning of upper and lower anterior region was performed under local anaesthesia. At the control visit on 24 April 2006 (Fig. 3), same procedure was repeated when her thrombocyte count was 180.00 K/mm³.

She was scheduled for another control appointment however, she did not visit the periodontology clinic or the haematology clinic again.

Discussion

Childhood ITP is a rare condition seen in children. We present a female patient with ITP with gingival haemorrhage and oedematous enlargement due to the stopping of oral hygiene procedures after the initial diagnosis. While bruises and petechiae are common clinical symptoms in ITP, gingival bleeding is not common. There is very limited report in literature regarding spontaneous gingival bleeding.

Severe bleedings are rare in childhood ITP despite the fact that the majority of patients present severe thrombocytopenia. A study performed with 863 children newly diagnosed with ITP showed that severe bleeding is uncommon at diagnosis and rare during the next 4 weeks irrespective of treatment given (14). More than 80–90% of children with ITP do not have serious bleeding problems, and management of the bleeding is usually supportive. However, recurrent and prolonged bleeding from the nose, gastrointestinal or genitourinary tracts would usually necessitate treatment. Those with wet mucosal bleeding in the mouth, but marked bleeding including purpura or petechiae of the palate and oropharynx are thought to be at increased risk of significant bleeding and hence are treated (3). The true incidence, timing, specific sites and risk factors for major bleedings in childhood ITP are not precisely known (7).

A multisite cohort study showed that acute illness before ITP diagnosis and the presence of mucosal bleeding symptoms at diagnosis were inversely related to the risk for development of chronic ITP. Because mucosal bleeding is more likely to bring a child to diagnosis, insidious onset of symptoms was associated with chronic illness (15). Both age and presenting platelet count was shown to modify the risk for progression to chronic illness (15). Clarification of the pathways that lead to childhood ITP have influenced the therapist’ approach to therapy and may ultimately aid in the early identification of individuals who may need more aggressive interventions versus no treatment at all (12). The need for treatment should be guided by the clinical signs and not the platelet count.

Idiopathic thrombocytopenic purpura is generally a self-limiting benign disorder. It is not curable but treatment may result in a temporary improvement in the platelet account, sufficient to stop immediate bleeding. Investigation of a child for thrombocytopenia would be achieved by full blood count, blood film examination, coagulation screening and bone
marrow examination. As the platelet account does not change the management after the disease is diagnosed, repeated platelet count is unnecessary.

In this case, the patient had stopped brushing their teeth to prevent gingival bleeding, as do many others who experience gingival bleeding; this resulted in the accumulation of dental plaque and inflammation of the gingival and periodontal tissues. The treatment of plaque induced gingivitis is primarily self-administered plaque control in the otherwise healthy patients. However, professional intervention is needed to obtain and maintain proper oral hygiene in the patients who have haematologic disorders such as ITP, leukaemia and blood dysplasias. Dental plaque can exacerbate the gingival changes which include gingival swelling, glazed appearance, hyperaemia and bleeding due to changes in gingival vascular complex, the cellular content of the connective tissue and in the junctional epithelium (16). Disrupted junctional epithelium, together with the increased number of patent vessels in the plexus of vessels contiguous to junctional epithelium, is responsible for the tendency of inflamed gingiva to bleed on gentle stimulation. Nevertheless, persistent and unexplained gingival bleeding may indicate an underlying thrombocytopenia associated with any condition affecting platelet deficiency (16). Adequate oral hygiene must be maintained throughout the course of such diseases.

In the present case, mouth-rinsing with 0.2% chlorhexidine was prescribed additional to oral hygiene motivation and initial plaque removal since adjunctive effect of chlorhexidine mouth-rinsing in mechanic instrumentation was found more effective in reducing gingival inflammation, previously (17). The reduction of gingival inflammation may also minimize episodes of gingival bleeding.

On the other hand, the unique anatomic and physiological character of the oral structures predisposes the oral cavity to manifestations of systemic disturbances of the blood. The dentist must be aware of systemic importance of intraoral lesions and unexplained gingival alterations and bleeding. The management of the gingival bleeding may require a systemic medical team approach (18). In the present case, the treatment protocol consisted of consulting with paediatric haematologist, oral hygiene motivation and mechanical debridement of the plaque and calculus deposits.

The importance of adequate dental plaque control techniques in order to prevent inflammation, bleeding and infection in these patients are essential. Individualized caries prevention protocols, in relation with the patient’s caries risk, must be applied. Measures may include: the use of fluoridated toothpaste; dietary recommendations, daily fluoride mouth-washes; systemic fluoride supplements and procedures at the dental office such as fluoride or chlorhexidine application/varnish application, plaque control and pit and fissure sealants (19). The dentist may perform standard dental procedures, taking special care not to traumatize the oral tissues by minimizing needle puncturing, and carefully adapting and activating orthodontic appliances, dental restorations and endodontic treatment are to be preferred when possible, rather than surgical options (19).

Careful explanation, parental counselling and education of the patient and parents are essential for the successful management and maintenance. The child should be encouraged to have a normal lifestyle.

The dentist must be vigilant in detecting abnormal oral tissues and tissue alterations between subsequent visits which would be an initial sign of the most haematologic disorders and collaboration with a haematologist is required to ensure a good outcome of treatment for patients with ITP. With the use of well-supervised treatment protocols, the dental management of individuals with ITP can be effective and safe.

Conflict of interest, source of funding statement

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References

3 Thachil J, Hall GW. Is this immune thrombocytopenic purpura? Arch Dis Child 2008; 93: 76–81.