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Review Article

Orthodontic consideration with patients with bleeding disorders

ABSTRACT

Orthodontist must be aware of the impact of bleeding disorders on the management of orthodontic treatment. Initial recognition of a bleeding disorder, which may indicate the presence of a systemic pathologic process, may occur in dental practice. Patients should be queried about any previous unusual bleeding episode after surgery or injury, spontaneous bleeding, and easy or frequent bruising. The purpose of this paper is to review bleeding disorders and their effects on the delivery of orthodontic treatment.

Keywords: Hemophilia, orthodontic treatment, sickle cell anemia, von Willebrand's disease

INTRODUCTION

Proper dental and medical evaluation of patients is therefore necessary before treatment, especially if an invasive dental procedure is planned.[1,2] Patient evaluation and history should begin with standard medical questionnaires. Patients should be queried about any previous unusual bleeding episode after surgery or injury, spontaneous bleeding, and easy or frequent bruising.[1]

Orthodontic therapy is no longer earmarked for only healthy patients. Orthodontists need to be aware of the possible clinical implications of many of bleeding disorders and see their patients every 6–8 weeks to avoid rapidly developing medical problems which can manifest themselves at any age.

The present article elaborates bleeding disorders conditions that are relevant to orthodontic practice. A thorough medical history should be taken. This might involve seeking guidance from the patient's physician. Patients should be well informed of all the options and made aware that any orthodontic treatment has been planned with their best interests in mind. The importance of excellent oral hygiene should be emphasized to all patients.

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HEMOPHILIA A AND B

Hemophilia is an X-linked disorder affecting approximately 1 in 10,000 people (World Federation of Hemophilia – www.wfh.org).[3,4] It is the most common congenital bleeding disorder.[4] Hemophilia is a congenital hematological condition inherited and caused by a deficient activity or absence of clotting factors that are essential for normal hemostasis. Thus, hemophiliacs cannot efficiently form a clot. The severity of hemophilia depends on the amount of this coagulation factor. The normal concentrations of clotting factor are between 50% and 150% of average value, and the minimum level of a factor for adequate hemostasis is 25%. Hemophilia A is a deficiency of Factor VIII which is the most

Vaidehi N. Arekar, Pushpak Ladhe[1], Apurva Nikte[2], Pawankumar Dnyandeo Tekale[3]

Department of Orthodontics, S.M.B.T. Dental College and Hospital, Sangamner, [1]Consultant Endodontist, Private Practice, Mumbai, [2]Consultant Orthodontist, Private Practice, Mumbai, [3]Department of Orthodontics, Dr Rajesh Ramdasji Kambe Dental College and Hospital, Akola, Maharashtra, India

Address for correspondence: Dr. Pawankumar Dnyandeo Tekale,
Senior Lecturer, Department of Orthodontics, Dr Rajesh Ramdasji Kambe Dental College and Hospital, Akola, Maharashtra, India.
E-mail: pawan0804@gmail.com

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common hemophilia affecting 90% of sufferers. Hemophilia B (Christmas disease) is a deficiency of Factor IX. Both diseases result in a prolonged bleeding time. The diseases are classified into various categories as shown in Table 1.

Factor levels of >40 iu/dl are unlikely to be associated with a bleeding tendency, with a level of >50 iu/dl being the lower end of the normal range.

Orthodontic consideration

The management of hemophilia involves replacement of the deficient clotting factor by intravenous infusion to either control or prevent bleeding. The current replacement involves recombinant factor concentrates. Before this, plasma-derived factor concentrates led to further complications in this patient group with exposure to human immunodeficiency virus and hepatitis C virus. Concern also has been expressed regarding exposure to variant Creutzfeldt–Jakob disease.[5,6]

Years ago, orthodontics was avoided in patients with bleeding disorders. Currently, the bands are prefabricated and can be replaced by tubes and the technique is not multiband but multibrackets, bonded directly or indirectly, thereby reducing the possible damage to the gingival tissue. In patients with hemophilia, the best dental treatment is preventive. The patients with hematological disorders are now treated in the same way as the normal orthodontic patients; in this sense, the criteria for the indication and duration of orthodontic treatment are the same.[1,2]

VON WILLEBRAND'S DISEASE

von Willebrand's disease (VWD) is the most common bleeding disorder affecting up to 1% of the world's population.[3,7] The central feature of the disease is an abnormality in von Willebrand factor, either quantitative (Type 1 – partial quantitative, Type 3 – complete absence) or functional (Type 2 – qualitative).

The symptoms of the disease are as follows:

- Easy bruising
- Prolonged bleeding from lacerations
- Epistaxes
- Bleeding from gums
- Menorrhagia
- Postdental procedure bleeding
- Postsurgical bleeding
- Excessive postpartum bleeding
- Muscle hematomas (Type 3 VWD)
- Hemarthrosis (Type 3 VWD).

Table 1: Classification of hemophilia

Plasma factor levels	Degree of hemophilia	Characteristics
<1 iu/dl	Severe	Frequent spontaneous bleeds APTT significantly prolonged
2-5 iu/dl	Moderate	Few spontaneous bleeds. Bleeding after minor trauma APTT significantly prolonged
6-40 iu/dl	Mild	Bleed only after trauma or surgery APTT may be normal or prolonged according to the plasma concentration and sensitivity of the laboratory assay

ATTP: Activated partial thromboplastin time

Treatment of VWD can be divided into two types: adjunctive therapies that aim to provide indirect hemostatic benefit and treatments that increase the plasma levels of von Willebrand factor and Factor VIII.[3,7] The main adjunctive therapy in use is tranexamic acid, which can be used for its local and systemic effects. To increase levels of von Willebrand factor and Factor VIII, either desmopressin (1-deamino-8-D-arginine vasopressin), after a successful therapeutic trial, or intermediate purity plasma-derived Factor VIII concentrates (which contain von Willebrand factor, the carrier molecule for Factor VIII) can be used.

Orthodontic considerations

Orthodontic treatment is not contraindicated provided the patient has no or very mild complications and the oral hygiene is excellent. A multidisciplinary approach is needed. The nonextraction treatment approach is preferred. If extractions are necessary, the patient should be well ventilated and at an ambient temperature. Orthodontic forces should be reduced and rest intervals between activations should be increased to restore the regional microcirculation.[8]

VITAMIN K DEFICIENCY

Vitamin K plays an important role in metabolism of prothrombin the precursor of thrombin which is active coagulative enzyme thought to originate in the liver.

In human beings, deficiency of Vitamin K may occur due to food completely devoid of Vitamin K. However, this is extremely rare because it is manufactured by intestinal putrefactive bacteria. Deficiency may be caused by conditions, which prevents its intestinal absorption such as diarrhea, colitis, dysentery, fistulas, and strictures which are uncommon factors.[9]

Warfarin is the most important cause of Vitamin K deficiency. Use of drugs such as phenytoin, phenobarbitone, and carbamazepine during pregnancy has been associated with neonatal hemorrhage.[9]

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Orthodontic implications

Maternal deficiency of Vitamin K or use of therapeutic agents during gestation may result in deficiency of Vitamin K in embryo. Deficiency in the first trimester results in maxillonasal hypoplasia in neonate with resulting facial and orthodontic implications.

Maxillonasal hypoplasia has been classified on the basis of facial features as Binder's syndrome. It is characterized by broad flat nose, horizontal nostril, short columella, broad philtrum, pouting upper lip, marked groove at the nasolabial junction, and concave profile.

Surgical treatment for Binder's syndrome is usually performed by plastic surgeons and is limited to nasal dorsum reconstruction, elevation of the tip of the nose and lengthening of the nasal dorsum. Surgical correction of nasal and maxillary abnormalities is usually followed by orthodontic treatment. Planning the orthodontic treatment depends on the severity of the malocclusion.[9]

SICKLE CELL ANEMIA

Sickle cell disease is a commonly used term for designating a family of various blood disorders characterized by the presence of hemoglobin S (Hb-SS). Many years later, sickle cell anemia was defined as a hereditary type of chronic hemolytic anemia caused by genetic mutation of the hemoglobin molecule.[10]

Stability and physical–chemical properties of the hemoglobin molecules (Hb-AA) are modified and hemoglobin S (Hb-SS: derivation of "sickle"), results from the amino acid switch (glutamic acid replaced by valine) in the beta-chain of hemoglobin present in chromosome 11. Sickle cell syndromes are caused by different inheritance patterns involving the sickling gene, which is autosomal recessive. Therefore, homozygous individuals have nearly S-type hemoglobin only (Hb-SS), and sickle cell anemia develops as a result. Nevertheless, heterozygous individuals (Hb-SA) have approximately 40% of S-type hemoglobin (Hb-SS) and the remaining red blood cells are normal (Hb-AA), which define a trace of sickle cell and milder characteristics concerning the disease. In this case, the parents are asymptomatic carriers, and consequently, the mutated gene is transmitted to their offspring.[10]

Among the clinical systemic manifestations, the following are frequently observed: paleness of both the skin and the mucous membrane, icteric sclera, apathy, cardiac alterations by myocardial hypoxia, cephalalgia, convulsion, osseous alterations (e.g. osteonecrosis), chronic hemolytic anemia, impaired growth, low body weight, decreased production of testosterone, delay in skeletal and sexual maturation, learning difficulties, cerebral hemorrhage, and propensity to infections, which are all important causes of either morbidity or mortality.

Orthodontic implications

In radiological terms, the changes involving maxillofacial and dental tissues are similar to those observed in cases of rickets, fluorosis, and after thyroidectomy. The dura-lamina remains intact, although areas of osteoporosis may be found. In addition, trabecula with a parallel pattern among the teeth, an aspect called "step ladder," was not found to involve the edentulous area.[10]

The increased number of malocclusions in patients with sickle cell disease can be related to muscular imbalance, absence of labial sealing, or changes in the osseous base, thus leading to orthodontic intervention.

Malocclusion can be observed because of maxillary protrusion and retrusion of the anterior teeth. The former can be associated with the increased medullar activity and marked maxillary growth. In addition, lip pressure caused by overjet results in retrusion of the incisor teeth. Similar to what happens to the maxilla, the medullar hyperplasia leads to an increase in the diploic space, thus thickening the frontal bones as well as the flattened ones.

DISCUSSION

The patient should be asked for any history of significant and prolonged bleeding after dental extraction or bleeding from gingivae. A history of nasal or oral bleeding should be noted. Many bleeding disorders, such as hemophilia and VWD, run in families; therefore, a family history of bleeding disorders should be carefully elicited.[11,12] A complete drug history is important. If a patient is taking anticoagulant drugs, it will be important to consult his or her physician before any major surgical procedure. In addition, a number of medications may interfere with hemostasis and prolong bleeding. Drugs of abuse, such as alcohol or heroin, may also cause excess bleeding by causing liver damage resulting in altered production of coagulation factors. Illicit injection drug use carries an increased risk of transmission of viral pathogens that may lead to viral hepatitis and altered liver function.[1,2,12,13]

Special orthodontic consideration:

1. Maintaining and establishing the good oral hygiene will prevent the gingival bleeding
2. Self-ligating brackets are preferable to conventional brackets

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3. The archwire should be secured with elastomeric modules rather than ligature wire, later might have risk of laceration of mucosal surface
4. Any form of mucosal or gingival irritation because of the chronic irritation of orthodontic appliance should be avoided which may cause bleeding
5. Fixed appliances are preferable to removable appliances as the latter can cause gingival irritation
6. The duration of orthodontic treatment should be kept to a minimum to reduce the potential for complication
7. Although medical science in the field of blood transfer and blood factors has been many advances, but still not possible to eliminated transmit the virus in these patients completely. There is possibility viral infection and bleeding risk. Hence, the dentist must follow infection control and health measures severely.

CONCLUSION

During orthodontic treatment, emotional stress should be avoided, and care should be taken to have both adequate levels of oxygenation and bodily temperature, as well as the use of biologically compatible mechanical forces. The clinical appointments should be arranged during the chronic phase of the disease because orthodontic procedures are not performed during periods of crisis or acuteness. In general, orthodontic treatment is not contraindicated in patients with bleeding disorders. If tooth extraction of other surgery is required in patients with severe bleeding disorders, they are usually hospitalized and given transfusion of the missing clotting factor in advance of the procedure. Where possible, the nonextraction treatment is adopted.

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Conflicts of interest

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