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DENTAL CONCERNS OF CHILDREN WITH PLATELET DISORDERS - A SHORT COMMUNICATION

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ABSTRACT

Purrpura is a disease included in the large group of hemorrhagic disorders and is distinguished by cutaneous hemorrhage and blood loss from mucous membranes and internal organs, which is always related to vascular or platelet alterations Thus thrombocytopenia, which is distinguished by the decrease in the number of blood platelets, is included in this large group. This article discusses about etiologiy, clinical features and management of children with platelet disorders.

KEYWORDS: Children, dental considerations, purpura.

INTRODUCTION

Hemopathies are categorized in three large groups, according to the nature of the altered hematologic component. Thus, they are divided in disorders that affect erythrocytes (red blood cells), leucocytes (white blood cells) and hemorrhagic disorders, among which are included platelet and coagulation factors alterations.[1] Platelets are small granular corpuscles measuring 2 to 4µ in diameter found in the bloodstream. Their role in the coagulation process is the production of a loose aggregate of platelets (temporary hemostatic plug) immediately after a lesion in the blood vessel. This plug is maintained and posteriorly converted into a definite fibrin clot. [1]

Platelet disorders can be divided into two types.

1. Qualitative - those caused by platelet dysfunction
2. Quantitative - those caused by inadequate number of platelets.

Platelets normally the number between 1, 50,000 and 4, 00,000/cu mm of blood. Patients with counts less than 1,00,000 have a moderate disease, and those with less than 50,000 have a severe thrombocytopenia, when level drops to 39,000/cu mm spontaneous bleeding occurs,.

Classification of platelet disorders are given in table 1.

Table 1: Different platelet disorders and their characterstics.

Congenital	Acquired	Conditions associated with platelet disorders
Glanzmann's syndrome	--- Platelet disfunction due to drugs. eg- Asprin and other NSAIDs, phenytoin, Cotrimoxazole	--Renal Failure
Benard – doulier syndrome	--Throbocytopenias eg: Idiopathic thrombocytopenia Immune thrombocytopenia associated withinfection. eg: Infectious mononucleosis, HIV, Rubella, Varicella	--Associated disease processes eg; leukemia, Aplastic anemia
Wiskott-aldrich syndrome	---Increased platelet activity eg: Nephrotic syndrome, Kawasaki's disease	--Cyonotic congenital heart diasese

Thrombocytopenic purpura

After injury to a blood vessel, extravasation of blood is controlled initially by the platelets which produce a local vasoconstrictor and also plug the gap physically. Deficiency of platelets results in prolonged bleeding, particularly from small vessels for which this is the principal method of control. In larger vessels the clotting mechanism of the blood then comes into play to prevent further escape and seals off the site of damage, but platelets also effect clot retraction at a later stage and a deficiency allows further prolonged bleeding.[2]

Such a deficiency occurs in the thrombocytopenic purpuras. In this group of conditions typically there are petechiae of the skin and mucous membranes, ecchymoses and prolonged bleeding from trivial injuries. There may be spontaneous bleeding from the gastro-intestinal and genito-urinary tracts and the blood loss may cause anaemia. Bleeding problems are rare unless the platelet level falls to 50,000 or less, but there is variation from patient to patient as to the level at which it occurs. The clottingtime is normal but the bleeding time prolonged, though this varies from day to day. The tourniquet test is positive butunnecessary in the presence of petechiae and other signs.[4]

Four types of thrombocytopenic purpura are described, namely, secondary, neonatal, thrombotic and idiopathic, but only the last may be long-standing or recurrent and in need of continuing supportive care.

Idiopathic Thrombocytopenic Purpura

Idiopathic thrombocytopenic purpura is a condition which may possibly be due to an altered immunological reaction, and occurs in acute and chronic forms.

The acute form has a sudden onset, often following some infective illness, and manifests itself as spontaneous petechiae of the skin and mucous membrane, and ecchymosed at the site of mild trauma. There is enlargement of spleen or lymph glands. There may be rapid fatal termination or the disease may last a variable period up to two months or more and clear up. This form is the most common in children and rarely recurs.[5]

The chronic form is less common in children and has a more insidious onset. The first evidence may be excessive bleeding after a dental extraction or similar traumatic incident, and enquiry may disclose a prolonged history of easy bruising and persistent bleeding from trivial wounds. Many cases have a family history of this. The bleeding time is prolonged but the clotting time is normal, and the patient may have anaemia due to persistent or repeated haemorrhage.

Approximately one-third of children with the chronic form have persistence or recurrence of the condition and treatment may have included splenectomy to reduce the destruction of platelets. The patient is likely to have had considerable periods of corticosteroid therapy, which appears to decrease antigen-antibody reaction. It is given in high dose at first, with reduction as improvement occurs.[6]

Idiopathic Thrombocytopenic Purpura (ITP) is considered one of the most common disorders in children and the incidence of asymptomatic illness is approximately 3-8:100,000 children/year12 IPT differential diagnosis must be performed in order to distinguish it from other conditions that may cause the symptoms of thrombocytopenic purpura, such as thrombocytopenia induced by medication (barbiturate, quinine, lengthen therapy with glucocorticoids), hereditary thrombocytopenia.[7]

Currently, IPT is considered an autoimmune disease and its etiology is related to immunological mechanisms such as antibodies and antiplatelet immunoglobulin in the blood16 Genetic studies show the correlation of clinical development of IPT and certain genotypes, suggesting a genetic predisposition and modulation of the illness 16progression15 IPT is the result of the increment of the destruction of antiplatelet antibodies (autoantibodies) through cells of the reticuloendothelial system, especially the spleen. The autoantibodies bind to platelets and are eliminated by the spleen, thus the disease severity reflects the balance of the capacity to produce platelets by the bone marrow mega-karyocyte and platelet destruction by the reticuloendothelial system.[8]

Medical treatment for IPT patients adopts different intervening forms, according to individual conditions of each patient given that the immune system of each patient demands up-to-date therapeutics. Asymptomatic children or those whose illness is moderate without severe hemorrhagic episodes can be monitored and don't need specific medicinal therapy. In these cases follow-up without medication intervention is the best form of treatment and success of intermittent controls of platelet count until the disease remission.[9]

In more severe cases with risks of intracranial, gastro-intestinal and genitourinary hemorrhage, there is the option of platelet transfusion. In cases of chronic IPT, in which no success was achieved with all the attempted therapeutic resources, splenectomy is recommended, despite its performance restricted success.[10]

In the case of acute IPT in children, there are treatment options with corticoids with high doses of intravenous immunoglobulin doses, and more recently with Anti-D immunoglobulin.[11]

Mechanisms of action of corticoids employed in the treatment of acute IPT include the decrease of the antibody-platelet complex clearance and prevention of platelet phagocytosis. High doses of corticoids for a short period are used to increase platelets within 2 to 4 days. The dose ranges from 4mg/kg per day to 30mg/kg of prednisone or methyl prednisone for up to four days. No

excessive collateral effects were observed for doses of 4mg/kg per day for four days.[12]

Another treatment option for acute IPT is intravenous immunoglobulins. The most utilized dose is of 0.8 to 1.0/kg/day for 1 or 2 days. In this dosage, it was observed anincrease of platelet counts 24 hours after infusion. Collateral effects verified in the usage of intravenous immunoglobulin are headaches, body tremor, fever, nausea, vomit and aseptic meningitis.[13]

Dental Treatment

Children with non-persistent types of idiopathic thrombocytopenic purpura should, of course, avoid any surgical procedures and injections for the duration of the illness, but is it those with chronic persistent purpura who need continuous dental support. Because of the bleeding tendency, the aim is to avoid the nod for extractions, and dental care should be directed to conservative measures as are used for patients with haemophilia with the additional hazard of corticosteroid therapy in many cases.[14,15]

Non-Thrombocytopenic Purpuras

Purpuras also occur in which the platelet count is normal and have the typical petechiae and spontaneous bleeding from mucous membrane surfaces due to damage to or unusual permeability of the capillary walls. They include the infectious and allergic types of which only the second may need special dental support.

Allergic purpura (Schoenlez'n-Henoch purpura)

This is a condition which recurs at variable intervals of weeks or even years and in which the symptoms may be very variable. There is increased capillary fragility which causes petechiae and ecchymoses, but there may also be moderate or severe gastrointestinal involvement with diarrhoea and vomiting, and nephritis commonly occurs. Joint symptoms may be present in some cases.

The aetiology is uncertain but investigation may produce an allergy to some foodstuff, and in somecases there appears to be an association with a streptococcal infection.

Treatment is largely supportive in the avoidance of any identified allergen, and the elimination of sepsis, the latter being particularly important in view of the frequency of nephritis.[16]

Oral Condition: There are no special dental features of this condition.

Dental Treatment

This should be directed to the elimination of sepsis initially, and any children with a history of nephritis associated with the condition within the previous two years or so are safer with an antibiotic cover for extractions. These should not be done without the consent of the physician in charge if the patient is not in a period of normality. The mouth should then be maintained in a healthy state as a general supportive measure.

Table 2: Laboratory findings for platelet disorders.

TEST	DISORDERS	
	QUANTATIVE	QUALITATIVE
Bleeding time	Abnormal	Abnormal
Capillary fragility	Abnormal	Abnormal
Platelet count	Abnormal	Normal
ADP and Collegen	Normal	Abnormal
Clot retraction	Abnormal	Variable
Clotting time	Normal	Normal
Plasma recalcification time	Abnormal	Variable
PTT	Normal	Normal
Thromoboplastin generation time	Abnormal	Abnormal
Thrombin time	Normal	Normal

Dental management of platelet disorders[16]

- Elective dental treatment should be postponed until a platelet count is above 50,000/mm^3
- Steroids a dose of 1-2mg/kg is preferred to bring up the platelet level.
- Local hemostasis measures should be used.
- During acute phase nerve blocks and extractions should be avoided.
- Prior to any surgical procedures and extraction, use IV Immunoglobulin 1g/kg /day twice.
- Avoid NSAIDs and aspirin 7 days preoperatively.
- Replacement therapy usually involves platelet concentrate transfusion or whole blood transfusion before surgical procedures.
- One unit of platelets usually increases a patient's count by 6000/mm^3
- Improve red cell mass supply coagulation factors.

CONCLUSION

It is important to emphasize the significance of the medical history and the appropriate physical examination during the diagnostic process, as well as collaboration with the patient's medical clinic. The medical history

should compile data such as the beginning of signs and symptoms, time of development, previous history and types of bleeding, drug intake, and the presence of other signs and symptoms, such as fever, adenopathy and anemia not only from the patient, but also from the family. The most important sign during the physical examination is the presence or report of petechiae occurrence.

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