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PURPURA IN INFANTS AND CHILDREN

Essay

*Submitted in partial fulfillment For Master's
Degree of Dermatology and Venereology*

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ABSTRACT

Purpura is defined as the extravasation of red blood cells from the vasculature into the skin. Depending on their size, purpuric lesions are classified as petechiae, purpura per se or ecchymoses. Some of the disease processes that can lead to purpura are unique to the newborn period. Blue berry- miffin baby, protein C and S deficiency and infectious purpura are the three for which dermatologic consultation is usually requested. Diseases characterized by purpura in older children are classified as platelet disorders, coagulation abnormalities and vascular defects.

Key words: Purpura- petechiae – Echymoses – vibices – newborn – children.

Acknowledgement

I would like to express my deepest gratitude and thanks to eminent professor Dr. Hada Mohamed Hussen Resheed Professor of Dermatology, Faculty of Medicine, Cairo University, and for her enthusiastic encouragement, constant support, guidance, advice and helpful efforts throughout the entire work whose efforts and assistance enable the accomplishment of this work.

I am greatly indebted to Dr. Heba Helmi El Hadidi Lecturer of Dermatology, Faculty of Medicine, Cairo University, for her most valuable guidance and continuous keen supervision.

My sincere thankfulness to all my family and my friends.

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List of Abbreviations

PS	Protein S.
PC	Protein C.
PIC	Protein C inhibitor.
DIC	Dissiminated intravascular coagulation.
CNS	Central nervous system.
FFP	Fresh Frozen plasma.
PCC	Prothrombin complex concentrate.
APT T	Activated partial thromboplastine time.
ITP	Idiopathic thrombocytopenic purpura.
HSP	Henoch-Schonlein purpura.
SLE	Systemic lupus erythematosus.
AHE	Acute hemorrhagic edema.
PAN	Polyarteritis nodosa.
CMV	Cytomegalovirus.

Huemostasis

HAEMOSTASIS

Haemostasis may be defined as the sum total of those specialized functions within the circulating blood and its vessels that are designed to stop hemorrhage. Haemostasis is a highly integrated set of functions, but it is conceptually helpful to divide the overall process into components according to their approximate sequence.

- 1- Vascular phase.
- 2- Platelet phase and,
- 3- Plasma phase.

The vascular phase is largely understood in terms of its known interactions with platelet and plasma factors. Thus vasoconstriction is mediated by serotonin released from platelets. Also, it is known that the vessel makes the following major contributions to platelets and plasma phase of haemostasis.

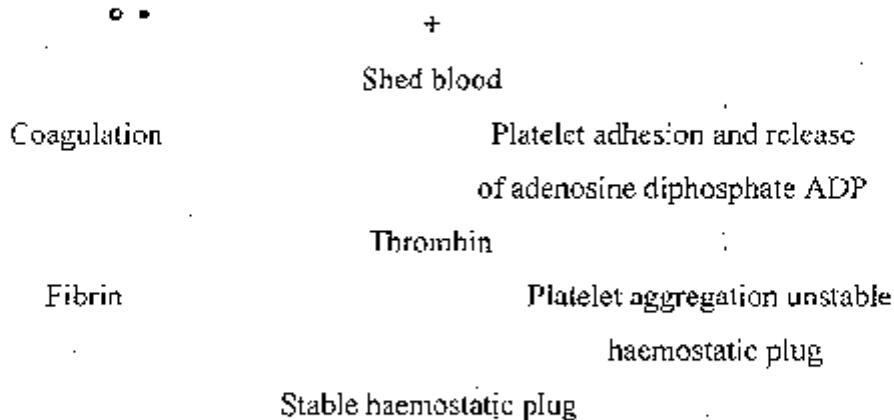
- a- Exposed subendothelial structures, including collagen fibres, becomes the site of platelet adhesion and participate in stimulating platelet aggregation.
- b- Injury induced vascular surface changes interact with factor XII and other factors to initiate the intrinsic pathway of coagulation.
- c Tissue thromboplastin released by vascular injury interacts with factor VII and calcium to initiate the extrinsic pathway of coagulation.
- d- Stimulated endothelial cells release prostacyclin (PGI_2), which inhibits platelet aggregation (*Ladisch, 1990*).

Haemostasis is also influenced by extravascular factors like support of the vessels which play an important subsiding role, particularly in venous bleeding (*Jandl, 1987*).

The relative importance of various factors concerned with haemostasis varies with the size of the vessel involved. In small arterioles, capillaries and venules haemostasis depends mainly on vessel constriction and on platelet plugging. In large vessels, although constriction is important in limiting the initial loss of blood, the formation of the haemostatic plug to seal the defect in the vessel plays the major role in haemostasis (*Jandl, 1987*).

Vascular defects are the commonest cause of bleeding disorders seen in the clinical practice. Most cases of bleeding due to a vascular defect alone are not severe, and frequently the bleeding is mainly or wholly into the skin causing petechiae, or ecchymoses, or both. Petechiae may be rather pale and tend to be confluent, ecchymoses are usually small. In some disorders, there is bleeding from mucous membranes, and bleeding into internal organs may occur (*Castaldi, 1983*).

TRAUMA



(Gruchy, 1983).