

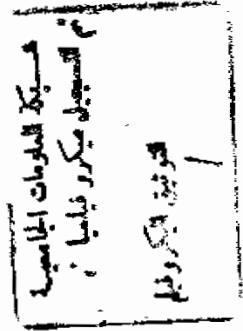
Hypersplenism

Essay

*Submitted in partial fulfilment of
Master Degree in
Clinical & chemical pathology*

Presented by

Mervat Mohammed Samy El-Hady
M.B., B.Ch.



Supervised by

Prof. Dr. Azza Ahmed Mohammed
*Professor of Clinical & Chemical pathology
Faculty of Medicine
Cairo University.*



Prof. Dr. Mohammed Salem
*Professor of Internal Medicine
Faculty of Medicine
Cairo University*



Prof. Dr. Nabil El-Danasory
*Ass. Professor of Clinical & Chemical pathology
Faculty of Medicine
Cairo University*

1996





To My Dear Family and My Lovely Little Son, Omar

Acknowledgment

*"First and foremost, thanks are due to **GOD**, the most Beneficent and Merciful".*

*It is an honour to express my utmost greatfullness, sincerest gratitude and appreciation to **Prof. Dr. Azza Ahmed Mohamed**, Professor of Clinical and Chemical Pathology, Faculty of Medicine, Cairo University for her kind supervision, unlimited support and without her aid and continuous encouragement, this work would not come to light.*

*I am very much grateful to **Prof. Dr. Mohammed Salem**, Professor of Internal Medicine, Faculty of Medicine, Cairo University for his assistance and continuous guidance.*

*I wish to offer my profound appreciation and gratitude to **Prof. Dr. Nabil El-Danasoury**, assist. Professor of Clinical and Chemical Pathology, Faculty of Medicine, Cairo University, for his valuable suggestions and good advice.*

*I would like to express my deepest thanks and gratitude to **Prof. Dr. Somaia El-Gohary**, Assist. Professor of Clinical and Chemical Pathology, Faculty of Medicine, Cairo University, for her sincere help and valuable advice.*

Contents

	Page
• Introduction and aim of the Work -----	1
• Review of Literature : -----	2
<i>The Normal Spleen</i> -----	2
a. Anatomy -----	2
b. Physiology -----	4
c. Functions -----	6
<i>Hypersplenism</i> -----	10
1. Difinition -----	10
2. Pathogenesis -----	10
3. Aetiology : (Common potential causes of hypersplenism e.g. Schistosomiasis, Malaria, Thalassemia, Lymphomas, Lipoid storage disease, etc..) -----	13
4. Diagnosis -----	49
5. Treatment -----	54
• Technique and Interpretation of Splenic Aspiration -----	65
• English Summary -----	72
• References -----	74
• Arabic Summary	



Abbreviations

Cr	: Chromium
FCC	: Follicular center cell.
HMS	: Hyperactive malarious splenomegaly
ML	: Malignant lymphoma
NML	: Nodular mixed lymphoma
PDL	: Poorly differentiated lymphocytic
PTCL	: Peripheral T-cell lymphoma
Tc	: Technitium



Introduction and Aim of the Work

Introduction And Aim Of The Work

The clinical syndrome, hypersplenism, was first recognized in 1866 by Grestel as “Splenic anemia”.

By time, the term Banti’s disease (1880) was attached to every case showing splenomegaly and blood cytopenias.

In 1907, Chaurrffard introduced the term hypersplenism, to refer to exaggerated activities of the spleen (*Jacob, 1974*).

Hypersplenism is a syndrome characterized by splenomegaly and any or all of the following cytopenias: anemia, leucopenia, or thrombocytopenia as well as hypercellular bone marrow. These cytopenias are mostly corrected by splenectomy (*Hoffman, 1991*).

The aim of this work is to review on the different causes of hypersplenism, it’s pathogenesis, diagnosis and management.



Review of Literature

The Normal Spleen

Anatomy of the Spleen:

The spleen is a small, well perfused organ receiving about 5 percent of the cardiac output (*Sills, 1987*).

The normal spleen weighs about 150 g and is situated posteriorly between the fundus of the stomach and the diaphragm in the line of the 10th rib. The tail of the pancreas is adjacent to the splenic hilum and in children the spleen vests on the left adrenal gland.

The cut surface of the spleen consists of areas of “red pulp” within which can be seen pale, ovoid nodules (about 1 mm in diameter) of white pulp. The splenic artery divides at the hilus into branches which run along the trabeculae. These trabecular arteries pass into the white pulp (fig. 1) where they give off branches which are almost perpendicular to the central trunk. this produces a skimming effect by which plasma tends to pass down the branches to the white pulp and most of the red cells pass in the trabecular artery to the red pulp. The white pulp has an immune function whereas the red pulp filters abnormal red cells from the circulation. Phagocytosis of blood born particles occurs in both areas.

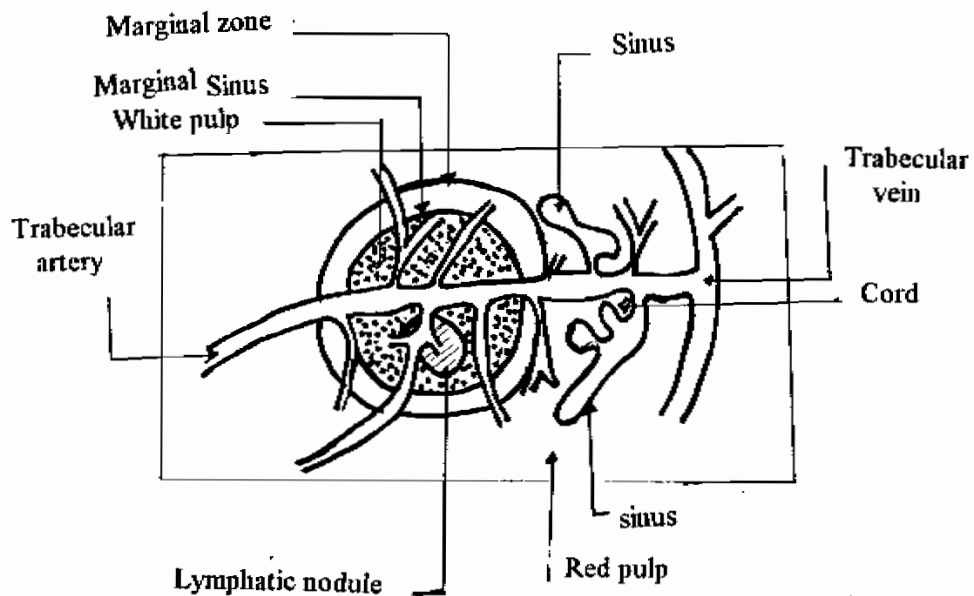


Fig. (1): A central trabecular artery passing through the white pulp into the surrounding red pulp. A blood flow skimming effect results in most of the plasma passing down branches of the artery while the cells pass in the central trabecular artery directly to the red pulp.

The white pulp consists of a central trabecular artery surrounded by lymphatic nodules with germinal centres and periarterial lymphatic sheaths which provide a framework filled with lymphocytes and macrophages. At the edge of the white pulp is marginal zone into which pass arteries from the central artery. Plasma rich blood which has passed through the central lymphatic nodules is filtered as it passes through the sinuses within the marginal zone, and particles are phagocytosed.

Immunoglobulins produced in the lymphatic nodules enter the circulation through the sinuses in the marginal zone. Beyond the marginal zone is the red pulp which consists of cords and sinuses. Cell concentrated blood passes in the trabecular artery through the centre of the white pulp to the red pulp cords. In order to pass from the cords to the sinuses, the red cell must elongate and become thinner. This filters abnormally shaped or rigid cells out of the circulation.

Ninty percent of the blood passing through the spleen moves through an "open" circulation in which blood flows from arteries to cords and then to sinuses. The remaining 10% bypasses the cords and sinuses by direct arteriovenous connections (*Allen-Mersh, 1988*).

The blood exits through the splenic vein into the portal system. Since the veins in the portal system lack valves, any increase in portal pressure is transmitted to the splenic microcirculation (*Hoffman, et al., 1991*).

The Physiology of The Spleen:

The spleen acts as a blood reservoir. The capsule of the spleen in

many lower animals contains large amounts of smooth muscle, and sympathetic stimulation causes intense contraction of the spleen. Conversely, sympathetic inhibition results in considerable splenic expansion with consequent storage of blood.

In humans, the splenic capsule is nonmuscular, but even so, dilatation of vessels within the spleen can still cause the spleen to store several hundred milliliters of blood at times. Then under the influence of sympathetic stimulation, constriction of the vessels will express most of this blood into the general circulation. But the spleen is so small, only 150 to 200 ml. in volume, that this reservoir function in human beings is of relatively little importance.

Two areas exist in the spleen for the storage of blood: the venous sinuses and the pulp. Small vessels flow directly into the venous sinuses, and when the spleen distends, the venous sinuses swell, thus storing blood.

In the splenic pulp, the capillaries are very permeable, so that much of the blood passes first into the pulp and then oozes through it before entering the venous sinuses. As the spleen enlarges, many cells (but not the plasma) become stored in the pulp. Therefore, the net quantity of red blood cells in the general circulation decreases slightly when the spleen enlarges. The spleen can store enough cells that splenic contraction can cause the hematocrit of the systemic blood to increase in humans as much as 1 to 2 percent and as much as 3 to 4 percent in some lower animals. This increased hematocrit is an aid to the body during periods of stress.

Functions Of The Spleen:

Although the spleen is not necessary for life, it performs important functions that are generally divided into two major categories; those related to cellular elements in the circulating blood (haematologic functions), and those that are immunologic in nature (*Eichner, 1979*).

A- *The Haematologic Functions Include:*

- (a) **Hematopoiesis:** Which supplies erythroid, myeloid, lymphoid cells and platelets in fetal life, essentially ceases by the seventh intrauterine month.
- (b) **Pooling:** This means that the concentration of different blood cells, are greater in splenic blood than in the circulating blood. Pooling has been demonstrated for platelets and also for reticulocytes. In the normal human, about 30 percent of platelets are pooled in the spleen (*Aster, 1966*).
- (c) **Pitting:** Refers to the removal of rigid structures such as Heinz bodies, Howell-Jolly bodies, and hemosiderin granules from red cells. The process involves the removal of non deformable intracellular substances from deformable cells. The rigid body is phagocytized while the deformable cytoplasmic mass passes into the sinus and returns to the general circulation. The post-splenectomy blood smear is characterized by the presence of circulating erythrocytes with Howell-Jolly and Pappenheimer bodies (siderotic granules). Nucleated cells also have their nuclei removed in the same fashion.