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Short Communication

Pathophysiologic Mechanisms of Vascular Complications After Splenectomy

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DESCRIPTION

The spleen is a significant organ of the hematologic and reticuloendothelial systems, it performs crucial hematologic and immunologic activities. The white pulp and red pulp are the two main functioning divisions of the spleen. The white pulp, which has a significant amount of lymphoid tissue, is crucial for recognising antigens and producing antibodies [1]. The Billroth cords, which make up the red pulp of the spleen, are a closely packed meshwork of sinusoids that are largely used for hematologic purposes, including blood filtration. The red pulp's environment is comparatively acidic and hypoglycemic. Splenic macrophages eventually eliminate old or injured red cells that cannot survive in this hostile environment. These phagocytic cells lining the sinusoids also detect and eat antibodies-coated cells and bacteria. In order to eliminate encapsulated organisms from the bloodstream, a person's capacity to have a functional spleen is severely compromised [2]. Red cells are also cleared of particulate debris as they go through the splenic sinusoids, and these "polished" or "conditioned" red cells, which are free of surface flaws, are then released back into the bloodstream. Additionally, a lesser number of granulocytes and around one-third of the total platelet mass are stored in the red pulp.

Vascular events following splenectomy are likely multifactorial and are most likely the result of a number of factors, including hypercoagulability, platelet activation, endothelial disruption and activation, and changed lipid profiles. The primary phagocytic function of the spleen is the removal of senescent or aberrant red blood cells, infectious agents, and other insoluble cellular waste [3]. The crimson pulp of the spleen, which is coated with macrophages, performs this filtration role by aggressively swallowing substances that are difficult for the blood to flow around. In the absence of this incredibly sensitive filter, particles and damaged cells may be allowed to remain in the bloodstream, which could disturb and activate the vascular endothelium, changing the vascular homeostasis toward increased coagulation [4]. There is insufficient proof that splenectomy elevates levels of platelet count, haemoglobin concentration, plasma cholesterol, leukocyte count, and C-reactive protein, regardless of the indication. Together, these elevations may be expected to promote a very unfavourable prothrombotic condition because each of these elevations alone is linked to an increased risk of arteriothrombosis.

Clinical significance

To further identify the role of the spleen in vascular homeostasis, it is critical to better characterise the thrombotic hazards of splenectomy in individuals without continuous hemolysis (e.g., HS, ITP, trauma). When left untreated, Pulmonary Arterial Hypertension (PAH) has a high death rate in those with impaired or nonexistent splenic function, but novel medicines are being developed that may be able to treat it. It should be easier to decide whether and when to perform a splenectomy in patients with Hereditary Spherocytosis (HS), Dependent Immune Thrombocytopenia (ITP), autoimmune hemolytic anaemia, or other conditions for which splenectomy may be beneficial with more information about the prevalence, age, and risk factors for PAH and other vascular events after splenectomy. The current practise of advising splenectomy in many children with HS, for example, may need to be reevaluated if validated biomarkers of thrombosis and PAH or real arterial or venous thrombotic events are indeed troublesome after splenectomy [5]. Furthermore, even after they appear to have been "cured" of their initial disease, children and adults who underwent splenectomy for any reason may be encouraged to seek monitoring or thromboprophylaxis. Although no prospective trials have yet assessed the effectiveness of this strategy, several writers have argued for the use of short-term and/or long-term thromboprophylaxis after splenectomy, using anticoagulation or antiplatelet medications, particularly in thalassemia patients. Therefore, future research should focus on clarifying the risks of following thrombotic and vascular events related to hemolysis, splenectomy, or a combination of the two.

CONCLUSION

There seems to be strong support for a hypercoagulable state following splenectomy that was done for a variety of reasons. This discovery is most likely exacerbated by further underlying issues, particularly intravascular hemolysis. The strength of the data is less convincing that atherothrombosis (including myocardial infarction and stroke) happens more commonly

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following splenectomy, despite the fact that we believe there is strong evidence that PAH and venous thrombosis occur at greater rates in asplenic hosts.

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