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Behçet syndrome as a model of inflammation-induced thrombosis

Behçetov sindrom kao model tromboze inducirane upalom

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Behçet syndrome is a multisystem inflammatory disorder characterized by a distinctive propensity for vascular involvement, affecting arteries and veins of virtually any size. Venous thrombosis is among its most frequent and clinically relevant manifestations and differs substantially from conventional thromboembolic disease. Rather than being primarily driven by classical thrombophilic mechanisms, thrombosis in Behçet syndrome is closely linked to vessel-wall inflammation, endothelial dysfunction, and an exaggerated innate immune response.
Neutrophil activation, oxidative stress, neutrophil extracellular traps, platelet activation, and endothelial injury interact to create a highly prothrombotic inflammatory environment. This concept of immunothrombosis helps explain several characteristic features of Behçet-related vascular disease, including the strong adherence of venous thrombi to the vessel wall, the relatively low frequency of pulmonary embolism despite extensive thrombosis, and the association between thrombotic events and active systemic inflammation.
These mechanisms also have direct therapeutic implications. Immunosuppression remains the cornerstone of treatment for vascular Behçet syndrome, while the role of anticoagulation should be individualized according to vascular phenotype, bleeding risk, and the presence of arterial aneurysms. Behçet syndrome therefore represents a paradigmatic disease in which inflammation and thrombosis are tightly interconnected. Understanding this relationship may provide broader insights into thromboinflammation and vascular injury across systemic inflammatory diseases.

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