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Short Communication

Pathological Changes in Venous Valves and Their Clinical Significance

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DESCRIPTION

Venous valves are specialized bicuspid structures within the veins that maintain unidirectional blood flow toward the heart and prevent retrograde movement of blood. Proper functioning of these valves is essential for efficient venous return, particularly from the lower extremities, where blood must overcome gravitational forces. Dysfunction or structural damage to venous valves is a central feature of chronic venous disorders, including Chronic Venous Insufficiency (CVI) and varicose veins and plays a critical role in the pathogenesis of venous hypertension and venous ulceration. Understanding the pathological changes in venous valves and their clinical implications is important for accurate diagnosis, effective management and the prevention of long-term complications [1].

Pathological changes in venous valves can result from a combination of mechanical, inflammatory and degenerative processes. One of the primary alterations observed is valvular leaflet thickening and fibrosis. Chronic exposure to elevated venous pressure, often due to prolonged standing, immobility, or prior deep vein thrombosis, leads to endothelial injury and infiltration of inflammatory cells into the valve structure. This inflammatory response stimulates fibroblast proliferation and extracellular matrix deposition, causing thickening and stiffening of the valve leaflets. As a result, the valves lose their ability to coapt fully, leading to incomplete closure and retrograde blood flow, known as venous reflux.

Another significant pathological change is valvular retraction and elongation. The continuous hemodynamic stress on the vein wall and valve leaflets can cause progressive stretching and deformation of the valve structure. Retracted or elongated leaflets fail to close adequately, exacerbating venous reflux and promoting venous hypertension in the distal veins. Over time, this condition contributes to vein dilation and tortuosity, which are hallmark features of varicose veins. Additionally, venous valve cusp asymmetry or malformation, whether congenital or acquired, further impairs valve competence and predisposes individuals to chronic venous disorders [2-4].

Inflammation and endothelial dysfunction play pivotal roles in venous valve pathology. The endothelium, which lines the valve and venous wall, regulates vascular tone, maintains anti-thrombotic properties and modulates inflammatory responses. Chronic venous hypertension and oxidative stress induce endothelial activation, resulting in increased expression of adhesion molecules, cytokines and matrix metalloproteinases. These factors contribute to remodeling of the valve and surrounding vein wall, weakening the structural integrity of the venous system. In patients with post-thrombotic syndrome, residual thrombus and persistent inflammation can lead to permanent valve destruction, further compromising venous return.

The clinical significance of pathological changes in venous valves is profound, as valve dysfunction underlies the majority of chronic venous disorders. Incomplete closure of the valves leads to venous reflux, which increases venous pressure in the lower limbs, causing symptoms such as leg swelling, heaviness, aching and fatigue. Over time, chronic venous hypertension induces skin changes, including hyperpigmentation, lipodermatosclerosis and atrophy blanche. These changes impair local tissue perfusion and increase susceptibility to venous ulceration, particularly in the medial or lateral aspects of the ankle. Venous ulcers are often slow to heal, recurrent and significantly affect patient quality of life, mobility and independence [5-7].

Diagnosis of venous valve pathology relies on a combination of clinical evaluation and diagnostic imaging. Physical examination may reveal varicosities, edema and characteristic skin changes, while functional assessment of valve competence is typically performed using duplex ultrasonography. This non-invasive modality allows visualization of venous flow patterns, detection of reflux and assessment of valve morphology, providing critical information for treatment planning. Other diagnostic techniques, such as photoplethysmography and venous plethysmography, can supplement assessment by quantifying hemodynamic impairment and calf muscle pump efficiency [8-9].

Management strategies for venous valve dysfunction are guided by the severity of disease and symptom burden. Conservative measures, including compression therapy, leg elevation and exercise, aim to reduce venous hypertension and improve venous return. Compression stockings are particularly effective in reducing edema and alleviating symptoms by supporting vein

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walls and promoting valve function. Pharmacologic interventions, such as venoactive agents, can provide symptomatic relief by decreasing inflammation and venous wall stress. In cases of significant valvular incompetence or recurrent venous ulcers, interventional procedures such as endovenous ablation, sclerotherapy, or surgical valve repair may be indicated. These interventions aim to restore venous hemodynamics by closing or bypassing incompetent veins, thereby reducing reflux and preventing further valve-related complications [10].

CONCLUSION

In conclusion, pathological changes in venous valves, including leaflet thickening, fibrosis, retraction and endothelial dysfunction, are central to the development of chronic venous disorders. These structural and functional abnormalities contribute to venous reflux, venous hypertension and progressive complications such as varicosities, skin changes and venous ulceration. Early recognition of valve pathology through clinical and imaging assessment, combined with appropriate conservative or interventional management, is essential for mitigating symptoms, preserving venous function and improving patient quality of life. Understanding the pathophysiology and clinical significance of venous valve changes provides a foundation for effective prevention and individualized treatment of venous disorders.

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