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Vasculitides and Acute Vascular Syndromes: Pathophysiological Links Between Acute Coronary Syndrome and Acute Cerebrovascular Events

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Abstract: Vasculitides represent a heterogeneous group of systemic inflammatory disorders characterized by immune-mediated injury of blood vessels. Beyond their classical systemic manifestations, vasculitides are increasingly recognized as significant contributors to cardiovascular and cerebrovascular morbidity. Acute coronary syndrome (ACS) and acute ischemic stroke are among the most severe vascular complications in patients with systemic vasculitis. This review summarizes the current understanding of the pathophysiological mechanisms linking vasculitis with ACS and acute cerebrovascular events, the clinical spectrum, diagnostic approaches, and management strategies.

Introduction

Vasculitides, defined as inflammatory disorders of blood vessel walls, affect both large and small vessels, leading to ischemic and thrombotic complications. The cardiovascular system is frequently involved, and vascular inflammation can precipitate acute vascular syndromes such as myocardial infarction and ischemic stroke. The clinical burden of ACS and acute ischemic stroke is considerable worldwide, with shared risk factors including atherosclerosis, endothelial dysfunction, and systemic inflammation (1,2). In the context of vasculitis, these processes are accelerated and amplified due to chronic immune activation and vascular injury. This review will explore the relationship between systemic vasculitis, ACS, and acute cerebrovascular events, emphasizing pathogenesis, diagnostic challenges, and therapeutic perspectives.

Pathophysiology of Vasculitis and Vascular Complications

Immune-Mediated Vascular Injury

Vasculitides are characterized by immune complex deposition, T-cell activation, and autoantibody-mediated injury (3). Inflammation results in endothelial dysfunction, increased vascular permeability, and intimal hyperplasia, ultimately narrowing vessel lumina.

Pro-Thrombotic State

Inflammatory cytokines (TNF-α, IL-6) induce pro-coagulant pathways, platelet activation, and microthrombi formation (4). This mechanism is central to both myocardial infarction and ischemic stroke in vasculitis patients.

Accelerated Atherosclerosis

Patients with systemic vasculitis demonstrate premature atherosclerosis due to chronic systemic inflammation, glucocorticoid exposure, and endothelial injury (5). This predisposes to plaque rupture and acute coronary syndromes.

Vasculitides Associated with Acute Coronary Syndrome

Large-Vessel Vasculitis

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Takayasu arteritis affects the aorta and its branches, leading to coronary ostial stenosis and myocardial ischemia (6). Giant cell arteritis in elderly patients may also cause myocardial infarction due to critical stenosis.

Medium-Vessel Vasculitis

Polyarteritis nodosa is a necrotizing vasculitis of medium-sized arteries that can lead to aneurysm formation, thrombosis, and myocardial infarction (7). Kawasaki disease in children is associated with coronary artery aneurysms and increased risk of myocardial infarction (8).

Small-Vessel Vasculitis

ANCA-associated vasculitis, such as granulomatosis with polyangiitis and microscopic polyangiitis, increases the risk of coronary microvascular dysfunction and myocardial injury (9).

Vasculitides and Acute Cerebrovascular Syndromes

Ischemic Stroke

CNS involvement is common in primary CNS vasculitis, but systemic vasculitides such as lupus vasculitis, Behçet's disease, and ANCA-associated vasculitis also increase ischemic stroke risk (10,11).

Hemorrhagic Stroke

Vessel wall fragility and aneurysm formation in vasculitis predispose to intracerebral hemorrhage (12). Polyarteritis nodosa and Behçet's disease are particularly associated with cerebral aneurysm rupture.

CNS Vasculitis

Primary angiitis of the CNS presents with recurrent ischemic events, seizures, and cognitive decline. Diagnosis requires angiography and sometimes biopsy confirmation due to nonspecific imaging findings (13).

Shared Mechanisms Between ACS and Stroke in Vasculitis

Endothelial dysfunction reduces nitric oxide bioavailability and vascular compliance (14). Inflammatory cytokines promote both coronary plaque rupture and cerebrovascular thrombosis (15). Hypercoagulability through platelet aggregation links myocardial and cerebral ischemia. Glucocorticoid therapy contributes by increasing hypertension, diabetes, and dyslipidemia risk (16).

Clinical Presentation

Patients with vasculitis may present with atypical ACS or stroke manifestations: chest pain with systemic symptoms such as fever, weight loss, and arthralgia, or neurological deficits with concurrent renal dysfunction and rash (17).

Diagnostic Approaches

Key tools include laboratory markers (ESR, CRP, ANCA, ANA), echocardiography, coronary angiography, MRI/CT angiography, and biopsy in select cases (18).

Management Strategies

Vasculitis Treatment

Glucocorticoids remain first-line therapy, with immunosuppressants (cyclophosphamide, rituximab, methotrexate) used for remission induction (19).

Management of ACS and Strok

PCI or CABG is used for ACS in vasculitis, with careful balancing of standard ACS medications. Thrombolysis or thrombectomy may be indicated for stroke, although risks must be considered (20).

Secondary Prevention

Aggressive control of cardiovascular risk factors and maintenance of vasculitis remission reduces recurrence risk (21).

Future Directions

Promising areas include biomarker discovery, advanced imaging for subclinical vascular inflammation, and development of biologics to reduce vascular complications (22).

Conclusion

Vasculitides significantly increase the risk of both ACS and acute cerebrovascular events through inflammation, endothelial dysfunction, and atherosclerosis. Integrating immunosuppressive therapies with vascular interventions may improve outcomes.

References

1. Jennette JC, Falk RJ, Bacon PA, et al. 2012 Revised International Chapel Hill Consensus Conference Nomenclature of Vasculitides. Arthritis Rheum. 2013;65(1):1–11.
2. Libby P. Inflammation in atherosclerosis. Arterioscler Thromb Vasc Biol. 2012;32(9):2045–2051.
3. Jennette JC, Xiao H, Falk RJ. Pathogenesis of vascular inflammation by anti-neutrophil cytoplasmic antibodies. J Am Soc Nephrol. 2006;17(5):1235–1242.
4. Esmon CT. Crosstalk between inflammation and thrombosis. Maturitas. 2004;47(4):305–314.
5. Shoenfeld Y, Gerli R, Doria A, et al. Accelerated atherosclerosis in autoimmune rheumatic diseases. Circulation. 2005;112(21):3337–3347.
6. Seyahi E, Ugurlu S. Vasculitis and the heart. Curr Opin Rheumatol. 2017;29(1):1–7.
7. Pagnoux C. Updates in systemic vasculitis. Best Pract Res Clin Rheumatol. 2017;31(1):111–123.
8. McCrindle BW, Rowley AH, Newburger JW, et al. Diagnosis, treatment, and long-term management of Kawasaki disease. Circulation. 2017;135(17):e927–e999.
9. Ntatsaki E, Watts RA, Scott DG. Epidemiology of ANCA-associated vasculitis. Rheum Dis Clin North Am. 2010;36(3):447–461.
10. Salvarani C, Brown RD Jr, Hunder GG. Adult primary central nervous system vasculitis. Lancet. 2012;380(9843):767–777.
11. Banerjee S, Chandra SR. Stroke in systemic vasculitis. Ann Indian Acad Neurol. 2011;14(Suppl 1):S45–S50.
12. Nakazawa D, Masuda S, Tomaru U, Ishizu A. Pathogenesis and therapeutic interventions for ANCA-associated vasculitis. Nat Rev Rheumatol. 2019;15(2):91–101.
13. Benseler SM, Silverman ED. Central nervous system vasculitis in children. Curr Opin Rheumatol. 2007;19(5):493–499.
14. Virdis A, Taddei S. Endothelial dysfunction in systemic vasculitis. Curr Opin Rheumatol. 2011;23(1):49–55.
15. Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. N Engl J Med. 2005;352(16):1685–1695.
16. Souverein PC, Berard A, Van Staa TP, et al. Use of oral glucocorticoids and risk of cardiovascular and cerebrovascular disease in a population-based case-control study. Heart. 2004;90(8):859–865.
17. Cid MC, Prieto-González S. Vasculitis and vascular involvement in systemic autoimmune diseases. Best Pract Res Clin Rheumatol. 2020;34(1):101473.
18. Terrier B, Mouthon L. Diagnostic work-up of vasculitides. Presse Med. 2013;42(4 Pt 2):575–582.

19. Stone JH, Merkel PA, Spiera R, et al. Rituximab versus cyclophosphamide for ANCA-associated vasculitis. N Engl J Med. 2010;363(3):221–232.
20. Powers WJ, Rabinstein AA, Ackerson T, et al. Guidelines for the early management of patients with acute ischemic stroke. Stroke. 2018;49(3):e46–e99.
21. Wallace ZS, Miloslavsky EM. Management of ANCA-associated vasculitis. BMJ. 2020;368:m421.
22. Terrier B, Guillevin L. Biologic therapies in vasculitis. Curr Opin Rheumatol. 2018;30(1):15–21.