Ischemic cerebrovascular disease

see also Cerebral ischemia, Ischemic stroke, transient ischemic attack or TIA.

Recognize the symptoms and signs of anterior and posterior circulation ischemia emphasizing carotid disease and contrasting it with hemorrhagic stroke.

Differentiate among the types of ischemic stroke: embolic, hemodynamic, lacunar.

Categorize etiologic factors of brain ischemia including atherosclerosis, cardiac disease, arterial dissection, fibromuscular dysplasia, vascolitis, venous thrombosis and hematologic disease.

Understand the treatment options in ischemic disease and their indications, including medical management, risk factor modification and surgical therapy.

Diagnose and monitor carotid occlusive disease using noninvasive methods and understand indications for angiography and carotid endarterectomy.

Knowledge of the symptoms and signs of occlusive cerebrovascular disease is necessary for effective diagnosis and management of these patients. Symptoms are often transient, may be subtle, and can be unappreciated by both patient and physician. As most occlusive cerebrovascular is secondary to atherosclerosis of the internal carotid artery, the following brief review will focus on patients with this particular problem.

Patients hospitalized in the Osaka Rosai Hospital for acute ischemic cerebrovascular disease from August 2002 to February 2018 were divided into two groups at February 2010.

Hashimoto et al., retrospectively identified patients with rheumatoid arthritis (RA). The incidence of RA, occurrence of acute exacerbation of inflammation due to causes other than synovitis preceding ischemic cerebrovascular disease (iCVD) (non-synovitis AEI), and serum C reactive protein (CRP) were compared.

In the first and second periods, 23/1203 patients (1.9%) and 22/1094 patients (2.0%) with acute iCVD had RA, respectively. Non-synovitis AEI was significantly less frequent in the second period (5%, n=1) than the first period (35%, n=8) ($p < 0.05$). CRP was significantly lower at iCVD onset in the second period (median and interquartile range: 2.72 [0.89-4.5] vs. 0.34 [0.12-1.19 mg/dl], $p<0.01$). Excluding 9 patients with non-synovitis AEI, CRP was still lower in the second period (1.21 [0.47-2.72] vs. 0.33 [0.11-0.98 mg/dl], $p < 0.01$). CRP levels before both iCVD and non-synovitis AEI tended to be lower in the second period (1.53 [0.3-2.78] vs. 0.69 [0.06-1.28 mg/dl], $p=0.059$). Two patients using tocilizumab developed iCVD despite persistently low CRP levels.

With progress in treatment, RA-related inflammation was better suppressed and CRP decreased, but the prevalence of RA among acute iCVD patients was unchanged. Strategies for tighter control of inflammation are needed, and a new biomarker may be required in patients using tocilizumab [1)].

1)

Hashimoto H, Kawamura M, Yukami T, Ishihara M, Bamba Y, Kaneshiro S, Tsuboi H, Yamamoto K.

Etiology of acute ischemic cerebrovascular disease associated with rheumatoid arthritis: Changes with progression of anti-inflammatory therapy. Eur J Neurol. 2018 Jul 11. doi: 10.1111/ene.13751. [Epub ahead of print] PubMed PMID: 29995999.