Abstract
The underlying pathophysiology of atrial fibrillation (AF) is aberrant and excessive electrical activities in atrium chamber. AF does not directly cause hemodynamic changes because blood can still passively pass following gravity force. Otherwise, blood stasis in left atrial appendage will start to coagulate and form potential thrombi. If left atrium suddenly contracts, the clot will split into minute pieces and course along systemic circulation.

Theoretically, cardioemboli can be passed into any arteries and arterioles. Some will flow through carotid circulation especially in the left side as it is more linear from aortic arch. Emboli will follow the carotid artery and obstruct any smaller diameter arteries/arterioles of its size.

If middle cerebral artery (MCA) flow is impeded, the expected manifestation is total anterior circulation infarction (TACI) which consists of sudden episode of unilateral weakness, sensory deficits, hemianopia, and aphasias. MCA lacks collateral flow and only relies on leptomeningeal artery supply. This demands thrombectomy or thrombolysis as soon as possible from the onset of stroke. The outcome is far superior than those treated with conventional therapy.

It can be seen that cardioembolic stroke causes greater manifestations, longer length of stay, and worse prognosis. Stroke incident can be prevented by detecting and treating AF properly in an early phase. The application of routine Holter monitoring should be a common in daily practice. Patient with paroxysmal AF will benefit more as they are undetected in weekly or monthly visits.

Anticoagulation is the gold standard of treatment in those with valvular or non-valvular AF. Either warfarin or NOAC can be given to patients with non-valvular AF based on stroke risk stratification score of CHA2DS2-VASc or HASBLED. It can be concluded that early diagnosis of AF is the key in preventing cardioembolic stroke.

Keywords: stroke, prevention, atrial fibrillation, NOAC