Cerebral microemboli and cognitive impairment

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Abstract

Transcranial Doppler Ultrasound (TCD) may be used to detect cerebral microemboli in patient groups with an increased stroke risk and during invasive cardiovascular examinations and operations. Although these microemboli do not cause immediate symptoms, there is growing evidence which suggests that they may cause cognitive impairment if they enter the cerebral circulation in significant numbers. This has been studied in detail in patients who have had coronary artery bypass surgery. In these patients, an association has been found between the number of intraoperative cerebral microemboli detected by transcranial Doppler and postoperative neuropsychological outcome.

It is also possible that cerebral microemboli may be the cause of cognitive impairment in patients with cerebrovascular disease. Cerebral microemboli are often found in patients with atherosclerosis, especially of the carotid arteries and aortic arch, and in patients with heart disease. There is also an increased risk for silent strokes and cognitive impairment in these patients.

Prospective clinical studies are therefore required to determine if continuous cerebral microembolization to the brain will lead to progressive cognitive impairment.

Keywords: Atherosclerosis; Cerebral embolism; Cognitive impairment; Heart surgery; Multifrequency Doppler; Silent strokes

1. Introduction

Transcranial Doppler Ultrasound (TCD) may be used to detect asymptomatic cerebral microemboli in patient groups with an increased stroke risk and during invasive cardiovascular examinations and operations [1–6]. This is possible because an embolus causes an increase in the amount of reflected ultrasound compared to that normally caused by the red blood cells (Fig. 1). Although these microemboli do not cause immediate symptoms, there is evidence which suggests that they may cause cognitive impairment if they enter the cerebral circulation in significant numbers. This has been studied in detail in patients who have had coronary artery bypass surgery. Following this operation 50–80% of patients have cognitive impairment at discharge, 20–50% at 6 weeks, and 10–30% at 6 months [7–10]. Cerebral microemboli have been implicated in the pathogenesis of this cognitive decline and there are some reports which have shown a positive association between the number of intraoperative cerebral microemboli detected by transcranial Doppler and postoperative neuropsychological outcome [11–13].

Autopsy studies following cardiopulmonary bypass have shown multiple focal dilatations or microaneurysms [14]. The walls of the affected arterioles or capillaries were apparently stretched, and their appearance suggested the effect of microembolic occlusion with subsequent disappearance of the embolus. These microvascular lesions have been called Small Capillary and Arteriolar Dilatations (SCADs), and they are strong evidence of widespread microembolism to the brain during bypass surgery.

It is also possible that cerebral microembolization may be the cause of cognitive impairment in patients with cardiovascular disease. Asymptomatic cerebral microemboli are often detected using TCD in patients with atherosclerosis, especially of the internal carotid arteries and the aortic arch [1], and several studies have reported an association between cognitive impairment and carotid atherosclerosis [15,16]. Silent infarcts are commonly observed in these patients on neuroimaging [17]. Silent strokes due to cerebral microemboli may therefore be the cause of cognitive decline in some of these patients. This supports other evidence which strongly suggests an overall downward shift in the elderly population towards lower levels of cognitive functioning with increasing age and greater atherosclerosis, indicating that a considerable proportion of cognitive impairment in the elderly population may be vascular in origin [18,19].
A similar mechanism may play a role in patients with heart disease where asymptomatic cerebral microemboli are also frequently detected using TCD. This includes patients with atrial fibrillation, coronary heart disease, valvular heart disease and mechanical heart valves (MHVs) [3,20–22]. Silent infarcts, which may be due to cerebral microemboli and decreased cognitive function, have been demonstrated in patients with atrial fibrillation [23,24].

The frequency of microemboli in patients with heart disease varies considerably with the highest prevalence and frequency in patients with mechanical heart valves (MHV) (Fig. 2). These patients may have several hundred microemboli per hour without experiencing clinical symptoms [3]. Assessment of the significance of these microemboli detected by TCD in this and other patient groups has been difficult due to the fact that it has been impossible to...
differentiate between solid and gaseous microemboli. This is now possible using a newly developed multifrequency transcranial Doppler (DWL), which insonates the emboli simultaneously with two different frequencies (2.0 and 2.5 MHz). Differentiation is possible due to the fact that solid microemboli reflect more ultrasound at the higher 2.5 MHz frequency compared to the lower 2.0 MHz, whereas the opposite is the case for gaseous microemboli (Fig. 3).

Studies have shown that although the majority of microemboli in MHV patients are gaseous, approximately 15% are due to solid elements [25,26]. The ability to determine the composition of these microemboli also provides the possibility of assessing their size. In MHV patients, the gaseous microemboli are approximately 4 µm and may therefore pass through the cerebral microcirculation, whereas this would seem very difficult for the solid microemboli which are approximately 130 µm in diameter (Fig. 4). It is these larger solid microemboli which may be the cause of silent strokes.

More than a million of these solid microemboli may enter the brain each year, and it is therefore possible that they may lead to progressive brain injury. Prospective clinical studies...
are therefore required to determine if continuous microembolization to the brain will lead to progressive cognitive impairment.

References


