Identification of “high risk” asymptomatic carotid stenosis: we need to get a better yield from invasive treatments

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Would you put your money on a stock option, with a 3% probability of totally losing your capital within 1 month and, if this disaster does not happen, a 1% higher yield each year, compared with your usual investment? Many readers (and the writer as well!) would answer “no thanks” to this offer. This is, roughly speaking, the situation of endarterectomy for asymptomatic carotid stenosis,¹ with a 3% perioperative stroke or death rate, and an absolute risk reduction of approximately 1% per annum over the first few years of follow-up.

However, were we able to identify a way of obtaining a higher yield (3 or 5% per year), we would accept the investment—that is, we would recommend the operation to those patients with a higher risk of events, and therefore a higher absolute risk reduction with endarterectomy. Therefore, we are looking for markers of risk for an apparently silent carotid plaque. In this issue of *J Neurol*

Neurosurg Psychiatry, Tang and colleagues² describe a new method of identifying inflammation in the plaque, which may be a significant risk factor for cerebrovascular events in the near future (*see page 1337*). They found that inflammatory activity, a putative marker of vulnerability of the plaque, was higher in those patients who had contralateral symptoms than in those without any history of cerebrovascular disorders, suggesting that inflammation of atheroma may be a systemic event, and that symptoms in one territory may increase the risk in other territories. Tang and colleagues² rightly point out that only prospective large scale trials can validate their suggestion. Also, I believe that a temporal factor should be taken into account; we know from systematic reviews³ that the risk of a new event in the territory of a symptomatic carotid stenosis (as well as the advantage of the operation) is much higher in the first few weeks after the clinical event; after this period, the lesion

is functionally no longer “symptomatic” and the advantage of endarterectomy is similar to that in asymptomatic patients.⁴

Therefore, we need to understand which biological factors make a plaque “symptomatic”, and certainly inflammation may be one of them. Since this, and possibly other processes, are thought to be systemic, then a pharmacological treatment could be a reasonable solution: statins had been advocated, but no definitive clinical results are available, and trials of pharmacological treatments are needed.

“We’ve got a better offer which perfectly suits your needs”; we are waiting for this sentence both for stock option purchases and for treatment of asymptomatic carotid stenosis. The use of new tools, as suggested by Tang and colleagues,² may well help to solve the puzzling problem of a treatment which still has, on average, a low appeal, but might be extremely important for some patients where we were able to correctly identify them.

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