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Incidence of stroke after myocardial infarction

Evidence presented from a recent community-based cohort designed study by Witt and colleagues (2005) suggests that the risk of stroke substantially increases following myocardial infarction (MI). While this suggestion is not new, this particular study was longitudinal in design, spanning a period of 2 decades with a median follow-up of 5.6 years (range 0 to 22.2 years). The study was somewhat limited as the cohort under investigation comprised a white population specific to Olmsted County, MN, USA and the measured outcome included only a review of medical records. Notwithstanding these limitations, the study provides some quite interesting epidemiological information regarding the incidence of stroke following MI (Witt et al 2005).

Epidemiological studies have demonstrated a link between coronary artery disease and stroke (Heyman et al 1980; Visser et al 1985; Rose 1989) with the association between coronary artery disease, MI and stroke being ascribed to a common pathophysiology, atherosclerosis (Hess et al 1993). Interestingly, prior to the era of thrombolytic and anticoagulation therapy, the incidence of stroke reported was approximately 2.4% in patients with acute MI (Thompson and Robinson 1978; Komrad et al 1984) with intracranial hemorrhage being quite a rare event. In the current climate in which thrombolytics and anticoagulants are employed in patients with MI, the incidence of nonhemorrhagic stroke seems to be less than 1.4%, whereas intracranial hemorrhage is reported as 1.6% or less (O'Connor et al 1990; Maggioni et al 1991; Longstreth et al 1993; Gore et al 1995). Views have been expressed that thrombolytic, anticoagulant, and antiplatelet therapy may be helpful in reducing the incidence of stroke (Mahaffey et al 1998; Chen et al 2005), but strong evidence in support of this view is not that apparent.

In the study by Witt and colleagues (2005) a marked increase in the incidence of stroke was found within 30 day post-MI. This amounted to a 44-fold (95% confidence interval [CI], 32 to 59) increase in the incidence of stroke in patients with MI when compared with the rate of stroke in the general population. A sharp decline followed thereafter in the incidence of stroke to 3.1-fold (95% CI, 2.0 to 4.5) within 31 days to one year. Furthermore, the rate of stroke following MI stabilized within 3–4 years (standardized morbidity ration being 1.6, 95% CI, 0.9 to 2.8), which was no different than in the general population for that region (Witt et al 2005).

Univariate analysis indicated that there is a higher risk of stroke following MI in older patients ($p < 0.001$), women ($p < 0.001$), diabetics ($p < 0.001$), hypertensives ($p < 0.001$), and those with previous history of stroke ($p < 0.001$) to mention some sub-groups. However, hyperlipidemia ($p = 0.114$) did not appear to be associated with a higher incidence of stroke after MI. It is assumed that vascular problems are a major contributor to vascular mortality, but surprisingly the evidence in this particular study does not strongly link the incidence of stroke after MI in patients with hyperlipidemia. While cardiovascular risk factors such as diabetes ($p < 0.001$) was found to be also independently associated with higher risk of stroke in this population, hypertension was not reported as an independent risk factor in the population studied. Older age ($p < 0.001$) and previous history of stroke ($p < 0.001$) were also reported as independent risk factors (Witt et al 2005).

There appears to be a significant ($p = 0.002$) increase in the risk of stroke after MI in patients on warfarin (relative risk [RR] 1.7, 95% CI, 1.22–2.36), whereas there

was no significant increase in the incidence of stroke in patients on thrombolytics ($p = 0.39$), aspirin ($p = 0.41$), low-molecular weight heparin ($p = 0.40$), or antiplatelet agents ($p = 0.36$). The difference between warfarin and other agents that affect blood physiology may relate to its complex pharmacokinetics and could possibly be associated with an increase in incidence of hemorrhagic stroke. Of the 273 strokes recorded in the population studied, 13 (5%) were due to intracerebral hemorrhage and 1 was subarachnoid, but it was not apparent if these patients were being treated with warfarin. Furthermore, it appears that a significantly ($p < 0.001$) higher occurrence of strokes following MI in patients on diuretics (RR 1.82, 95% CI, 1.42–2.33). Whether this observation is related to the use of diuretic agents or linked to the type of patients on diuretics remains unclear (Witt et al 2005).

While the study by Witt and colleagues (2005) does provide a unique perspective on the incidence of stroke after MI over 2 decades, the authors indicated some shortcomings such as its observational nature and the fact that data review did not permit for the determination of whether anticoagulant or antiplatelet medications were prescribed to treat or prevent stroke after MI, clearly limiting conclusions on the use of such medications and the risk of stroke. This study can perhaps set the foundation for future studies to assess the impact of anticoagulant and antiplatelet medications on the occurrence of stroke following MI in longitudinal design as well as in a less homogenous population.

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