Stroke Literature Synopsis: Clinical Science

(diagnosis and prognosis)

STROKE/2017/019655

December 2017

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Word Count: 839 in text

Disclosures: Nil

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In acute stroke we are lucky to have a number of efficacious treatments supported by a robust evidence-base. However, before we think about treatment we first need to make the diagnosis of stroke and make some estimate of likely outcome. This is not always straightforward and much of the skill, challenge and reward of stroke medicine comes from the diagnostic and prognostic decision making required for each patient. For this month's Literature Synopsis I have selected articles that are themed around diagnosis and prognosis. This is an area of stroke where I feel we need more research and I hope to see, and discuss, more articles on these themes in future Synopses.

I consider myself an evidence-based practitioner but there will always be areas of practice where we have limited research to guide decision making. In this context it is reassuring to see results that confirm something I had, based on experience, always assumed to be the case. Most stroke physicians will recognise that patients who have made recovery from a previous disabling stroke can appear to show a re-emergence of clinical deficits when they are subject to physiological stressors such as infection or metabolic disturbance. In a registry based case-control analysis using data from the Massachusetts General Hospital, the existence of this syndrome was proven (Topcuoglu et al Recrudescence of Deficits After Stroke Clinical and Imaging Phenotype, Triggers, and Risk Factors. JAMA Neurol. 2017;74(9):1048–1055) and was given a new name and abbreviation - post stroke recrudescence (PSR). The description of a typical case of PSR may give clinicians more certainty in making this diagnosis. In the case series, patients with PSR had a symptom complex that could occur many years after the index stroke. Symptoms were generally mild and lasted less than twenty-four hours. This is a nice diagnostic study, but I was left wondering about prognosis. Does a systemic illness with associated PSR have a poorer prognosis than a similar illness with no PSR?

One of the most difficult prognostic decisions is when to withhold or withdraw active stroke treatment. In a study of 60 patients with severe stroke admitted to two centres in the Netherlands, stroke physicians' estimates of prognosis were compared to the actual six month patient outcomes. (Geurts et al Predictive accuracy of physicians' estimates of outcome after severe stroke. PLOSOne. 2017 ONE12(9): e0184894) Physicians were relatively good at predicting death, one patient predicted to die, survived and one patient predicted to survive was dead at six months. However, predictions around functional outcome were less good and generally too optimistic (false positive rate 0.3). Predictions around quality of life were even less accurate. For a cohort with very severe initial stroke, I was surprised at the high proportions of patients reporting favourable quality of life. This suggests that my predictive accuracy is also limited and is a salient reminder of the dangers of making prognostic decisions too early.

Atrial fibrillation (AF) and stroke is an exemplar of an area where we are increasingly aware of the shortcomings in our prognostic understanding. Patients with 'true' paroxysmal AF, the definition of which varies from guideline to guideline, are at greater risk of cardioembolic stroke than those with sinus rhythm. Frustratingly, the most common result from ambulatory cardiac monitoring is neither sustained sinus nor AF, but bursts of atrial arrhythmia that are too short to meet any diagnostic after use. The Find-AF investigators used their data to describe the implications of supraventricular runs (Weber-Kruger et al Relevance of supraventricular runs detected after cerebral ischaemia. Neurology.2017,89:1545-1552.) The results of 3.7 years follow-up confirmed that even short supraventricular runs were associated with more stroke events and more incident AF. Given the frequency of these short runs of arrhythmia on ambulatory monitoring (around a third of patients in this study) this is an important finding. Now the important question is, what should we do with patients who have this abnormality? Anticoagulation would seem intuitive but the efficacy and economics of anticoagulation in patients who do not fulfil AF diagnostic criteria remains to be proven.

Can we better stratify the risk of incident AF? Certainly various modifiable risk factors for incident AF have been described. We know that high levels of alcohol consumption can predispose to AF, but the importance of moderate alcohol remains a subject of debate. We will never see a randomised controlled trial of therapeutic alcohol and so we are reliant on observational research, with all the caveats that come with such studies. A national registry based study from Norway (47002 participants, 1697 new AF diagnoses) gives a reasonably precise estimate of risk. (Gemes et al Does Moderate Drinking Increase the Risk of AF? JAHA2017;6:e007094) The authors describe a curvilinear relationship between alcohol and incident AF, but the risk of moderate alcohol (defined in this study as 1 drink per day for women and 2 drinks per day for men) was negligible, with a population attributable risk of 0.07 (95%CI:-0.01 to 0.13). This is reassuring news for my quality of life, so I'll drink to that. Cheers!

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