Obesity and migraine: A population study

To the Editor: We read with interest the recent article by Bigal et al.1 concerning migraine and obesity. They concluded that obesity is not comorbid to migraine but is significantly associated with the number of headache days, severity, and some clinical features. We would like to introduce our survey of headache in nurses who are working in our hospital. Almost 700 nurses participated in this survey, and 10% of them have migraine. According to the results, the proportion of migraine subjects with 10 or more headache days per month is nearly 10% in normal or underweight subjects. There was no one whose body mass index was greater than 25 and who had headache of 10 days or more.

We would like the authors to clarify the following points. Are those who are obese in childhood or adolescence at greater risk for developing migraine than those who are not? In addition, overweight people may have sleep apnea syndrome (SAS), hyperglycemia, and hypercholesteremia. These factors may also play a role in migraine. Migraine is associated with elevated cholesterol level2 and mitochondria plays a role in migraine pathogenesis.3

In addition, is it possible that reduction of body weight may improve headache in obese subjects? It would be interesting to investigate whether reduction of body weight in normal or underweight subjects would impact migraine.

Yasuo Iwasaki, Ken Ikeda, Tokyo, Japan

Disclosure: The authors report no conflicts of interest.

Reply from the Authors: We appreciate the opportunity to respond to Drs. Iwasaki, Ikeda, and Gilbert. Although obesity was not a risk factor for migraine in our study, we found that obese migraine sufferers had more frequent, painful and disabling headaches than those who were of normal weight.4 In separate studies, we also showed that obesity is a risk factor for incident chronic daily headache (headache >15 days per month) and that the association of obesity with transformed migraine is robust while the association of obesity with chronic tension type headache is modest.5,6

Gordon J. Gilbert, St. Petersburg, FL

Disclosure: The author reports no conflicts of interest.

Prospective study of symptomatic atherothrombotic intracranial stenoses: The GESICA study

To the Editor: I read with interest the article by Mazighi et al.1 on the natural history of symptomatic intracranial stenosis. Their population resembles that of the WASID trial regarding the risk factor profile.2 However, the reported rates of cerebrovascular events are misleading considering the composite endpoint alone. The annual rate of cerebrovascular events was 19% (38% at 2 years) although the rate of TIA was twice as frequent as stroke (12% vs 7%). Additionally, two thirds of patients who underwent angioplasty did so following a TIA, and the periprocedural rate of neurologic complications was 14%.

The composite endpoint issue is important because one is the apparent magnitude of risk when the composite endpoint is considered alone and the other is the relative importance of the different component endpoints to patients, particularly in a situation where we also showed that obesity is a risk factor for incident chronic daily headache (headache >15 days per month) and that the association of obesity with transformed migraine is robust while the association of obesity with chronic tension type headache is modest.5,6

Drs. Iwasaki, Ikeda, and Gilbert ask about the potential role of sleep apnea in the exacerbation of migraine in obese subjects. Although we cannot directly test this hypothesis, obesity and snoring are independent risk factors for chronic daily headache.7 If snoring is a reasonable proxy for sleep apnea, then the influence of obesity on migraine frequency appears to be independent of sleep apnea. This is an excellent area for further research.

Drs. Iwasaki and Ikeda also identify hyperlipidemia and mitochondrial dysfunction as factors that may confound or contribute to the relationship between migraine and obesity. We do not have the data to test these reasonable hypotheses. Obesity is a proinflammatory state, associated with hyperlipidemia, hypertension, and insulin resistance in the metabolic syndrome.8 Additional research is required to disentangle the contribution of these features, separately and in combination, to the worsening of migraine and the emergence of chronic daily headache.

Although we recently reviewed mitochondrial dysfunction in migraine,9 we do not know how this is connected with obesity. Nor do we know if the duration of obesity contributes to migraine exacerbations or if weight loss reduces headache frequency or severity.

The identification of factors that exacerbate migraine and contribute to its progression is in its infancy. We look forward to finding more satisfactory answers to the excellent questions raised in these letters.

Marcelo E. Bigal, Richard B. Lipton, Bronx, NY

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when an invasive procedure becomes a treatment alternative and it is associated with a high periprocedural risk.

When presented with an annual risk of a cerebrovascular event of 19% and a treatment procedure with a complication rate of 14%, one may feel compelled to get the angioplasty. However, if the risk of stroke is considered alone (7%) we might decide for medical treatment. Even when TIA and stroke have the same biological mechanism and the very high early risk of stroke that TIA portends, it can be argued that for patients or physicians a TIA may have different importance as outcome.

This problem is illustrated with the VA Cooperative Study on Carotid Endarterectomy. In that trial, the benefit of the procedure was observed only when the composite endpoint included crescendo TIA and it was outweighed by the risks when TIA was not considered. Some authors recommended that to base a clinical decision on a composite event one has to determine 1) if the component endpoints are of similar importance to patients, 2) if the endpoints occur with similar frequency, and 3) if the expected risk reductions are similar with treatment.

When there are wide variations between the components then the composite endpoint should be abandoned. Mazighi et al. did not need the addition of TIA to represent the high risk that patients carry. Moreover, death was a better component of a composite endpoint. I commend the authors’ restraint on recommending angioplasty or stent placement and acknowledging the need for further clinical trials.

Salvador Cruz-Flores, St. Louis, MO

Disclosure: The author reports no conflicts of interest.

Reply from the Authors: We appreciate Dr. Cruz-Flores’ comments and agree that the relevance of a composite criterion is certainly inferior to an individual endpoint. In the GESICA study, the recurrence of ischemic events in the territory of a symptomatic intracranial stenosis defined the major endpoint. To what extent TIA and ischemic stroke (IS) are different entities and represent components of a composite endpoint is unclear. They both constitute a recurrent cerebrovascular ischemic event, which is clearly different from a composite endpoint including stroke and myocardial infarction.

TIA and IS share pathogenic mechanisms, and in this specific population of patients with stroke (i.e., symptomatic intracranial stenosis), they are probably part of a continuum. The use of a conventional clinical definition for TIA (which does not include imaging) in the GESICA study suggests that a significant number of TIA may have been classified as IS if the new definition of TIA is used. TIA are often a concern for patients and physicians and they are a serious condition which require adequate therapeutic decisions. TIA are an important determinant of stroke, with risks of stroke reported as high as 12% within the first month and looking back after an IS, 23% of patients experience a TIA before their stroke, mainly in the preceding week.

Currently, endovascular treatment remains under investigation. The initial high complications rates are decreasing as a result of interventionists’ increasing experience and the development of dedicated devices. The most recent data report periprocedural risks between 5.8% and 9.5% and the long-term follow-up available in patients treated with intracranial angioplasty or stenting shows annual stroke rates ranging between 3.2% and 7.1%. These stroke rates constitute a better outcome compared to the 22% stroke rate in the highest risk cohort in the WASID study.

However, indications for endovascular intervention in patients with intracranial atherosclerotic remain to be established, but the identification of patients with the highest risk of recurrence of ischemic events will probably maximize the benefit/risk ratio of this technique.

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We expect that the underlying causes for the dementia in ET will become more apparent with rigorous postmortem studies of ET brains.9

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The authors report no conflicts of interest.

References

An almost missed leptomeningeal angioma in Sturge-Weber syndrome

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Although leptomeningeal angioma is the hallmark of Sturge-Weber syndrome (SWS),1 some patients with suspected SWS show no MRI evidence of pial contrast enhancement.2 A girl with a left port wine stain developed right-sided motor seizures. MRI was performed at 18 months of age. Post-gadolinium T1-weighted images (slice thickness: 2 mm) showed transmedullary enhancement in the left central region (figure) but no clear evidence of leptomeningeal angioma. MRI scanning was continued with a three-dimensional time-of-flight (TOF) MR venography to further evaluate possible venous abnormalities. The axial source images (slice thickness: 0.8 mm) showed left parietal leptomeningeal enhancement (figure) in 20 consecutive planes. Three-dimensional TOF MR venography may increase the chance of detecting a leptomeningeal angioma in children with suspected SWS.

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Figure. Axial MR images taken after gadolinium injection. (A) Early post-gadolinium T1-weighted images showed no evidence of leptomeningeal angioma, only transmedullary veins in the left central region (arrowheads). (B) Post-gadolinium source images from three-dimensional time-of-flight MR venography showed left superior parietal contrast enhancement consistent with a pial angioma (arrows), in addition to visualizing the deep veins.

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