Prevalence of Patent Foramen Ovale in Patients with Migraine

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SUMMARY

Cerebral embolization through a PFO is considered to be a possible cause of migraine attack, therefore the size of the PFO and its 'functional potential' should play an important etiological role in migraine. However, this relationship remains complex due to various methodological issues. We have highlighted some of the important issues related to the diagnostic methodology for PFO and the interpretation of the results.

Mere detection of the presence of PFO in patients with migraine may not delineate their true relationship for a better understanding and planning a definitive treatment. There is an urgent need for standardizing the diagnostic methods as well as the criteria for the grading the 'functional status' of PFO.

Key words: patent foramen ovale, migraine, transcranial Doppler
Sir: We read with great interest the study by Demirtas et al (2007) regarding the relationship between migraine and the presence of patent foramen ovale (PFO). There are some important considerations for establishing this association.

Compared to the general population, an increased incidence of PFO has been reported in patients suffering from migraine (Anzola et al. 1999). Migraine attack is believed to be triggered by cerebral embolization through a right-to-left intracardiac shunt (Sztajzel et al. 2002), therefore the size of the PFO and its ‘functional potential’ should play an important etiological role in migraine. This relationship however, remains complex due to various methodological issues.

Trans-esophageal echocardiography (TEE) is considered the gold-standard for the diagnosis of PFO. However, it is poorly tolerated by patients and the sedation during the procedure limits patients’ ability for performing an adequate valsalva maneuver. Although TEE helps in visualization of the PFO as well as estimation of its size, assessment of its ‘functional potential’ may not be accurate. Transcranial Doppler (TCD) has proven to be a reliable technique and has been reported to be more sensitive and specific than TEE (Jauss et al. 1994), for PFO detection as well quantification of its ‘functional potential’ (Belvis et al. 2006). Jeserum et al (2007) have recently shown that migraineurs have a higher grade right-to-left shunt (on TCD) despite similar atrial septal characteristics (on intracardiac echo), further emphasizing the importance of estimating the ‘functional potential’ of the PFO rather than its mere presence in patients with migraine.

Since cerebral embolization is considered as the cause of migraine aura as well as the following headache, demonstration of paradoxical emboli in the intracranial arteries is important. However the methodology for performing the right-to-left shunt detection by TCD remains variable and requires standardization. A mixture of 9 mL of normal saline with 1 mL of air and small amount of patients’ own blood (Spencer et al. 2004) remains the most accepted contrast solution. However, some studies (Domitrz et al. 2007) have used different amounts of these ‘ingradients’ to prepare the ‘contrast mixture’. The technique and the number of times this mixture is agitated through a 3-way connector (Sastry et al. 2007), number of injections and the position of the patient during PFO-testing (Lao et al. 2007) before the injections also affects the number of microemboli and, may influence the test results. The sensitivity of TCD may be enhanced by using power M-mode owing to its overlapping and contiguous multiple gates (Moehring et al. 2002).

The position of the patient during the PFO diagnostic testing needs special mention. Echocardiography, transthoracic or transesophageal, is traditionally performed in the left lateral position. Since the gaseous microemboli in the ‘contrast mixture’ are lighter and move up due to their buoyancy, the left lateral position is physiologically unreasonable. Since TCD can be easily performed any anatomic position, we demonstrated that changing body position with repeat injections can increase the grading of a right-to-left shunt and is well tolerated (Lao et al. 2007). Since PFO is usually located anteriorly and superiorly in the right atrium and helped by their buoyancy, sitting position may allow an increased number of microbubbles traversing the deficient atrial septum. Additionally, patients’ ability of performing an effective valsalva maneuver is not impaired during TCD.

We believe that mere detection of the presence of PFO in patients with migraine may not delineate their true relationship for a better understanding and planning a definitive treatment. There is an urgent need for standardizing the diagnostic methods as well as the criteria for grading the ‘functional status’ of PFO. One suggested method could be the use of power M-mode TCD and repetitive evaluations in different body positions with a uniformly prepared ‘air-saline-blood’ contrast mixture and using a quantitative grading scale (Spencer et al. 2004). This diagnostic methodology can be validated initially by performing power M-mode TCD simultaneously with TEE and then using the former as an alternative technique for the quantitative diagnosis of PFO.

References


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