Cryptogenic stroke is a form of cerebral vascular accident that has an unknown origin and is rarely associated with patent foramen ovale (PFO) and migraine headaches. This is an uncommon occurrence in young, healthy, active adults, and it is increasingly rare for the episode to occur during an athletic competition. Stroke is easily recognizable with its distinct signs and symptoms, but it is also easily confused with many of its differential diagnoses, such as seizures or head trauma, if the episode occurs during an athletic competition. This case report describes the stroke episode, incidence of migraine headaches, diagnostic testing, and surgical management of a college female volleyball athlete who suffered a cryptogenic stroke associated with PFO during an athletic competition. Diagnostic testing included magnetic resonance imaging with contrast, electroencephalograph, lower extremity Doppler testing, and a transesophageal echocardiograph with agitated saline study. Surgical correction of the PFO included a cardiac catheterization percutaneous procedure based on fluoroscopic and echocardiograph imaging. After release from physicians, the athlete returned to full participation in the sport of volleyball, where she competed for the next 2 years without complications. The patient has reported no symptoms from stroke or PFO closure procedure in 3 years, and migraine headaches have decreased in severity, frequency, and duration.

Keywords: cryptogenic stroke; patent foramen ovale; athlete; migraine headaches with aura; oral contraceptives

CASE REPORT

A 22-year-old female collegiate volleyball player presented with acute right-side facial droop, aphasia, and loss of muscular control and sensation in the right upper extremity during a volleyball match. The patient had a recent history of extended airline flight, was taking oral contraceptives, and had a history of MHA for the previous 10 years. No indications of prior cerebrovascular accidents or cardiac problems were present in the patient’s medical or family history. She had a normal 2-dimensional echocardiograph 1.5 years prior, during her preparticipation physical exam. Each athlete at this institution undergoes this testing as part of his or her initial preparticipation physical exam.

The patient was examined by the host team physician and team’s staff athletic trainer and was immediately transported to the nearest emergency medical facility. During transport and upon arrival to the emergency facility, the patient’s symptoms slowly resolved, and the patient was completely asymptomatic 20 to 25 minutes from onset. The emergency facility’s initial working diagnosis was syncope, facial droop, and right-hand numbness. After further evaluation by emergency physicians, a computed tomography scan of the brain without contrast was used to identify intracranial hemorrhage or extra-axial fluid collection, the result of which was negative. A computed tomography angiogram of the neck was selected to identify aneurism or arteriovenous malformation; again, the result was negative. Blood tests ruled out dehydration, electrolyte imbalance, and other causative factors. After initial blood tests, she was placed on intravenous saline for dehydration and anticoagulants because the working diagnosis at this time was transient ischemic attack and was admitted for...
Further testing and observation. Tests revealed borderline hypercholesterolemia and a genetic mutation of MTHFR gene (C677T) associated with premature cardiovascular disease and clotting. There was no known family history of clotting disorders reported in the athlete’s preparticipation physical exam or in discussion with the family.

Over the next 3 days, magnetic resonance imaging with contrast identified ischemia in the brain: a 2.6-cm anterior left insular and temporofrontal infarction and, just superiorly, a 6-mm infarction. Inferiorly, a left posterior temporoparietal infarction measuring 5 mm was found (Figure 1). Electroencephalograph was found to be within normal limits. A lower extremity Doppler test for deep vein thrombosis was used to search for the thrombus source, the result of which was negative. The tests confirmed that the athlete had suffered a cryptogenic stroke, and the estrogen-based oral contraceptives were thought to be a contributing factor to the development of the thrombus. A transesophageal echocardiograph with agitated saline study (bubble study) identified a 14-mm PFO without an atrial septal aneurysm (Figures 2 and 3). A neurologist and stroke specialist determined that the risks of not performing the PFO closure procedure were higher than the risks associated with surgical closure. A cardiac catheterization procedure based on fluoroscopic and echocardiograph imaging and an Amplatzer mesh closure device (AGA Medical Corporation, Plymouth, Minnesota) were used (Figure 2). A follow-up transesophageal echocardiograph confirmed the position and efficacy of the device. The patient was released from the hospital later that afternoon and placed on Plavix (Bristol-Myers Squibb/Sanofi Pharmaceuticals, New York, New York) and aspirin for 3 months postsurgery.

At 1 month poststroke, the patient was cleared for full athletic participation and instructed to discontinue oral contraceptives and to continue with 325 mg of aspirin indefinitely. She resumed full participation in competitive volleyball. No symptoms from the stroke or PFO closure have occurred in 3 years, and migraine headaches have decreased in severity, frequency, and duration.

**DISCUSSION**

Cryptogenic stroke should be ruled out with testing in a younger, healthy, physically active person who is participating in a sporting event and presents with a focal neurologic deficit. In some cases, the embolus is as small as 1 to 3 mm and can pass to the left ventricle through a PFO. Approximately 112,000 cryptogenic strokes are diagnosed each year in the United States, which is approximately 20% of the ischemic strokes that occur each year; of these, 30,000 to 60,000 occur in patients who have a PFO. Rarely does an athlete with a PFO have a stroke. Advanced cardiac diagnostics included in the preparticipation physical exam may not identify PFO. Autopsy has found that 46% of patients under the age of 55 who suffer cryptogenic stroke have a PFO. Patients under the age of 55 diagnosed with cryptogenic stroke are 5 times more likely to have PFO.
Predisposing factors for stroke can go unnoticed in healthy individuals and can include oral contraceptive use, migraine headaches, genetic mutations that affect clot formation, and PFO. In the general population, approximately 27% of people have PFO, and most cases remain undiagnosed. PFOs are present in 50% of those with MHA. At most institutions, athletes with MHA are not screened for PFO with a diagnostic transesophageal echocardiograph. Screening an athlete with MHA may be indicated if the patient’s symptoms are not controlled with medication and/or if other risk factors are present. Percutaneous PFO closure is considered a safe and effective treatment option for cryptogenic stroke patients. Seventy percent of patients with MHA have reduced occurrence and symptoms after PFO closure. In this case, migraine occurrence and severity were reduced after PFO closure. It is unknown if all patients with PFO and MHA should undergo closure. An athlete with a PFO without complications or additional risk factors should be evaluated by a cardiothoracic surgeon.

This report serves as an important reminder to team physicians and athletic trainers of the early recognition of the signs and symptoms of stroke so that emergency medical care can be provided to prevent a potential fatality. Although it is a rare occurrence in healthy, young, athletic individuals, stroke can occur during participation in sports training or competition.

REFERENCES

1. Chambers J. Should percutaneous devices be used to close a patent foramen ovale after cerebral infarction or TIA? Heart. 1999;82(5):537-538.
2. Landzberg MJ, Khairy P. Indications for the closure of patent foramen ovale. Heart. 2004;90(2):219-224.
3. Overell JR, Bone I, Lees KR. Interatrial septal abnormalities and stroke: a meta-analysis of case-control studies. Neurology. 2000;55(8):1172-1179.
4. Schwartz SM, Petitti DB, Siscovick DS, et al. Stroke and use of low-dose oral contraceptives in young women: a pooled analysis of two US studies. Stroke. 1998;29(11):2277-2284.
5. Seiler C. How should we assess patent foramen ovale? Heart. 2004;90(11):1245-1247.
6. Serra W, De Iaco G, Reverberi C, Gherli T. Pulmonary embolism and patent foramen ovale thrombosis: the key role of TEE. Cardiac Ultrasound. 2007;5:26.
7. Tobis MJ, Azarbal B. Does patent foramen ovale promote cryptogenic stroke and migraine headache? Tex Heart Inst J. 2005;32(3):362-365.
8. Walsh KP, Wilmshurst PT, Morrison WL. Transcatheter closure of patent foramen ovale using the Amplatzer septal occluder to prevent recurrence of neurological decompression illness in divers. Heart. 1999;81(3):257-262.
9. Windecker S, Meier B. Patent foramen ovale and atrial septal aneurysm: when and how should they be treated. ACC Curr J Rev. 2002;11:97-101.