Case Report
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Multidetector Row CT Detection of a Patent Foramen Ovale Causing Neurologic Deficits in an Adolescent: A Case Report[1]

청소년에서 만성적 신경증상과 연관된 난원공개존증의 심장 전산화단층촬영 소견: 증례 보고[1]

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A patent foramen ovale (PFO) is a persisting fetal circulation structural abnormality that can cause neurologic deficits such as migraine and cryptogenic stroke. Here we report a case of PFO diagnosed by cardiac multidetector row CT in an adolescent male with chronic migraine and stroke.

Index terms
Computed Tomography
Heart, Computed Tomography
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INTRODUCTION

A patent foramen ovale (PFO) is a congenital cardiac lesion that can cause migraines with aura and stroke (1). Echocardiography is a first line diagnostic imaging modality for PFO (2). However, CT techniques have been improving, and some interesting papers demonstrating that PFO can be diagnosed by ECG-gated cardiac CT angiography have been published (3, 4). They reported that it is less sensitive to detect PFO by CT than by echocardiography. Recently, using cardiac multidetector row CT (MDCT), we detected PFO in a male adolescent patient. The PFO had been the hidden cause of a long-standing migraine.

CASE REPORT

A 17-year-old male was admitted to the hospital with a severe headache. He had a past history of chronic migraine that had been diagnosed 5 years earlier. At that time, he presented with syncope, and CT and MR imaging of the brain showed chronic infarction at the left basal ganglia and corona radiata. Additionally, in magnetic resonance angiography images, no significant stenosis or occlusion was observed in either the carotid or intracranial arteries (Fig. 1A-D). The patient had no known congenital heart or coronary vessel diseases. He complained of continuing migraine, and a generalized tonic clonic seizure occurred one time. He had a normal transthoracic echocardiogram. He was diagnosed with cryptogenic stroke and was treated conservatively.

The patient's vital signs were within normal limits except that blood pressure was 140/80 mm Hg. On physical examination after admission, no abnormalities were noted. Laboratory tests showed a normal coagulation profile, normal vanillyl mandelic acid, epinephrine, norepinephrine, and metaneph-

rine. The measured lipid panel was TG 110 mg/dL, total cholesterol 135 mg/dL, LDL 93 mg/dL, HDL 32 mg/dL; these values were within normal limits.

He underwent ECG-gated cardiac MDCT for evaluating possible heart disease causing the cerebral infarction and excluding causes of hypertension. Cardiac CT was performed using a 64-channel scanner (Sensation 64; Siemens Medical Systems, Erlangen, Germany). An oral beta-blocker (80 mg of propranolol hydrochloride; Pranol, Dae Woong, Seoul, Korea) was administered to reduce the patient's heart rate 1 hour before examination. The acquisition protocol included a gantry rotation time of 420 msec, a collimation width of 1 mm, pitch 1.5, a tube voltage of 120 kV, and a current of 550 mA. To trigger the start of the scan, a real-time bolus tracking technique was used, and contrast enhancement was achieved with 80 mL of iopamide (Ultravist 370, Bayer Schering Pharma, Berlin, Germany) injected at a rate of 4 mL/sec, followed by an injection of 40 mL of saline at 4 mL/sec. CT scans showed that a jet of contrast material flowed from the left atrium into the right atrium (Fig. 1E, F). Definite thrombi in cardiac chambers and occlusion or tight stenosis in coronary arteries were not seen in the CT scans. We suspected PFO and recommended transesophageal echocardiography for confirmation of PFO. Transesophageal echocardiography with injection of agitated saline and a Valsalva maneuver showed leakage of microbubbles into the left atrium, confirming the diagnosis of PFO (Fig. 1G). The PFO was too small to be occluded by an intracardiac occlusive device. Therefore, he was treated with aspirin to prevent thrombus formation and discharged.

DISCUSSION

PFO is a normal fetal connection between the left and right atrium, allowing blood from the placenta to bypass the lungs. In about 75% of individuals, this connection is closed during the first month of life. However, in the other 25%, the patency

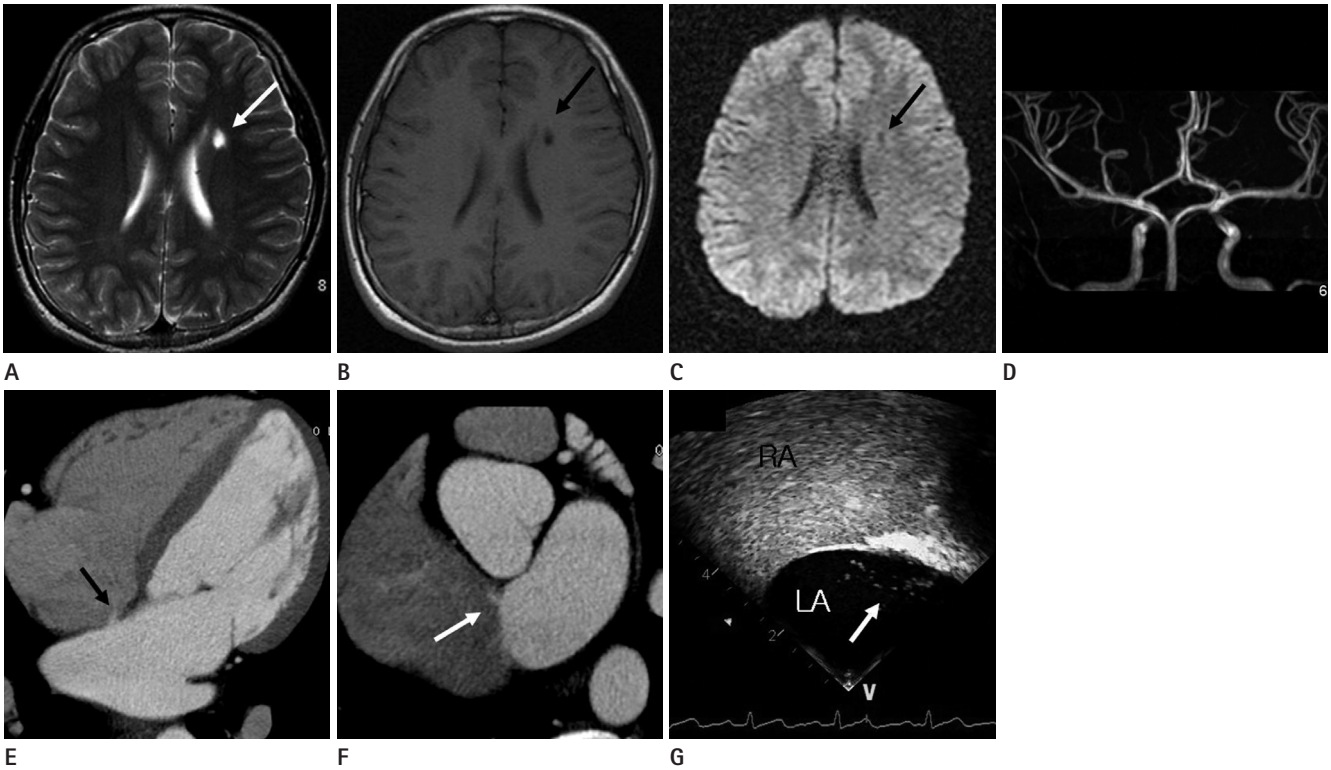

Fig. 1. A 17-year-old boy with a patent foramen ovale. A T2-weighted axial scan during brain MRI **(A)** reveals a high signal intensity lesion at the head of the left caudate nucleus (arrow). That lesion shows low signal intensity in T1- **(B)** and diffusion-weighted **(C)** axial images. An MR angiogram using the time-of-flight technique **(D)** shows no significant stenosis in the cerebral arteries. ECG-gated cardiac multidetector row CT 4-chamber scans **(E)** and short-axis scans **(F)** shows a small patent foramen ovale and leaking of a contrast jet from the left atrium into the right atrium. Transesophageal echocardiography with agitated saline injection and the Valsalva maneuver **(G)** shows shunting of microbubbles (arrow) from the right atrium to the left atrium.

persists into adult life (1). The presence of PFO is closely related to cryptogenic stroke in both older and younger patients (5). It is understood that PFO can cause ischemic stroke by means of paradoxical embolism (6), thrombus migration from the venous system to the left atrium (a migrating thrombus trapped in a PFO), and systemic circulation via a PFO (7). In addition, PFOs have been associated with migraine not only in adults but also in children. The mechanism is not fully understood. However, in a previous study, a relationship between headache and right-to-left shunt have been shown (8).

PFO is a virtual channel located in the inter-atrial septum, which causes a right-to-left shunt. As a result, diagnosis is made by direct visualization of the structure and its functional consequences (2). Generally, PFO is detected by echocardiography. In particular, agitated saline contrast medium injection and the Valsalva maneuver during transesophageal echocardiography is a highly sensitive method for the diagnosis of PFO (9). However, it is semi-invasive, and sedation makes performance of the Valsalva maneuver difficult. For this reason, transthoracic echocardiography (TTE) or transcranial Doppler ultrasonography are commonly used for screening purposes (2). In addition to these techniques, the recent literature has shown that one can achieve high diagnostic accuracy in detecting PFO by using ECG-gated cardiac CT angiography (3, 4).

Williamson et al. (3) used three criteria for detection of PFO in a cardiac CT image: a distinct "flap" in the left atrium at the expected location of septum primum, a continuous "column" of contrast material between the septum primum and septum secundum, and a "contrast jet" from the column into the right atrium. Similarly, Kim et al. (4) defined PFO by a contrast jet from the left atrium to the right atrium and a channel-like appearance of the inter-atrial septum. According to their results, a CT finding of a "contrast jet" had relatively lower sensitivity than the finding of a "channel-like appearance," but the opposite pattern was found for specificity (4). Also, there was a shunt direction discrepancy between transesophageal echocardiography (TEE) and CT. By definition, PFO is a valve which allows a right-to-left shunt to occur during elevated right atrium pressure. However, a previous study reported that PFO, like other left-sided cardiac lesions, is a channel that can induce left-to-right shunting by increasing left atrium size and pressure (10).

In our case, the patient had a cryptogenic stroke, and TTE missed his PFO due to its small size. However, in a cardiac CT image, there was a contrast jet from the left atrium into the right atrium. In addition, a small channel-like structure was found in the inter-atrial septum, and an atrial septal defect was excluded. We suspected PFO at CT, which was subsequently confirmed by TEE. We assumed that his recurrent migraines and cryptogenic stroke could be due to PFO.

Due to its relatively low sensitivity, cardiac MDCT cannot be a substitute for echocardiography as a gold standard in screening for PFO. However, when findings indicate a suspicion of PFO with cardiac MDCT, it has sufficient specificity as a diagnostic tool for PFO (4). Currently, cardiac MDCT is increasingly used for the evaluation of patients who have many risk factors for cardiovascular disease causing cerebrovascular symptoms. If we include the brain and neck in the cardiac scan range, we would be able to evaluate patients with suspected cryptogenic stroke not only for brain lesions that could influence prognosis, but also for underlying cardiac and carotid causes simultaneously (11).

In conclusion, our case demonstrates the usefulness of ECG-gated cardiac MDCT for PFO detection. It is also a powerful tool for the evaluation of the underlying cause of cryptogenic stroke. Cardiac MDCT may be a robust diagnostic modality of PFO that improves the chance of early diagnosis and prognosis.

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청소년에서 만성적 신경증상과 연관된 난원공개존증의 심장 전산화단층촬영 소견: 증례 보고[1]

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난원공개존증은 정상 태아순환구조물로 출생 후에도 남아있는 경우 편두통이나 잠복뇌졸중 등과 같은 신경증상의 원인이 된다. 저자들은 잠복뇌졸중이 있었던 17세 남자의 간과되었던 기저 질환인 난원공개존증 1예를 심장 전산화단층촬영으로 진단하였기에 이를 보고한다.

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