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Case Report

Meticulous blood pressure control is mandatory for symptomatic primitive trigeminal artery

Abstract

One 57-year-old lady with uncontrolled hypertension and right-side pupil-sparing oculomotor palsy visited our emergency department. Computed tomographic angiography revealed right-side persistent primitive trigeminal artery. Her symptoms dramatically improved after her blood pressure went under control. To our knowledge, this is the first case report that describes the relation between blood pressure control and symptomatic improvement of oculomotor palsy due to primitive trigeminal artery. Diagnostic approach, image modalities, and treatment options for oculomotor palsy are also reviewed.

Primitive trigeminal artery is the most common persistent primitive carotid-basilar anastomosis with occurrence arranging 0.1% to 0.6% in angiography, mostly as an incidental finding [1,2]. In embryonic stage, primitive trigeminal artery serves most of the blood supply to the hindbrain and regresses after development of the posterior communicating and vertebral arteries [2]. Persistent primitive trigeminal artery may also, though rare, contribute to cranial nerve dysfunction including trigeminal neuralgia, hemifacial spasm, and incomplete oculomotor palsy [3-5]. We report one 57-year-old lady who had hypertension and visited our emergency department due to pupil-sparing oculomotor palsy secondary to primitive trigeminal artery. Oculomotor palsy responded well to adequate blood pressure control in our patient. This may serve as a clue to proper treatment of symptomatic primitive trigeminal artery in the emergency department setting.

We report one 57-year-old lady who visited our emergency department due to sudden onset of right-side ptosis for one week. She had medical history of hypertension without medication control. She also denied head trauma history. Meningeal signs were absent. Initial physical examination revealed right-side pupil-sparing oculomotor palsy. No other neurologic deficits were present. Her blood glucose level and hemoglobin A1c (HbA1c) were also within normal limit. High blood pressure was found upon arrival (systolic, 170 mm Hg; pulse pressure, 66 mm Hg). To rule out life-threatening etiologies such as aneurysm or tumor, we arranged computed tomography with angiography. Computed tomographic angiography (GE Lightspeed VCT KV/mA/rotation time [s], 120 kv/smart mA [100-330]/1.0 s; IV contrast volume/type/rate, 80 mL omni 350/4 mL/s) showed one serpiginous vascular structure arising from right intracranial internal carotid artery through right anterior portion of right ambient cistern and continuously to basilar artery and bilateral posterior cerebral artery, indicating primitive trigeminal artery (Fig. 1A, B). Symptomatic unilateral persistent primitive trigeminal artery with pupil-sparing oculomotor palsy was diagnosed (Fig. 1C). We later started oral labetolol to control her blood pressure during emergency department stay. Her symptoms dramatically improved as her blood pressure went under control (systolic, 127 mm Hg; pulse pressure, 43 mm Hg). Ptosis has subsided on day 3 since arrival, and she received neurology outpatient follow-up thereafter. No recurrence of oculomotor palsy was found.

The most common etiologies of oculomotor palsy remain ischemia (mostly diabetic third nerve palsy) [6]. Other ischemia causes include hypertension, giant cell arteritis, and systemic lupus erythematosus [7]. Oculomotor nerve palsy may also herald life-threatening conditions such as brainstem infarction, aneurysm, trauma, and neoplasm [8]. Nonisolated oculomotor nerve palsy warrants neurologic imaging tailored to accompanied neurologic signs. The term *pupil-sparing* indicates that the pupil remains normal and reactive in the setting of extraocular muscle dysfunction [9]. It is an important hint that favors microvascular ischemia largely owing to the more abundant collateral blood supply for the peripherally located papillary fibers than the central nerve trunk [10]. On the other hand, non–papillary-sparing oculomotor palsy often leads to the suspicion of compressive lesion, especially posterior communicating artery aneurysm [11]. As much as 10% to 20% diabetic third nerve palsy, however, present non–pupil-sparing lesion [12], and compression lesions may also present with pupil-sparing lesions [11]. The reason why persistent primitive trigeminal artery, in contrary to posterior communicating aneurysm, commonly lead to pupil-sparing lesion may be its pulsatile injury [3] in nature rather than compressive injury. Trigeminal artery may itself play a role in additional blood supply to oculomotor nerve. Radiologically, persistent primitive trigeminal artery is an abnormal flow void coming off the posterior aspect of the cavernous internal carotid artery and running a somewhat horizontal course

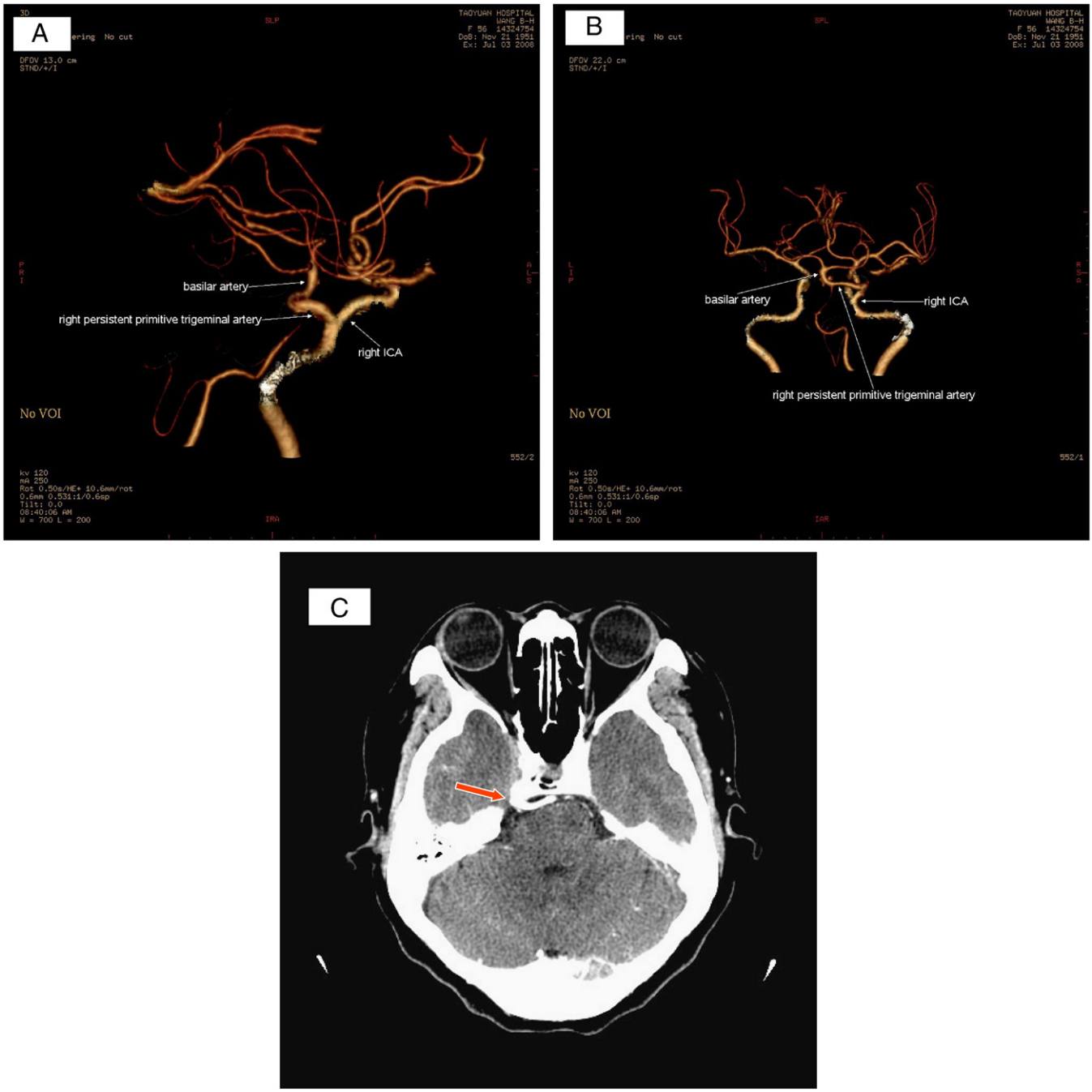

Fig. 1 A and B, Primitive trigeminal artery originated from right internal carotid artery, creating the "tau (τ) sign." C, Primitive trigeminal artery circled around over right anterior aspect of middle brain stem where right oculomotor nerve just leave off (arrow).

posteriorly to the basilar artery, known as the τ *sign* [13]. Pulsatile injury of primitive trigeminal artery to adjacent cranial nerve have been postulated (neurovascular compression syndrome) in symptomatic patients [3,14,15]. Similar concepts have also been proposed in the etiology of hemifacial palsy [16,17]. Patients with self-limited course often report resolution of symptoms for weeks to months [3,14]. Those with ischemic third nerve palsy usually recover after 2 to 4 months under treatment [7]. Steroid therapy have been recommended in symptomatic primitive trigeminal artery, and surgery was thought to be reserved for whose symptoms are refractory to medical treatment [3]. Reduction of blood pressure was also found to be effective in our case. We hypothesized that it may act through reducing neurovascular compression [3,15]. Our hypothesis is further supported by the close anatomical relation [1] and dramatic clinical course. Furthermore, image study of choice for isolated oculomotor palsy remains in debate. Magnetic resonance angiography has long been favored because of its high sensitivity for detecting aneurysms [18]. Computed tomographic angiography,

however, is now gaining its clinical importance [19]. In the latest study, Mathew et al [20] has proposed computed tomographic angiography as safe and effective in detecting intracranial aneurysms in isolated third nerve palsy.

In conclusion, persistent primitive trigeminal artery may be a rare cause of isolated pupil-sparing oculomotor palsy. Magnetic resonance angiography and computed tomographic angiography are both suitable imaging tools. Meticulous control of blood pressure is mandatory in symptomatic trigeminal artery in the emergency department setting.

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