

Hemicrania Continua Subsequent to Vertebral Artery Dissection: A Case Report

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We herein report the case of a 45-year-old woman who developed a continuous hemicranial headache subsequent to vertebral artery dissection (VAD). After remission of VAD, the patient repeatedly experienced right forehead and temporal region throbbing headache, accompanied by nausea, ocular hyperemia and lacrimation of the right eye, nasal congestion, and rhinorrhea. Magnetic resonance angiography did not reveal the recurrence of dissection. Daily use of indomethacin (190.8 mg/day) showed an excellent effect on the headache, suggesting that the patient had developed hemicrania continua subsequent to VAD.

Key Words: Hemicranias continua—vertebral artery dissection—headache—indomethacin

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Introduction

Hemicrania continua (HC)-like headache subsequent to cervical artery dissections (CADs) have been rarely reported.¹⁻³ All of the reported case occurred on the internal carotid artery (ICA) and external carotid artery (ECA). We herein report a patient with vertebral artery dissection (VAD) who developed a continuous hemicranial headache later. During 2 years of follow-up, the patient repeatedly experienced severe headache. We suspected recurrence of VAD several times, but finally diagnosed the patient with HC based on the excellent response to indomethacin.

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Case Presentation

The case is a 45-year-old woman who had no history of headache, alcohol abuse, or smoking. The patient visited to our hospital with a 3-weeks history of periodic intense headache. She had initially experienced a throbbing pain in her right forehead with nausea. Migraine treatment did not improve the symptoms. The characteristic of the pain gradually transformed, and she was complaining of continuous right occipital and neck pain when she visited our hospital. There were no neurological symptoms, except for severe headache. Magnetic resonance angiography (MRA) revealed dissection of the right vertebral artery (VAD; Fig. 1A-C). There were no complications of hemorrhaging or infarction. The patient was treated conservatively with bed rest, blood pressure control, and acetaminophen. The symptoms gradually improved. Nine days of hospital care were necessary until she could be discharged home.

The patient underwent follow-up MRA 1 and 3 months after the onset of VAD. Although the double-lumen sign of the right VA remained after 1 month (Fig 1D), it disappeared after 3 months (Fig 1E), suggesting closure of the false lumen. The patient was symptom-free between the discharge and follow-up study 3 months later.

Several days after follow-up MRA, however, she experienced headache again. The characteristic of the headache this time was right forehead and temporal region

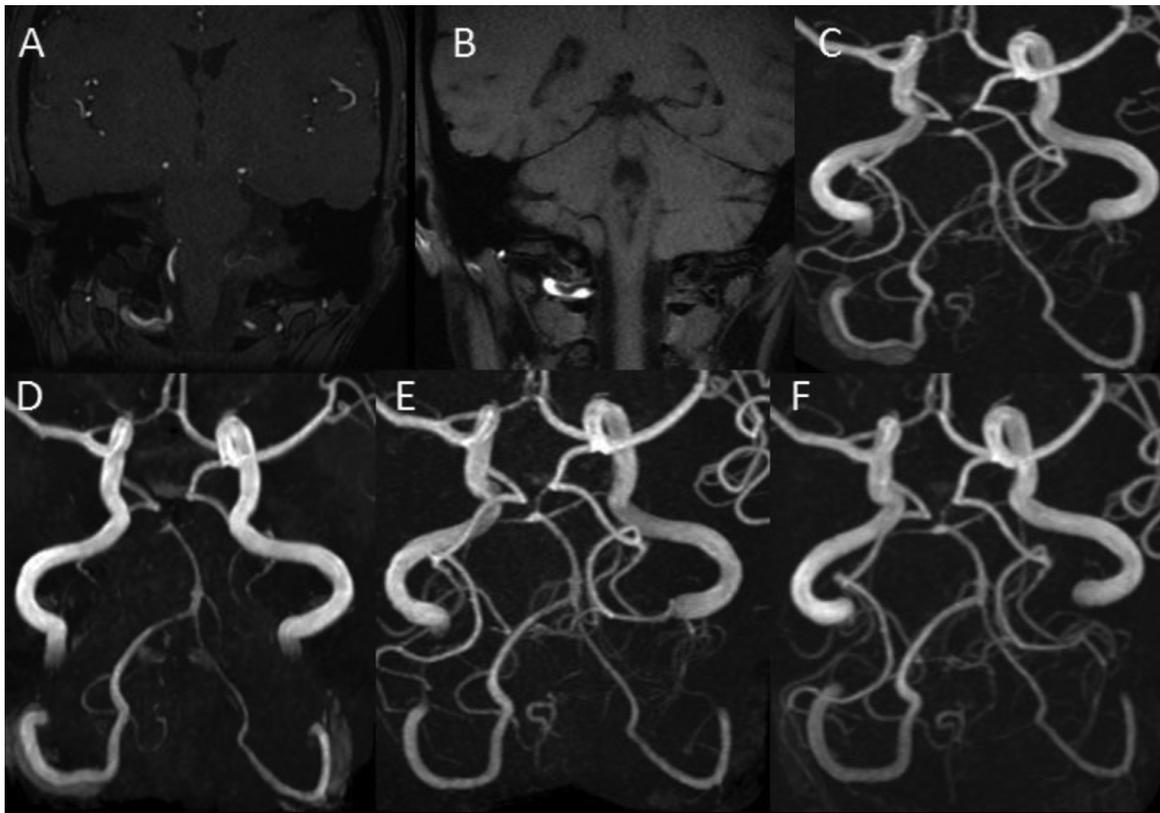


Figure 1. Imaging studies of vertebral artery dissection. Coronal section of MRA original image showing double-lumen sign (A, arrow head), the same coronal section of VISTA (Volume ISotropic T2w Acquisition) as in 1A (B), and MRA (C). 3D-MRA findings 3 months after the onset of VAD (D). Follow-up MRA performed 13 months after the onset of VAD (E). Follow-up MRA performed 19 months after the onset of VAD (F).

throbbing with moderate to severe daily pain, sometimes with nausea, ocular hyperemia and lacrimation of the right eye, nasal congestion, and rhinorrhea. The recurrence of dissection was suspected, but MRA showed no evidence of dissection. Nonsteroidal anti-inflammatory drugs (NSAIDs) and indomethacin farnesyl (a prodrug of indomethacin, up to a daily dose of 100 mg) as abortive agents, and lomerizine (10 mg/day) as a prophylactic agent were partially effective but did not lead to immediate total relief. Mild to moderate continuous headache with occasional worsening persisted for more than 3 months. Seven months after the onset of dissection, the headache showed gradual improvement under the same medications. All abortive and prophylactic drugs were stopped after 3 months.

Fifteen months after the first episode, the patient experienced a relapse of right headache again. The characteristics of the headache were continuous mild to moderate pain with occasional worsening, accompanying by nausea, ocular hyperemia and lacrimation of right eye. Nonsteroidal anti-inflammatory drugs, lomerizine, and amitriptyline (up to 20 mg/day) failed to improve her symptoms. At 19 months after the first onset, she visited our hospital because the headache had become severe and continuous. MRA showed no signs of recurrence of

dissection (Fig 1F). We administered 300 mg/day of indomethacin farnesyl. Regular use of indomethacin was remarkably effective, and the patient achieved total pain relief. The following characteristics of the headache are consistent with HC: unilateral headache, present for longer than 3 months, with exacerbations of moderate or greater intensity, conjunctival injection, lacrimation, nasal congestion, rhinorrhea, a sense of restlessness, and a remarkable response to indomethacin. Indomethacin was slowly tapered, and there was no relapse of headache at 24 months after the onset of VAD.

Discussion

HC subsequently to CADs has rarely been reported, with only 2 case reports^{1,2} and 5 case series available.³ Six cases involved ICA dissection, and a single case involved external carotid artery dissection. To our knowledge, the present case is first report of HC subsequent to VAD. In the previous cases, the effective dose of indomethacin ranged from to 225 mg/day. The longest duration between the onset of CADs and relapse of headache was 7 weeks. Our case required 190.8 mg of indomethacin (corresponding to 300 mg/day of indomethacin farnesyl) to obtain total relief, and the duration between the onset

of dissection and HC (3 months) was longer than in previous cases. The occasional use of indomethacin farnesyl (up to 100 mg/day) showed insufficient efficacy on the headache, suggesting that the use of an adequate amount use indomethacin was important for obtaining total relief.

In the present case, the patient presented with a headache satisfying the ICHD-3 criteria of HC, except for “E: Not better accounted for by another ICHD-3 diagnosis”. The final diagnosis of the headache depends on the interpretation of preceding VAD. There are 2 possibilities; mimic-HC as a symptom of VAD (ICHD3 code 6: Headache attributed to cranial or cervical vascular disorder), and HC occurring after complete VAD remission (ICHD3 code 3.4: Hemicrania continua). The previous 7 cases were reported in the context of secondary HC.¹⁻³ The headache induced by the VAD occurred in the posterior part of the head in 83% of patients and showed steady pain in 56% and pulsating pain in 44%.⁴ Thus, the headache associated with VAD is not specific and may resemble HC. However, the recurrence of spontaneous CADs over long-term follow-up is rare.^{5,6} In the present case, the abnormalities in the MRA findings associated with VAD disappeared (Fig 1E), and no other abnormalities were found in follow-up MRA when a recurrence of VAD was suspected. Therefore, repeated headache is, at least on its own, not a symptom of VAD recurrence. Although the underlying pathology of the relationship between VAD and HC is poorly understood, sympathetic neural supply to the pterygopalatine ganglion via internal carotid plexus may contribute to the pain with autonomic symptoms.^{7,8} Further accumulation of similar cases is necessary.

Declaration of Competing Interest

None of the authors have any conflicts of interest to declare.

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