ISCHEMIC INFARCTION DISCLOSE AORTIC DISSECTION - A CASE REPORT

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Abstract

Untreated hypertension is highly prevalent and an important risk factor for hemorrhagic stroke and aortic dissection. Aortic dissection masquerading as ischemic stroke is practically challenging. We report a patient who initially presented as having a hemorrhagic stroke, following with ischemic stroke several days later and was finally discovered to have aortic dissection. The patient underwent craniectomy, ventriculostomy catheter installation, osmotic diuretics, analgesia, and sedation to maintain ICP within the acceptable range. Our therapeutic goal of blood pressure is the lowest level commensurate with adequate cerebral and vital organ perfusion. Although very rare, we should always remind ourselves that aortic dissection may occur after a hypertensive hemorrhagic stroke, and then present simultaneously with ischemic stroke as in our case.

Key words: Aortic dissection, Stroke, Hypertension

Case presentation

A 42-year-old male was admitted to the emergency department after the onset of slurred speech and right-sided weakness. He had a history of smoking for more than 20 years and hypertension without medication for several years. His occupation is car cleaning. On arrival, his blood pressure was 227/113 mmHg, his body temperature was 36.6°C and his pulse was 76 beats/min. There were no murmur, gallop or rub in his heart sounds. A pulmonary examination showed no jugular venous distension and breath sounds were equal bilaterally. Carotid pulses present with no vascular bruits. The neurologic examination revealed E4V3M6 on Glasgow coma scale and right hemiplegia with Babinski sign.

Laboratory data showed troponin I 0.065 ng/ml. Electrocardiogram revealed left ventricular hypertrophy, ST elevation on V1 and T-wave inversion on V2-V6. Chest roentogram showed borderline cardiomegaly, bilateral increasing lung marking and tortuosity of the aortic arch. Other laboratory test results were within normal range. Cranial computed tomography (CT) disclosed left putaminal hemorrhage (Fig. 1a). The patient was then treated in the intensive care unit. The cardiac enzymes did not further increasing on follow-up. During the hospitalization, he had high mean arterial pressure around 130 mmHg. Under the impression of refractory hypertension, we consulted the cardiologist for better blood pressure control.
control and the nephrologist for the possibility of secondary forms of hypertension such as pheochromocytoma. The biochemistry data of his urine showed vanillyl mandelic acid (VMA) 10.04 mg/24 hours, others (PRA, norepinephrine, epinephrine, dopaminealdactone and rennin) were within normal limits. The elevated VMA was thought to be stress-related. Renal echogram did not find adrenal tumor.

The patient’s consciousness became drowsy and complicated with systolic blood pressure higher than usual on day 6. Head CT showed infarction at posterior territory of left middle cerebral artery (MCA) (Fig. 1b). Cardiac echogram, transcranial color-coded duplex (TCD) scanning and neck duplex scanning were arranged. Cardiologist suspected aortic dissection after doing cardiac echogram. The diagnosis of acute aortic dissection (AAD), extending from the aortic arch through the entire length of the descending aorta to bifurcation (Stanford type B, see Fig. C), was confirmed with urgent CT (Fig. 2a). The chest X-ray on the same day also showed mediastinum silhouette was wider than that of day 1 (Fig. 2b).

The patient was treated medically in his aortic dissection. However, his increased intracranial pressure (ICP) made the control of blood pressure more difficult. Thus he underwent craniectomy and ventriculostomy catheter installation to ICP monitoring and adjustment. We gave serial abdominal, neurologic, and pulse examinations to detect visceral, limb, or spinal ischemia.

Follow-up chest CT 14 days later revealed a thrombosed false lumen in descending aorta (Fig. 2c).

We transferred the patient to the service of physical medicine and rehabilitation on the 45th hospital day. Three months after the insult, he was in a fair status (E4V4-5M6, right upper limb palsy, right leg muscle power MRC (medical research council) grade 4-, and walking with a stick).

Discussion

Chest pain is an important cardinal sign of AAD and can be hindered in patients with impaired consciousness, as in our case, further complicating the evaluation.1,2

The findings on physical examination most typically associated with aortic dissection-pulse deficits, the murmur of aortic regurgitation, and neurological manifestations-are more characteristic of proximal than of distal dissection.
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Cerebral ischemia/stroke is the most common neurologic manifestation associated with aortic dissection. In up to 10% of cases an ischemic stroke may be the first clinical sign of aortic dissection. In about one third of patients with Stanford type A aortic dissection and less frequently in those with type B dissection. The occurrence of neurological sequelae occurs in about one third of patients with Stanford type A aortic dissection and less frequently in those with type B dissection. The etiology of stroke during acute aortic dissection may be multifactorial and the manifestations are extremely variable. This is beyond our discussion. According to the report of aortic dissection in Taiwan by Wei-Ber Liao et al in 1995, hypertension (85%) was the major predisposing factor with another 7% having Marfan syndrome. Hypertension is a common finding on physical examination and is present in 70% of patients with distal aortic dissection, but in only 36 percent with proximal dissection.

The most common abnormality seen on a chest radiograph in cases of aortic dissection is widening of the aortic silhouette, which appears in 81 to 90 percent of cases. But the findings are nonspecific and rarely diagnostic. A plain chest radiograph is inadequate to rule out aortic dissection. The sensitivity of CT scanning for acute aortic dissection was 100% and specificity was 96%. High-quality helical CT techniques can be useful as the primary imaging modality in the evaluation of acute aortic dissection. CT angiography, including the aorta and carotid arteries, can also be performed quickly; it is a sensitive procedure for confirming aortic and cervical artery dissection.

Case fatality in stroke obeys a U-shaped relationship: blood pressures that are either too low or too high are associated with worse outcomes both in ischemic stroke and in intracerebral hemorrhage. Current guidelines support permissive hypertension in the treatment of acute is-
Comorbid conditions such as aortic dissection would override the guidelines for permissive hypertension. Higher or lower blood pressure becomes a dilemma in this condition. We use craniectomy to increase space for the swelling brain and ICP as a guide to adjust the use of osmotic diuretics and antihypertensive drugs.

Acute aortic dissection is a potentially life-threatening condition that requires prompt and accurate diagnosis to initiate appropriate surgical intervention or medical treatment. This case shows the difficulty of acute aortic dissection diagnosis when the presenting symptoms and signs are atypical. Although very rare, we should always remind ourselves that aortic dissection may occur after a hypertensive hemorrhagic stroke, and then present simultaneously with ischemic stroke as in our case.

References

因缺血性腦中風而發現主動脈剝離：病例報告

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摘要

高血壓未治療在臨床上常見且是出血性腦中風及主動脈剝離之危險因子。
主動脈剝離有時會以中風來表現，對臨床診斷是一大考驗。我們報告一例因出血性腦中風住院，又繼發缺血性腦中風及主動脈剝離。我們藉由對患者施以顱骨切除，置入腦室引流管，滲透性利尿劑，止痛及鎮靜等方法來控制顱內壓。
血壓之控制目標則是在能夠維持腦及重要器官之血液灌流。雖然這類病例很少
但我們仍不要忘記其發生之可能。

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