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## MEGADOLICHOBASILAR ANOMALY ASSOCIATED WITH BRAIN STEM INFARCTION: A CASE REPORT

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A rare case of megadolichobasilar anomaly associated with localized brain stem infarction showing right hemiparesis and disturbance of consciousness is described. The etiologic mechanisms of brain stem infarction in this anomaly are discussed. The tortuosity and dilatation of the basilar artery were thought to play an important role in the pathogenesis of the brain stem infarction.

#### KEY WORDS:

Megadolichobasilar anomaly; Hemiparesis; Brain stem infarction; Basilar paramedian branches; Tortuosity; Kinking

Megadolichobasilar anomaly (MDBA) is defined as an abnormal basilar artery that shows marked dilatation and tortuosity. Many cases have been reported with regard to the complications due to the compression to the brain stem and/or cranial nerves. We report a rare case of MDBA associated with localized brain stem infarction diagnosed by cranial computed tomography (CT).

#### CASE REPORT

A 65-year-old woman presented with a 3-day history of acute headache, nausea, and right hemipa-

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resis. She had been in generally good health before admission and there was no history of transient ischemic attack, facial spasm, or trigeminal neuralgia.

On admission she was in a deep stuporous state. Right hemiparesis was noted by noxious stimulation. Deep tendon reflexes were increased and Babinski's sign was seen in the right side. Her eyes conjugately deviated to the left side. She was incontinent in urination. Vital signs (blood pressure, pulse rate, and respiration) were stable. Further examinations could not be carried out because the patient was uncooperative.

Routine blood examinations including blood coagulation studies were normal. Plain CT revealed tubular high-density areas with partial calcification in the prepontine, interpeduncular, and basal cistern as well as in the carotid arteries. These highdensity areas were partially enhanced after intravenous injection of contrast medium. We consequently diagnosed the MDBA (Figure 1).

With respect to the brain stem infarction, plain CT on admission disclosed no low-density area in the pontine base (Figure 2). However, the follow-up CT scan 10 days after admission revealed a clear low-density area localized in the left half of the midbrain extending to the left half of the pontine base. This led to the diagnosis of brain stem infarction (Figure 3). The patient gradually deteriorated. She developed pneumonia and died 30 days after admission.

### DISCUSSION

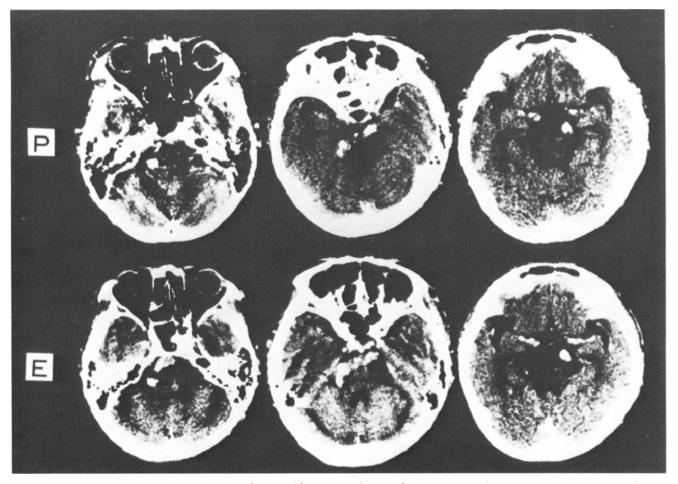
A portion of normal basilar bifurcation is found within a surface that consists of one quadrant of a circle with a radius of 20 mm, with its center close to the dorsum sellae (1). It is suggested that a tor-

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**FIGURE 1.** These CT series showed the abnormal basilar and carotid arteries. On plain CT (P, upper row), tubular high-density areas were seen in the preportine, interpeduncular, and basal cistern. Note that these areas were enhanced after venous injection of contrast medium (E, lower row).

tuous and dilatated basilar artery, the MDBA, causes compression to the cranial nerves (2). Moreover, cases of MDBA are sometimes accompanied by hydrocephalus that is created by a "water-hammer effect" to the third ventricle and/or by compression to the fourth ventricle and the aqueduct (3). In another report, a MDBA is described as rupturing in a manner similar to cerebral aneurysms or arteriovenous malformations in other portions of the central nervous system (4).

It is our opinion that our case was a rare one that showed hemiparesis originating from brain stem infarction without cranial nerve symptoms and hydrocephalus. Although there are some reports on complications of MBDA, the literature contains no discussions with regard to the responsible lesions of hemiparesis except for the cortical lesions (1, 5, 6).

There are two theories regarding the etiologic mechanisms of MBDA: the first is a congenital

theory, and the second is the arteriosclerotic theory emphasized by Boeri and Passerini (5). When we considered some pediatric cases of MDBA, we could not completely deny the congenital factors (7). It is speculated that arteriosclerosis accelerates the original tortuosity of the basilar artery and its paramedian branches are strongly stretched and kinked.

We speculate that one of the mechanisms of the brain stem infarction complicated with MDBA is a circulatory insufficiency based on severe arteriosclerosis of the basilar artery and stretching of the paramedian branches. Though we could not perform vertebral angiography because of the patient's respiratory distress, we obtained sufficient CT findings to explain our speculation with regard to the mechanisms of brain stem infarction complicated by MDBA.

The remarkable findings on the CT scan were di-

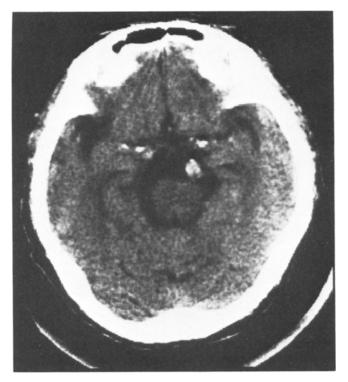


FIGURE 2. Plain CT on admission dislosed no low-density area in the pontine base.

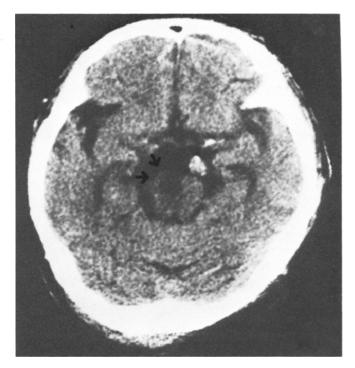
latated and tortuous arteries with extensive calcification of the walls, as confirmed by contrast medium injection (8, 9). Cranial CT is useful for clarifying the causes of the neurologic signs and symptoms complicated by MDBA, and for evaluating the correlation between the position of the basilar artery and the lesion of the brain stem infarction.

### CONCLUSION

We experienced a rare case of MDBA that showed hemiparesis originating from brain stem infarction. Cranial CT was useful for evaluating the causes of the neurologic symptoms in this case of MDBA. The brain stem infarction seemed related to the stretching and kinking of the basilar paramedian branches.

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**FIGURE 3.** Plain CT 10 days after admission presented the focal low-density area in the left half of the pontine base. Note that basilar artery deviated to the right side at the level of the lesion.

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