Short Communication

Idiopathic Basilar Artery Occlusion in Childhood
(Case Report)

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SUMMARY A case of basilar artery occlusion in a 16-year-old girl has been reported. Reports of 20 such cases occurring in children have been reviewed. In children, basilar artery occlusion is more common in males; no previous case above the age of 3 years has been reported in females. There were preceding TIA's and RIND in the vertebral-basilar territory.

STROKES DUE TO OCCLUSION of the basilar artery are seen frequently in adults but are uncommon in children.1-2 Among children, it is more common in males.2 The prognosis of basilar artery occlusion has been reported to be better in children as compared to adults.2 We report one such case in a 16-year-old girl.

Case Report

A 16-year-old girl was admitted to the Neurology services of All India Institute of Medical Sciences on November 7, 1982 for evaluation of dysarthria and ataxia. She had been entirely well until August 29, 1982 when she complained of a sudden sensation resembling an electric current in the right lower limb, lasting for half an hour. Next day she complained of a similar sensation in the right half of the body, followed by right hemiparesis. The weakness improved considerably in the next 5 days. On September 22, 1982 she had severe vertigo and repeated vomiting followed by weakness of the left half of the face with increased weakness of the right upper and lower limbs. Two days later she became progressively dysarthric. Speech improved after 6-7 days. During this period, she noticed diminished taste sensation over the right half of the tongue lasting for three days.

On October 12, 1982 she had sudden loss of consciousness for two and a half hours. After this, her gait was ataxic, speech was dysarthric and she started having choreiform movements of all four limbs. Ataxia and involuntary movements improved in the next five days. On October 24, 1982 she had no involuntary movements. About two weeks after this she was admitted to this hospital because of persistence of the dysarthria and ataxic gait.

After admission, while waiting for angiography, she suddenly became stuporous with signs of peripheral circulatory failure. Her blood pressure had fallen down to 86/50 mm of Hg and pulse rate was 116 per minute. She was given 5% i.v. dextrose saline (2 litres) and injection dexamethasone, 4 mg i.v. stat and then repeated every six hours for twenty four hours. She responded to this treatment by the same evening. Blood pressure went up to 110/70 and pulse rate came down to 80 per minute. She was fully conscious and orientated. Next day, a few hours after a transfemoral angiogram she developed left hemiplegia. The weakness started improving after 48 hours. Her speech, although dysarthric could be understood and she could walk with support.

Her blood pressure was 112/70 mm of Hg. All peripheral and neck pulses were normal. There was no bruit. Examination of chest, heart and abdomen was normal. She was fully conscious, but grossly dysarthric. Pupils and fundi were normal. There was no diplia or nystagmus. She had a left VII N. paresis and left XII N. paresis. Examination of the motor system revealed hypotonia in all four limbs and left hemiparesis. Tendon jerks were exaggerated, more on the left side, planter reflexes were bilaterally upgoing. She had bilateral cerebellar signs with pendular knee jerks. Proper sensory testing was not possible due to a communication problem. Her gait was ataxic but there was no trunkal ataxia.

The results of screening serum chemistry were unremarkable. White cell count was 6,400/cumm. and ESR was 46 mm in the 1st hour; platelet count was 150,000/cumm. The total and differential serum protein level were normal. Total serum cholesterol was 271 mg/dL, triglycerides 94 mg/dL. The individual lipoprotein fractions were within normal range. Blood was negative for VDRL and antinuclear factor. Lumbar puncture revealed clear, acellular CSF with protein level 34 mg/dL and sugar 50 mg/dL, which was sterile on culture, with a negative VDRL.

Chest X-Ray and ECG were normal. EEG showed an awake and natural sleep record. 2-D Echo-cardiography revealed no evidence of aortic stenosis, mitral valve prolapse nor atrial myxoma. Transfemoral four vessel angiogram revealed a complete block of the basilar artery at its origin with filling of the posterior cerebral arteries through the internal carotids (fig. 1, 

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No other abnormality was seen in the intracranial vessels. CT scan done on 25.11.82 revealed a large low attenuating area in the cerebellar hemispheres (more on the right side) interpreted as due to infarction.

At the time of discharge on December 8, 1982, her speech was still dysarthric. She had bilateral cerebellar signs. She had left hemiparesis and she could walk with support. Three and six months after discharge from hospital she was seen in the out patient department. Her speech, though dysarthric, could be understood.

### Table 1: Reported Cases of Basilar Artery Occlusion in Children (Up to 16 Years)

<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>Age/Sex</th>
<th>Etiology</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Okada et al⁴</td>
<td>1964</td>
<td>4 mo/F</td>
<td>congenital</td>
<td>survived</td>
</tr>
<tr>
<td>Fowler⁵</td>
<td>1962</td>
<td>18 mo/F</td>
<td>sepsis</td>
<td>died</td>
</tr>
<tr>
<td>Kowada et al⁶</td>
<td>1962</td>
<td>7 y/M</td>
<td>embolism</td>
<td>died</td>
</tr>
<tr>
<td>Komatsu⁷</td>
<td>1964</td>
<td>16 y/M</td>
<td>not given</td>
<td>survived</td>
</tr>
<tr>
<td>Schechter and Zingesser⁸</td>
<td>1965</td>
<td>16 y/M</td>
<td>not given</td>
<td>survived</td>
</tr>
<tr>
<td>Dooley and Smith⁹</td>
<td>1968</td>
<td>6 y/M</td>
<td>idiopathic</td>
<td>survived</td>
</tr>
<tr>
<td>Ouvrier and Hopkins¹⁰</td>
<td>1970</td>
<td>9 y/M</td>
<td>arteritis</td>
<td>survived</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4 y/M</td>
<td>idiopathic</td>
<td>survived</td>
</tr>
<tr>
<td></td>
<td></td>
<td>14 y/M</td>
<td>embolism</td>
<td>survived</td>
</tr>
<tr>
<td>DeVivo and Farrell¹¹</td>
<td>1973</td>
<td>10 y/M</td>
<td>congenital</td>
<td>survived</td>
</tr>
<tr>
<td>Shimizu et al¹²</td>
<td>1973</td>
<td>6 y/M</td>
<td>B-streptococcal infection</td>
<td>survived</td>
</tr>
<tr>
<td>Moscow and Newton¹³</td>
<td>1973</td>
<td>10 y/M</td>
<td>not given</td>
<td>survived</td>
</tr>
<tr>
<td>Marsden¹⁴</td>
<td>1974</td>
<td>3 y/F</td>
<td>arteritis</td>
<td>died</td>
</tr>
<tr>
<td>Sakata et al¹⁵</td>
<td>1975</td>
<td>10 y/M</td>
<td>congenital</td>
<td>died</td>
</tr>
<tr>
<td>Ackerman et al¹⁶</td>
<td>1977</td>
<td>10 y/M</td>
<td>idiopathic</td>
<td>survived</td>
</tr>
<tr>
<td>Matsumoto et al¹⁷</td>
<td>1977</td>
<td>9 y/M</td>
<td>idiopathic</td>
<td>survived</td>
</tr>
<tr>
<td>Zimmerman et al¹</td>
<td>1978</td>
<td>7 y/M</td>
<td>traumatic</td>
<td>survived</td>
</tr>
<tr>
<td>Thompson et al¹</td>
<td>1978</td>
<td>15 y/M</td>
<td>traumatic</td>
<td>survived</td>
</tr>
<tr>
<td>Mori K et al²</td>
<td>1979</td>
<td>13 y/M</td>
<td>idiopathic</td>
<td>survived</td>
</tr>
<tr>
<td>Present case</td>
<td>1983</td>
<td>16 y/F</td>
<td>idiopathic</td>
<td>survived</td>
</tr>
</tbody>
</table>
stood. Minimal left hemiparesis persisted and she still needed support for walking. Because of ataxic limbs she had to be helped in her daily activities including feeding and dressing.

Comment

Occlusion of the basilar artery is a well known and extensively documented entity in adults. Radiologically detectable occlusion of the basilar artery occurs one fourth as often as occlusion of the carotid artery. Occlusive disease of the basilar artery in children does occur but is rare. Twenty cases have been reported in childhood (table 1).

Ten patients were under the age of 10 years and the remaining ten were under 16 years. The sex ratio is approximately five males to one female. Only about one third of children with basilar artery occlusion have had preceding transient ischemic attacks (TIA's) or reversible ischemic neurological deficit (RIND) in the territory of basilar artery. In childhood TIA's may be less frequent than in adults, but the clinical symptoms are similar.

The common presenting symptom in children is alteration in sensorium. The classical symptoms i.e. altered sensorium, hemiplegia or tetraplegia, and pupillary abnormalities - have been seen in more than half of the cases. The symptoms are usually bilateral and there may be considerable improvement. The prognosis is better in children as compared to adults. Basilar artery occlusions in children have been attributed to congenital causes, embolism, arteritis, the result of trauma or have been of unknown cause (table 1). In the present patient, no cause was identified.

Autopsy verification of the site of occlusion is available in five cases. The mid-brain was the site of lesion in three cases and the pons in two cases; the thalamus and cerebellum were involved in one case each. Vertebral angiography is necessary to make a definite diagnosis. Four vessel angiography should be done to rule out any developmental vascular anomalies, laminar flow and spasm of the vertebral and basilar arteries. Evidence of appropriate collateral circulation is helpful in the diagnosis.

Of all the previously reported cases, the three females were under three years of age (table 1). Basilar artery occlusion occurring in older children (up to 16 years of age) has not been reported previously in a female.

References

Idiopathic basilary artery occlusion in childhood (case report).
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