Ataxic-Hemiparesis, Localization and Clinical Features


SUMMARY Five additional cases of ataxic-hemiparesis are reported. In 3 cases, computed tomography showed an area of decreased attenuation in the posterior limb of the internal capsule, and in 1 case, 2 areas of attenuation in the corona radiata. A review of previously reported cases suggest that brainstorm ataxic-hemiparesis may be separated from supratentorial forms of ataxic-hemiparesis by the presence of nystagmus, dysarthria, cranial neuropathy, and the absence of sensory abnormality.

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ATAXIC HEMIPARESIS IS an unusual clinical syndrome first described by Fisher and Cole, where there is weakness and ataxia on the same side. Since then there has been 10 cases reported, where it has been possible to localize the site of the lesion pathologically or by means of computed tomography. We wish to add 5 more cases studied by computed tomography and suggest there may be recognizable clinical differences between ataxic hemiparesis caused by a brainstorm and a supratentorial lesion.

Case Report

Case 1
A 67 year old man, known to be hypertensive and on medication for 10 years, suddenly developed a sense of unsteadiness, followed 3 hours later by weakness of the left arm in the afternoon. The next morning he found his left leg to be both numb and weak as well. There was no vertigo, diplopia or headaches. General examination was unremarkable. Blood pressure was 145/80, pulse 80 per minute, regular. There were no bruits heard. He was alert, orientated, with normal speech. There was minimal left facial weakness, with a grade 4/5 left hemiparesis. Stretch reflexes were increased on the left, with a positive Babinski sign. Position and vibration sense was lost on the left and there was subjective decreased touch and pin prick sensation. There was marked difficulty in performing finger-nose, and heel-shin tests, as well as difficulty with rapid alternate movements on the left. The cranial nerves and fundi were normal. No nystagmus was seen. On walking he would fall to the left. His fasting blood glucose was found to be elevated on several occasions (18.2–20.4 mmol/L). A C.T. scan on the third day revealed a low density lesion in the left putamen/posterior limb internal capsule region (fig. 1). When seen 1 year after discharge, he continued to have an ataxic upper limb, with absent position sense.

Case 2
A 71 year old man, known to be hypertensive and on irregular treatment for some years, suddenly developed right sided clumsiness and weakness. There was no diplopia, vertigo, or headache. On admission, general examination revealed a blood pressure of 160/100, pulse 80, regular. No bruit was heard. He was alert, orientated with normal speech. Cranial nerves and fundi were normal. There was a grade 4 right hemiparesis, and on walking he would fall toward the right. Reflexes were generally brisk, but more so in the right knee and ankle. Babinski response was present on the right. There was marked ataxia in performing the heel-shin and finger-nose tests on the right, and rapid alternate movements were also defective. Vibration was absent on the right, but other sensation appeared to be normal. C.T. scan on the third day revealed a low density lesion in the left putamen/posterior limb internal capsule region (fig. 2). His weakness subsided by the fourth day, but there was still ataxia at his discharge a week later.

Case 3
A 64 year old man, previously well, suddenly developed weakness of right leg, with unsteadiness of gait, whilst walking in the street. Some hours later he noted numbness and weakness of the right upper limb as well. There was no diplopia, vertigo or headaches. General examination revealed blood pressure of 170/100, pulse 72, regular. No bruit was heard. He was alert, orientated, with normal speech. Cranial nerves and fundi were normal. He had a hemiplegia gait, with dragging of his left leg. There was a grade 4 right hemiparesis which included the face. Reflexes were increased on the right ankle only, with absent Babinski response. There was decreased pain and touch sensation subjectively on the right with absent right lower limb position sense. Finger-nose testing was inaccurate, and rapid alternate movement defective in the right upper limb, and heel-shin response defective in the lower limb. Three days later, the ataxia remained, though the upper limb power had returned to normal. C.T. scan performed on the sixth day revealed two vague areas of low attenuation in the left corona radiata, with no enhancement after contrast (fig. 3).

Case 4
A previously healthy 72 year old man suddenly developed right sided weakness and numbness. On admission he had a grade 2 right hemiparesis, with hyper-reflexia and decreased sensation to touch and pin pricked on the right. Blood pressure was 190/110, pulse 70 regular. No bruit was heard. Over the next few days his power rapidly improved to grade 4, and his sensation returned to normal. However he was noticed then to be ataxic on doing the finger-nose and heel-shin tests, as well as have dysdiadochokinæsia, on the right. C.T. scan showed a low density lesion protruding into the posterior limb of the internal capsule on the left (fig. 4).
Case 5
A previously healthy 57 year old woman, developed mild unsteadiness in her gait suddenly. Four days later, she woke up from her sleep, with a hoarse voice, and left sided weakness and clumsiness. On admission, she had a normal blood pressure 140/80, pulse 70 per minute, regular.

There was no definite abnormality found in the cranial nerves. Nystagmus was not present. There was a left facial weakness with grade 4 left hemiparesis, and hyper-reflexia on the left. No sensory loss could be detected. She fell to the left on walking. Ataxia on performing finger-nose and abnormality in rapid alternate movement was present in the left upper limb. Heel-shin test was normal in the legs. C.T. scan on the seventh day revealed no abnormality.

Discussion
Fisher and Cole originally described the syndrome of ataxic-hemiparesis under the name of homolateral ataxia and crural paresis. They described 14 patients, who all had a picture of leg weakness, and homolateral ataxia, without face or arm weakness, and normal sensation in all but one. Two of the patients had nystagmus. In one of the cases an infarct was found in the posterior limb of the internal capsule. But as he also had other infarcts, clinicopathological correlation was limited. Since then, Fisher has reported 3 cases where
infarct was found in the basis pontis opposite the side of the neurological deficit. All 3 patients had face and arm weakness as well, though in one of them, the toes and ankles were very much weaker, and in another shoulder and grip weakness was more noticed. Three other cases of ataxic-hemiparesis have also been described in the brainstem. Schnapper described a case due to pontine haemorrhage documented by C.T. scan, causing nystagmus, face, arm, and leg paresis, and ataxia. Sakai, Murakami and Ito described a case with weakness of right arm, and leg, and slight facial asymmetry, with dysarthria, and left trigeminal weakness. C.T. scan revealed a pontine infarction. Bendheim and Berg described a case of a 10 year old boy with acute lymphocytic leukemia, who developed a right ataxic-hemiparesis which involved the face as well as limb equally. C.T. scan showed a mass lesion presumably due to leukemia infiltration, in the left rostral midbrain and caudal thalamus. Three cases of ataxic hemiparesis have previously been documented by C.T. scans to have infarct in the posterior limb of the internal capsule. The face have been spared in all 3 cases, and Ichikawa, Tsutsumishita and Fujioka reported that the foot was selectively weak, in their case. In our 3 cases of supratentorial infarcts causing ataxic-hemiparesis however, the face, have all been involved as well.

In discussing the possible site of lesions causing the syndrome, Fisher had suggested internal capsule, corona radiata, midbrain as well as basis pontis as possible sites. Lesions have now been demonstrated in all these sites in ataxic-hemiparesis. Of 8 cases of infarcts demonstrated on computed tomography, only 1 case was seen in the brainstem. This however is likely to be due to difficulties with demonstrating a brainstem infarct with computed tomography. A review of the clinical features (table 1) suggest that the brainstem lesions causing ataxic-hemiparesis may cause a different clinical picture from that seen in supratentional lesions. Nystagmus was noted in 4/6 of brainstem cases, dysarthria in 2 and trigeminal weakness was noted in 1 case. Sensory testing were all reported as being normal. On the other hand, nystagmus, dysarthria, and cranial neuropathy were absent in supratentional cases, and 6 out of 7 cases had abnormality on sensory testing. These features may serve to separate the two groups of ataxic-hemiparesis. If this is true, then our case 5, who had hoarseness of voice at onset, and a normal sensory finding, may have had a negative C.T. scan because the pontine lesion was not easily seen.

Although the original clinical cases of Fisher and Cole stressed the selective weakness, subsequent cases of ataxic-hemiparesis have shown more widespread and symmetrical involvement. Fisher has documented variable distribution and severity in hemiparesis due to a pontine lesion. However Ichikawa, Tsutsumishita and Fujioka's case of capsular infarct also showed selective foot weakness. Thus, selective weakness, especially of the lower limb may not necessarily indicate a brainstem lesion.

As in other cases of lacunar syndromes, ataxic-hemiparesis is not necessarily just due to lacuna. Haemorrhage, as well as tumour may be responsive. Apart from the single cases of leukaemic infiltrate, 22 of the 26 cases reported had hypertension. Transient ischaemic attacks was also documented in 9 patients and progression in deficits described in eight. Thus, large vessel disease may be present in some of the cases of ataxic-hemiparesis. However, angiogram studies have only been reported by Perman and Racy, who showed bilateral carotid and vertebral stenosis in their case. More long term studies of the natural history are necessary to document whether ataxic-hemiparesis patients behave as typical lacunar syndromes, or

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<th>Table 1</th>
<th>Pathology and Clinical Features of Ataxic-hemiparesis in Literature</th>
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<tr>
<td>Localisation</td>
<td>Pathology</td>
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<td>Fisher 1978</td>
<td>pons</td>
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<td>Sakai et al 1981</td>
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<td>Schnapper 1982</td>
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<td>Bendheim &amp; Berg 1981</td>
<td>midbrain</td>
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<td>Perman &amp; Racy 1980</td>
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<td>Ichikawa et al 1982</td>
<td>internal capsule</td>
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<td>Iragui &amp; McCutchen</td>
<td>internal capsule</td>
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<td>Present report (1)</td>
<td>internal capsule</td>
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as large vessel disease with small vessel embolisation.

The anatomical basis of the lesions causing ataxic-hemiparesis have been previously discussed. It is likely that interruption of pontocerebellar fibres from pontine nuclei plus corticospinal tracts in the basis pontis, is responsible for pontine ataxic-hemiparesis.\(^2\) Interruption of cerebral peduncle and the superior cerebellar peduncle after it has crossed the midbrain, in the ventral spec of the midbrain may be responsive for a midbrain ataxic-hemiparesis.\(^3\)\(^5\) Capsular ataxic-hemiparesis will cause the syndrome by involving the corticospinal tract plus the frontopontine fibres in the posterior limb of the internal capsule. Although capsule ataxia hemiparesis seems frequently to be associated with sensory loss, sensory loss is not essential for ataxia to be present, as in Iragui and McCutchen's case, and our case 4. Involvement of cerebellar projection to the cortex appears also possible.\(^1\)\(^8\) and underlie perhaps the ataxic hemiparesis when the lesion is above the internal capsule.

### References


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**The Isolated Occlusion of the Angular Gyri Artery**

A Correlative Neurological and Anatomical Study — Case Report

Slobodan V. Marinkovic, M.D.,* Miroslav S. Kovacevic, M.D.,† and Vladimir S. Kostic, M.D.

**SUMMARY** We examined a patient who had signs of a cerebral hemisphere lesion: right hemiparesis, facial weakness, right hemianopsia, acustico-mnestic dysphasia, “empty speech,” acalculia, visuo-spatial agnosia and constructional apraxia, but without changes in consciousness. Taking into account clinical signs, computed tomography and carotid angiography findings, we concluded that our patient had an infarction zone in the left temporo-parieto-occipital region, as a consequence of the isolated angular gyri artery (ANG) occlusion. Some clinical signs were a direct effect of the ANG's occlusion. Namely, this artery supplies the cortical regions of great functional significance: the planum polare and temporale, the transverse temporal gyri, the superior and middle temporal gyri, the angular and supramarginal gyri, as well as the superior, middle and inferior occipital gyri. But the other symptoms and signs could be explained by the pathophysiological effect of the cerebral edema on regions supplied by the non-occluded branches of the middle cerebral artery.

**IN THE COURSE of our clinical work we registered a case of an angular gyri artery occlusion. Numerous authors have studied complete or partial occlusion of the middle cerebral artery.**\(^1\)\(^6\) However, the isolated occlusion of its single pial branches has rarely been reported.\(^7\)\(^8\) That is the reason that made us examine this case in detail. We carried out two groups of examinations: clinical and anatomical. We studied and compared the collected results in order to find the corresponding correlation among the clinical, neuroanatomical and functional facts.

**Material and Methods**

We used the available neurophysiological, neuroradiological, neuroophthalmological and laboratory methods of examinations. Thirty-four forebrain hemispheres were used for anatomical study. The hemispheres were fixed for at least two weeks in 10% formaldehyde solution. The main stem and all pial (cortical) branches of the middle cerebral artery of each hemisphere were microdissected, with complete angular gyri artery examination.

**Case Report**

The patient M. B., aged 29, a farmer, was admitted into hospital with acute right hemiparesis, facial weak-
Ataxic-hemiparesis, localization and clinical features.
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