Ataxic Hemiparesis

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Abstract

Ataxic hemiparesis (AH) is characterized by the simultaneous presence of a pyramidal tract syndrome with homolateral ataxic syndrome. In the first description of AH by Fisher and Cole (1965), they elaborated the core symptoms as weakness and pyramidal signs on one side combined with ipsilateral cerebellar-like ataxia. However, subsequent reports have expanded the clinical spectrum of AH to include cases with persistent hemisensory deficits and additional symptoms like dysarthria or facial paresis. AH was initially described as a lacunar syndrome correlating with lacunar infarctions. However, recent reports have suggested that AH is not significantly correlated with these infarctions, and in fact a substantial number of studies have demonstrated that lacunar infarction as well as cardioembolic and large-artery atherosclerosis can cause AH. In addition, hemorrhagic strokes, tumors, head trauma, infection, and demyelinating diseases have all been associated with AH. The pons, corona radiata, thalamus, and internal capsule are the most commonly reported lesion sites in AH; however, cortical lesions have also been observed in some AH patients. Regardless of the precipitating insult, specific AH symptoms are thought to result from damage to corticospinal and cerebellar pathways (efferent or afferent), while variations in symptomology reflect the size and precise location of the infarct lesion. Optimal treatment strategies naturally depend on treating the underlying etiology, and to date, have included anticoagulant and antiplatelet drug therapies. The prognosis for AH following ischemic stroke is generally good. This chapter provides an update on the clinical features, lesion topography, treatment, and prognosis of AH.

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Introduction

Ataxic hemiparesis (AH) is characterized by the simultaneous presence of a pyramidal tract syndrome with homolateral ataxic syndrome (Fisher 1978). Fisher and Cole (1965) first described this syndrome showing *homolateral ataxia and crural paresis* as vascular syndrome, and later renamed it as AH (Fisher 1978). Pathological studies indicated that the precipitating insults were most probably infarcts (lacunes) resulting from occlusion of penetrating arteries (Fisher 1978). Since then, many case reports have described the various infarct sites, core and secondary symptoms, and putative etiologies of AH. AH due to hemorrhagic stroke and nonlacunar infarctions has also been reported. Moreover, AH cases associated with brain tumors, head trauma, central nervous system infection, or demyelinating diseases suggest that AH is not always a clinical manifestation of infarcts. Indeed, the single common denominator is damage to diffuse corticospinal (pyramidal tract) and cerebellar pathways, and these scattered targets serve to explain the variance in both etiology and infarct topography. This chapter discusses seminal articles on AH and provides an update on the clinical features, frequency, lesion topography, pathological mechanisms, and prognosis of AH occurring due to ischemic stroke (IS) as well as due to nonischemic causes.

A Brief History of AH

Fisher and Cole (1965) examined 14 stroke patients at Massachusetts General Hospital; all patients presented with an unusual neurological deficit in which the arm and leg on the same side exhibited a combination of severe cerebellar-like ataxia, mild limb weakness, and pyramidal signs. In typical cases, mild weakness of the lower limb, particularly of the ankle and toes, and Babinski's sign were associated with a striking dysmetria (inaccurate reaching to target with undershoot and overshoot) of the arm and leg on the same side. The arm showed little or no weakness relative to the leg. The facial muscles were spared, although two patients showed nystagmus, and there were no speech impairments (dysarthria). Sensation was intact in all but one case, and neurological function was otherwise normal in this patient group. Recovery was almost complete in 12 of 14 cases, suggesting that this syndrome was relatively benign. In one case, postmortem examination 3 years after symptom onset revealed at least 11 lesions in the brain; however, a lesion in the internal capsule was most consistent with the functional deficits observed. Although none of the other cases were studied pathologically, the etiology was believed to be ischemic rather than hemorrhagic, and the syndrome was named *homolateral ataxia and crural paresis*. Thereafter, Fisher (1978) reported three additional cases presenting with weakness and pyramidal signs on one side combined with ipsilateral cerebellar-like ataxia. These cases were examined pathologically and revealed lesions in the upper basis pontis, contralateral to both the pyramidal and the cerebellar signs. The basilar artery was patent, so these infarcts were probably the result of occlusion of the

penetrating arteries. This syndrome was renamed AH and, for years thereafter, AH was classified as a lacunar syndrome (Fisher 1982).

Core Symptoms of AH

Weakness and pyramidal signs on one side combined with ipsilateral cerebellar-like ataxia are the core symptoms of AH (Fisher and Cole 1965; Fisher 1978). Limb weakness is usually mild so that there is sufficient power to perform neurological tests of motor function; indeed, if the weakness was severe, cerebellar-type ataxia would have been difficult to detect using cerebellar function tests. One report explicitly defined hemiparesis as mild enough (Grade 4) to perform reliable tests of cerebellar function (Kim et al. 1995). However, cases of ataxia with severe limb weakness at first neurological examination with subsequent recovery of weakness also have been reported as AH (Huang and Lui 1984; Colombo et al. 1986). In early reports, hemiparesis was predominant in the lower limb (Fisher and Cole 1965); however, AH cases presenting as arm predominant or proportional hemiparesis were also described in many subsequent studies. One study comprising 27 patients with AH following intracerebral hemorrhage (ICH) or IS found that 20 had proportional hemiparesis and only seven had nonproportional (arm or leg dominant) hemiparesis (Colombo et al. 1986). A similar study demonstrated that 45% of subjects had proportional hemiparesis, another 45% had upper limb–predominant hemiparesis, and only 10% had lower limb–predominant hemiparesis (Sanguineti et al. 1986). A diffusion-weighted imaging (DWI) study on 29 patients with AH due to IS demonstrated that 8 patients had leg predominant, four had arm predominant, and the remaining 17 patients presented proportional hemiparesis. Furthermore, no clear correlation was found between lesion topography and the predominating pattern (Hiraga et al. 2007). Brisk or active deep tendon reflexes are frequently observed in the affected limbs, and Babinski's sign is often positive. A decreased deep tendon reflex in the affected limb is rare, but the same has also been reported (Helweg-Larsen et al. 1988).

Finger-to-nose and heel–knee–shin testing revealed cerebellar-type ataxia of the affected limbs. Repetitive movements of the affected arm are made with difficulty (adiadochokinesis). The intensity of ataxia is not influenced by visual input, indicating that the ataxic dimension of AH is not due to deficient proprioceptive feedback as in sensory ataxia. The Holmes-Stewart rebound phenomenon is also observed after passive displacement of the affected arm, consistent with cerebellar dysfunction. Patients tend to fall to the affected side when walking or standing, and some cannot walk without assistance. Hypotonia of affected limbs is rare (Besson and Hommel 1993) but has been reported. Similarly, cases with spasticity of affected limbs were rare in many studies, but one study reported spasticity in 9 of 20 AH patients (Magrotti et al. 1990). Intention (kinetic) tremor has been observed in a few cases (Besson and Hommel 1993), and this was sometimes severe (Jabbari et al. 1983).

Original reports demonstrated ataxia and ipsilateral hemiparesis but, in the absence of sensory loss, dysarthria, facial paresis, or motor signs typical of

brainstem dysfunction (with the exception of asymmetrical nystagmus) were seen. In subsequent reports, however, the clinical spectrum of AH was expanded to include cases with hemisensory deficits. The sensory loss in these cases was usually slight and involved modest decreases in touch and pinprick sensations; however, loss of vibratory and joint position sensation was also been reported (Huang and Lui 1984; Besson and Hommel 1993). Earlier reports also suggested that cases of AH with associated sensory abnormalities were more likely to be caused due to supratentorial dysfunction than brainstem lesions (Huang and Lui 1984; Mori et al. 1984). For example, several studies indicated that AH with accompanying hemihypesthesia more often indicated thalamocapsular lesions rather than pontine lesions (Huang and Lui 1984; Gorman et al. 1998). AH with an obvious ipsilateral hypesthesia has been termed *hypesthetic AH* (Helgason and Wilbur 1990; Melo et al. 1992); another report termed such a disturbance *sensorimotor AH* (Ghika et al. 1991). Objective sensory disturbances frequently accompany infarcts involving the thalamus, and such disturbances were reported in 75% (Sanguineti et al. 1986) and 78% (Crespi et al. 1988) of AH cases. However, there is substantial variability in the nature and severity of the sensory disturbances that accompany AH, as other clinical studies reported that only 10% (Magrotti et al. 1990), 20% (Gorman et al. 1998), and 24% (Hiraga et al. 2007) of patients presented with sensory disturbances. At the extreme, a study of 27 patients with AH due to IS or ICH reported no associated sensory disturbances (Colombo et al. 1986). Among sensory disturbances presenting with AH, ipsilateral hemisensory disturbances are the most frequent complaint. Sensory impairment restricted to a cheiro-oral topography has also been reported, stemming from capsular infarction with possible damage to the adjacent lateral thalamus (Combarros et al. 1992). Kim et al. (1994) reported three IS cases and one ICH case with posterior and lateral thalamic infarcts that exhibited pseudochoreoathetosis (abnormal movements associated with loss of proprioception), hypesthesia, and core AH symptoms. However, these patients demonstrated both cerebellar-type and sensory ataxia, so a pure AH diagnosis in these cases remains debatable (Kim et al. 1999).

Symptoms that occasionally manifest with the core symptoms of AH include dysarthria, facial paresis, nystagmus, Horner's syndrome (Rossetti et al. 2003), contralateral trigeminal weakness (Sakai et al. 1981), one and half syndrome (Bansal et al. 1989), and mirror movement (Radhakrishnan et al. 1981). Dysarthria has been observed in 22–63% of AH cases (Colombo et al. 1986; Sanguineti et al. 1986; Magrotti et al. 1990; Moulin et al. 1995; Gorman et al. 1998; Hiraga et al. 2007) and facial paresis manifested in 7–71% of AH cases (Colombo et al. 1986; Sanguineti et al. 1986; Magrotti et al. 1990; Moulin et al. 1995; Kumar et al. 1996; Gorman et al. 1998). These widely disparate cases underscore the ambiguity of an AH diagnosis in patients presenting with noncore symptoms.

The presence of additional neurological signs and symptoms, however, could help localize the lesions underlying AH, but attempts to correlate clinical features with the different infarct loci have been only partially successful. Older reports suggested that nystagmus, dysarthria (Huang and Lui 1984), trigeminal dysfunction (Sakai et al. 1981), and vertigo or diplopia (double vision) were associated with

pontine lesions. AH due to brainstem lesions was also suggested by Gorman et al. (1998), who reported that dysarthria and ipsilateral facial weakness were more often observed in AH cases following pontine lesions, and that ipsilateral limb and trigeminal sensory loss accompanying the core AH symptoms were more frequently observed due to thalamocapsular damage. However, these associated symptoms are not consistent and do not provide a reliable guide to predict the location of the precipitating infarct.

Cortical symptoms like aphasia and visual field deficits such as hemianopia (partial blindness in the ipsilateral visual field) were not described in the original report but have since been reported in AH patients (Taly et al. 1988). In fact, the inclusion of cortical symptoms has been inconsistent. A large patient study (Moulin et al. 1995) did not include cortical disturbance in the AH diagnosis criteria, whereas Gorman's criteria allowed minimal cortical signs (Gorman et al. 1998). Lacunar infarction generally does not cause cortical deficits such as aphasia or homonymous hemianopia (Fisher 1982), and AH has been historically classified as a lacunar syndrome that is rarely associated with aphasia or hemianopia. Therefore, cortical symptoms should not be included in the core diagnostic criteria for AH.

Variations of AH

Painful AH

Bogousslavsky et al. (1984) reported a case of right-side AH following lateral thalamic infarction without objective sensory deficits but with ipsilateral pain that was diagnosed as *painful AH*.

Sensory AH

Dobato et al. (1990) reported five patients with unilateral corticospinal tract signs, ataxia of the contralateral limbs, and severe impairment of proprioceptive sensation following contralateral dorsolateral thalamic hemorrhage. This syndrome was named *sensory AH*. However, in these cases, ataxia improved markedly when motor tests were performed under visual guidance, which contrasts with the observations seen in cerebellar-type ataxia, but is consistent with classic sensory ataxia. For this reason, sensory AH should be distinguished from classical AH.

Other Variations

Other variations of AH have been also described. *Ataxic monoparesis* is ataxia and weakness restricted to one arm, and one report demonstrated that this syndrome was due to infarction of the sensorimotor cortex (Ugawa 2009). When AH symptoms present bilaterally, it is designated as *ataxic tetraparesis*. Ataxic tetraparesis resulting from bilateral infarcts in the basis pontis (van Gijn and Vermeulen 1983) and in the internal capsule (Utku et al. 1991) have been reported. Another report named this syndrome *ataxic quadriparesis* and, in this study, the symptoms were explained by the observation of symmetrical lacunar infarcts involving the junction of the internal capsule with the corona radiata (Ambrosetto 1992).

Quadriataxic hemiparesis has also been reported following pontine infarctions in two patients showing core AH symptoms and additional limb ataxia on the side ipsilateral to the lesions (Kim et al. 1995). *AH with bilateral leg ataxia* has been reported as AH with ataxia of the contralateral leg due to pontine infarction (Withiam-Leitch and Pullicino 1995).

The Severity of AH

As mentioned above, AH symptoms do not include severe motor weakness because weakness precludes tests for cerebellar ataxia, a highlight of AH. This suggests that AH may be a milder affliction than pure motor hemiparesis. Indeed, one study showed that the mean National Institutes of Health Stroke Scale scores were 3.89 for AH patients, 6.16 for motor stroke patients, 5.34 for sensorimotor stroke patients, and 4.92 for patients with dysarthria-clumsy hand syndrome (De Reuck et al. 2008). A DWI study demonstrated that the mean National Institutes of Health Stroke Scale scores were 5.2 for AH cases associated with lesions of the corona radiata/internal capsule, 6.0 for AH due to lesions of the pons, and 4.7 due to lesions of the frontal cortex or subcortical regions. Therefore, AH precipitated by pontine lesions is associated with slightly more severe motor weakness than AH with other etiologies (Hiraga et al. 2007).

Nonischemic AH

AH usually follows IS; however, AH has also been diagnosed following nonischemic event. Fisher (1978) believed that AH was not limited to vascular lesions and that it could be associated with tumors and demyelinating lesions as well. In fact, AH is not a disease by itself, but is a manifestation of disruption to both the corticospinal tract and cerebellar pathways (regardless of cause).

Hemorrhagic Stroke Causing AH

Although much less common than AH due to IS, hemorrhagic stroke is the second most frequent cause of AH. Previous reports have demonstrated that both intracerebral and subdural hemorrhage can cause AH (Lazzarino et al. 1993). Mori et al. (1985) reported on five patients with ICH; four with hemorrhage near the internal capsule lesions and one with pontine hemorrhage. The other study reported that 5 of 20 AH patients had ICH; two with hemorrhage in the thalamus, and one patient each with hemorrhage in the pons, corona radiata, and internal capsule (Taly et al. 1988). Another large-scale study of 100 AH patients found five with ICH, two with hemorrhage in the lentiform nucleus and one each with hemorrhage in the corona radiata, pons, and cortex (Moulin et al. 1995). Colombo et al. (1986) reported that 3 of 26 AH cases had ICH (all in the internal capsule), while Kim et al. (1994)

reported that 2 of 28 small ICH patients (thalamocapsular area or in the basal ganglia) presented with AH. Further, other case reports demonstrated hemorrhagic stroke as the culprit for AH in the pontine, internal capsule, and thalamus.

Other Causes of AH

Several case reports have indicated that brain tumors are the third most common cause of AH (Sanguineti et al. 1986; Helweg-Larsen et al. 1988). Head trauma has also been reported as a cause of AH (Jain et al. 1982). Three patients with epilepsy also presented the core symptoms of AH in the postictal state, and all recovered within 7 days. The epileptic foci in these patients were in the frontoparietal or temporoparietal regions (Bansal and Chopra 1991). Infectious diseases, including neurocysticercosis (Barinagarrementeria and Del Brutto 1989), Creutzfeldt–Jakob disease (Kastenbauer et al. 2001), cryptococcus meningitis (Sanchetee 1998), Parvovirus B19 infection (Mandrioli et al. 2004), and brain tuberculoma (Taly et al. 1988) have also been reported to cause AH. In cases of AH concomitant with infection, it is believed that both the infection itself and secondary arteriolar occlusion due to inflammation can cause lesions leading to AH (Barinagarrementeria and Del Brutto 1989; Mandrioli et al. 2004). Finally, AH has also be reported in cases of demyelinating diseases (Taly et al. 1988) like multiple sclerosis (Lazzarino et al. 1993; Gorman et al. 1998); However, it was difficult to distinguish AH from vascular syndrome at first due to the acute onset of symptoms.

Frequency of AH

In the large-scale sample of stroke victims, only 2% were diagnosed with AH (Bamford et al. 1987). Other large-scale sample of IS patients found that only 1.25–2% developed AH (Gerraty et al. 2002; Arboix 2004), while other study reported 23 AH cases in 2,500 acute stroke patients (Arboix 2004). All 23 of these AH patients were IS cases (23 of 1,840 or 1.25%), and AH was present in 4.1% of patients with lacunar infarcts (Arboix 2004). The wide variation of AH cases most likely reflects the variable infarct topography of the stroke patients included. Other possible sources of variation are the imaging modality (magnetic resonance imaging [MRI] vs computed tomography [CT]), genetic predisposition, general health before stroke, and lifestyle. In any case, it appears that AH is a relatively rare occurrence following stroke because the presence of core symptoms (and absence of other symptoms) requires partial but relatively specific disruption of corticospinal (pyramidal) and cerebellar pathways.

Even within a sample of confirmed AH cases, however, lesion type and topography varied dramatically from study to study. Thus, AH with lacunar syndrome was reported in 3–21% of cases (Bamford et al. 1987; Arboix et al. 1990, Chamorro et al. 1991, Gan et al. 1997; Gerraty et al. 2002; De Reuck et al. 2008), and 3–26% of confirmed AH cases were linked to lacunar

infarction (Hommel et al. 1990; Clavier et al. 1994; Schonewille et al. 1999; Arboix et al. 2000, 2010; Arboix 2004).

AH was found in only 5 of 174 (3%) cases with spontaneous ICH (Mori et al. 1984), while a study of patients with lacunar syndrome following ICH found that 5 of 19 patients (26%) had AH, suggesting a relatively high incidence of AH in hemorrhage-induced lacunar syndrome (Mori et al. 1985). Indeed, AH occurred more frequently following ICH than following IS (Mori et al. 1985; Moulin et al. 1995). Similarly, another report found a relatively high (25%) frequency of ICH in confirmed AH cases (Taly et al. 1988). Conversely, a large-scale study of 439 patients with lacunar syndrome due to IS or ICH found that only 17 patients had AH, and that all were IS patients (Arboix et al. 2000).

Mechanisms and Lesion Topography of AH Following IS

A lesion resulting in AH must disrupt both the corticospinal tract and cerebellar afferent or efferent pathway in locations where these tracts are in close proximity (Withiam-Leitch and Pullicino 1995). However, by definition, the corticospinal tract cannot be severely disrupted because severe weakness would mask cerebellar ataxia. AH is rarely caused by lesions within the cerebellum, so cerebellar-type ataxia must be caused by impairment within a cerebellar efferent or afferent pathway. A kinematic and electromyography analysis of AH due to corona radiata infarction showed a similar pattern of cerebellar lesions (Wild and Dichgans 1993). The specific mechanism underlying a particular AH case depends on the location of the lesion, whether in the pons, thalamus/internal capsule, or cerebral cortex. Table 74.1 summarizes the infarct locations in AH patients accrued from a number of studies.

Cerebral Cortex

Large anterior cerebral artery (ACA) infarcts containing central paralobules have been recognized as the main cause of AH with leg predominant weakness (Bogousslavsky et al. 1992; Moulin et al. 1995). In AH associated with ACA territory infarction, hemiataxia from paracentral lesions could be due to the disruption of the paracentral contingent of Arnold's frontopontine bundle or of parietopontine bundle (Bogousslavsky et al. 1992; Giroud et al. 1994). However, neither lesions to Arnold's frontopontine nor to parietopontine bundle caused ataxia (Tredici et al. 1990). In CT-imaging studies, lesions to the central sulcus that were distal to the motor cortex were reported to cause AH (Sanguineti et al. 1986; Tredici et al. 1990). Conversely, a DWI study of AH patients with middle cerebral artery territory occlusion showed damage to the precentral gyrus, including the precentral knob or damage to the precentral knob and postcentral gyrus (Hiraga et al. 2007). The precentral knob has been reported to be the human hand motor area, and precentral knob damage produces isolated hand or finger palsy. DWI study has

Table 74.1 Infarct locations associated with ataxic hemiparesis

References	Year	Modality	Lesion locations
Magrotti	1990	CT	Junction lacunar infarction, 85%; posterior limb of internal capsule, 10%; pontomesencephalic lacunar infarction, 5% (n = 20)
Chamorro	1991	CT/MRI	Corona radiata, 31%; posterior limb of internal capsule, 23%; anterior limb of internal capsule, 15%; genu, 15%; basal ganglia, 8%; others 8% (n = 33)
Moulin	1995	CT/MRI	Internal capsule, 39%; corona radiata, 25%; pons, 19%; thalamus, 13%; lentiform nucleus, 8%; cerebellum, 4%; frontal cortex, 4%; leukoaraiosis or no visible region, 23% (n = 100, including 5 intracranial hemorrhage cases)
Kumar	1996	CT	Thalamus, 23%; internal capsule, 23%; basal ganglion, 18%; frontoparietal, 18%; frontotemporal, 5%; frontal region 5%, pons 5%, multiple regions 5% (n = 22)
Gan	1997	CT/MRI	Internal capsule/corona radiata, 40%; pons, 31%; basal ganglia, 14%; thalamus, 11%; and other brainstem, 3% (n = 39)
Gorman	1998	CT/MRI	Pons, 43%; thalamocapsular, 33%; internal capsule, 13%; striatocapsular, 10% (n = 30, a single lesion was identified as the probable cause of the syndrome in 30 of 45 cases)
Schonewillee	1999	MRI	Internal capsule, 55% (with thalamus, 9%; with thalamus/cerebral peduncle, 9%; with corona radiata, 9%); pons, 18%; Putamen, 9%; corona radiata, 9%; insular cortex, 9% (n = 11)
Hiraga	2007	MRI	Pons, 28%; distended internal capsule from corona radiata, 24%; internal capsule, 21%; corona radiata, 7%; precentral with or without post central gyrus, 7%; frontal subcortical area, 3%; multiple lesions, 7%; no visible lesion, 3% (Two lesions were found in two patients, in the pons and corpus callosum, and in the corona radiata and subcortical white matter), (n = 29)

Hiraga et al. (2007) and Schonewille et al. (1999) used diffusion-weighted imaging in all patients

also provided direct clinical evidence that the precentral gyrus, especially nearby the precentral knob, contains en passant fibers of the fronto-ponto-cerebellar tract that run close to the pyramidal tract, and that damage to this region can cause AH (Hiraga et al. 2007). Other DWI studies have shown that the insular cortex is the causative lesion in case of cortical AH (Schonewille et al. 1999).

Subcortical Lesions

The corona radiata is a common lesion site associated with AH, and the centrum semiovale also has been reported as an AH-associated lesion site (Bogousslavsky and Regli 1992). In 36 patients with centrum semiovale infarction (10 large and 26 small infarcts), only 2 of 26 patients with small infarcts presented with AH (Bogousslavsky and Regli 1992). A CT study suggested that infarcts in both anterior and posterior aspects of the corona radiata can cause AH (Sage and Lepore 1983). Some reports suggested that in AH due to corona radiata infarction, ataxia could be explained by interruption of corticopontine or thalamocortical fibers (Sage and Lepore 1983).

Thalamus and Internal Capsule

Thalamic lesions are the cause of hypesthetic AH. In 17 patients with hemiataxia due to thalamic infarction, 8 had hypesthetic AH and two more displayed core AH symptoms (Melo et al. 1992). In patients with capsular infarction, damage to both the anterior and posterior limbs was reported as the cause of AH (Chamorro et al. 1991). In AH cases with capsular infarctions, patients showed smaller infarct sizes than the sizes in pure motor hemiparesis or sensorimotor stroke (Sohn et al. 1990; Tei et al. 1993). On the other hand, in 23 cases of capsular hypesthetic AH, 22 had lesions in the posterior limb of the internal capsule and hypesthetic AH correlated with larger infarcts that were most often localized to the posterior medial superior territory of the anterior choroidal artery (Helgason and Wilbur 1990). The frequency of AH in capsular infarction also varies; 14 of 72 patients with capsular infarction showed AH in one study (Tei et al. 1993), while only 3 of 124 capsular infarction patients presented AH in another (Arboix et al. 2005). Thus, smaller capsular lesions may lead to core AH, while more extensive damage may cause hypesthetic AH.

Fisher and Cole (1965) first suggested that hemiataxia following a capsular lesion was probably due to the interruption of the corticopontine–cerebellar connections. Lesions to the region containing the posterior limb of the internal capsule and thalamus were likely to damage a cerebellar pathway, either the ascending dentatorubrothalamocortical pathway in the ventral lateral nucleus of the thalamus or the descending corticopontocerebellar pathway in the posterior limb of the internal capsule (Melo et al. 1992; Kim et al. 1999). However, the cause of hemiparesis following lacunar infarcts that are apparently restricted entirely to the thalamus remains unclear. Since the corticospinal tract does not pass through the thalamus, it must be assumed that in such cases there is ischemia or edema of the internal capsule or that the internal capsule is damaged by compression from edema (Besson and Hommel 1993).

Basal Ganglia

One report including nine cases with acute pure putaminal infarction showed that two cases had AH and four had AH with sensory syndrome (Russmann et al. 2003). Other reports have shown that infarction within the putamen (Gerraty et al. 2002) or lentiform nucleus (Moulin et al. 1995) can cause AH. An infarct to the anterior aspect of the lentiform nucleus is more frequently associated with AH than are lesions to the posterior lentiform nucleus (Ghika et al. 1991). However, the underlying mechanisms of AH from lesions in this location have not been determined.

Brainstem and Cerebellum

Lesions within the pons are frequently associated with ataxic hemiparesis (AH). Nabatame et al. (1987) reported three AH patients with lesions located in the

dorsomedial basis pontis. Likewise, other reports found that 11 of 67 patients with isolated pontine infarction had AH (Erro et al. 2005), and the frequency of AH was the same (16%) for both branch atheromatous disease and lacunar pontine infarction. AH was found in 1 of 12 patients with ventromedial pontine infarction, 3 of 9 patients with ventrolateral pontine infarctions, and 2 of 11 patients with tegmental pontine infarctions (Bassetti et al. 1996). Kim et al. (1995) reported four patients with AH and two with quadrataxic hemiparesis among 37 basis pontine infarction patients. This report demonstrated that the loci associated with AH were variable, with one case from a middle pons lesion, one from a rostral pons lesion, and two cases with lesions encompassing the middle and rostral pons. Two of these four cases showed large infarcts, and the other two showed infarcts restricted to the lateral aspect. These results suggested that pontocerebellar fibers are widespread within the basis pontis, and so they may be easily damaged by large infarcts at the different locations. In addition, lateral or ventrally situated midcaudal pontine lesions may damage fewer corticospinal axons, thus producing the more subtle pyramidal signs of AH.

Fisher and Cole (1965) hypothesized that the contralateral weakness results from disruption of corticospinal fibers, whereas contralateral dysmetria is a consequence of damage to pontine neurons or their axons, but it was not explained why cerebellar signs following pontine lesions were unilateral. If the pontine lesion produces contralateral dysmetria because of damage to pontocerebellar fibers crossing over to the opposite cerebellar hemispheres, then the same lesion should disrupt pontocerebellar fibers from the unaffected side that are coursing through the lesion, and there should be dysmetria on the side ipsilateral to the lesion (Schmahmann et al. 2004b). Some older reports suggested hypothesis about this mechanism (Kobatake and Shinohara 1983; Nabatame et al. 1987); however, there are no histological evidences. Study about the trajectory of the pontocerebellar fibers in rhesus monkey using the isotope autoradiographic technique suggested that small pontine strokes spare sufficient decussating pontocerebellar fibers to prevent ipsilateral dysmetria and that ipsilateral dysmetria after large pontine stroke presents a disconnection syndrome. Another isotope study showed motor projection to the basis pontis (Schmahmann et al. 2004c). In the attempt at a topographic map of the human pons using a lesion-deficit correlation analysis, Schmahmann et al. (2004a) reported that 9 of 25 patients with pontine infarction presented with AH and that the lesions were discrete and focused in the middle or the caudal third, or encompassed both regions of the pons. This study determined that pontine syndromes are not absolutely discrete but, rather, are distinguished from each other by the relative degree of involvement of each clinical feature. The clinical manifestations reflect the location of the pontine lesion and the well-organized topography of motor representation in the human basis pontis. Hand coordination is medial and ventral in rostal and midpons, and leg coordination is in the caudal half of the pons. Besides the brainstem lesions, Moulin et al. (1995) reported that infarction to the superior cerebellar artery territory resulted in AH but, in this rare case, the underlying mechanism has not been studied.

Besides imaging methods that localize infarcts, techniques like somatosensory evoked potentials (SEPs), cerebellar electrical stimulation, and studies of crossed

cerebellar diaschisis detected by single-photon emission CT/positron emission tomography have been employed to elucidate the mechanisms of AH.

The Role of Afferent and Efferent Pathways

Abnormal SEPs in AH patients indicate that ataxia in AH may be caused by disruption of afferent pathways (Kelly et al. 1987). Both Mori et al. (1984) and Kelly et al. (1987) reported delayed SEP responses or reduced N20 amplitudes in cases of AH due to stroke localized to the posterior limb of the internal capsule. These findings suggested that the ataxia observed in these AH patients was caused by dysfunction of the ascending cerebellothalamic pathway rather than by damage to the descending corticopontocerebellar fibers traversing the posterior limb of the internal capsule (Helgason and Wilbur 1990). On the other hand, normal SEP potentials were reported in another patient group with pontine AH (Huang and Chan 1984; Delgado et al. 1985). One report demonstrated that SEP changes were more frequent following gross infarcts, involving the cortex, and following small infarcts, involving the thalamus, than changes occurring following lacunar infarcts of the capsular and subcortical white matter (Crespi et al. 1988).

The cerebellar electrical stimulation method was used to differentiate cerebellar ataxia due to cerebellar efferent pathway lesions from other cerebellar ataxias or non-cerebellar ataxias. Suppression of motor cortical excitability was normally elicited in patients with cerebellar afferent pathway damage; in contrast, the suppression effect was not observed in patients with cerebellar efferent pathway damage. Ugawa (2009) employed cerebellar stimulation on one patient with AH due to capsular infarction; suppression of motor cortex excitability was not observed as would be expected in a case of cerebellar efferent pathway damage. In contrast, patients with ataxic monoparesis due to cortical infarction showed normal suppression, indicative of cerebellar afferent pathway damage. Therefore, it appears that both efferent and afferent damage can elicit AH.

Crossed Cerebellar Diaschisis in AH

Crossed cerebellar diaschisis is thought to result from deafferentation of the affected cerebellar hemisphere attributable to contralateral interruption of corticopontocerebellar fibers, and crossed cerebellar diaschisis is most often reported in association with large hemispheric infarcts. Some studies have reported the possibility of crossed cerebellar diaschisis in AH patients, mainly following cortical infarctions (Sakai et al. 1986; Giroud et al. 1994; Flint et al. 2006), suggesting that AH results from functional depression (diaschisis) concomitant to the interruption of the cerebro-cerebellar circuit. A small-scale case report found that three patients with AH caused by subcortical white matter lesions showed crossed cerebellar diaschisis (Flint et al. 2006). Conversely, Hiraga et al. (2007) reported that two of three AH cases resulting from cortical

infarctions who underwent single-photon emission CT did not show crossed cerebellar diaschisis, indicating that crossed cerebellar diaschisis is not always associated with cortical AH. In addition, AH patients with basis pontine infarction and crossed cerebellar diaschisis have been reported (Sakai et al. 1986), as well as a case of AH with crossed cerebellar diaschisis due to Creutzfeldt–Jakob disease (Kastenbauer et al. 2001). Clearly, the pathological conditions required for coincident crossed cerebellar diaschisis and AH have not been elucidated.

Stroke Etiology of AH

AH was initially described as a lacunar syndrome; however, many subsequent reports have demonstrated that in addition to lacunar infarction, ICH, cardioembolic stroke, and large-artery atherosclerosis can cause AH. Gan et al. (1997) showed that of 39 confirmed AH patients, 72% cases had lacunar infarction, 5% cases had atherosclerosis, 5% cases were likely caused by cardioembolic stroke, and 18% cases were cryptogenic. Similarly, Arboix et al. (2000) found that 15 of 17 AH patients had lacunar infarction, while one each had thrombotic or cardioembolic stroke. In another sample of 29 AH patients (Arboix et al. 2010), 24 showed lacunar infarction, and 5 had nonlacunar stroke. Thus, lacunar stroke is the most frequent stroke mechanism in AH. However, a large-scale study with 100 AH patients (including 5 ICH patients) showed that 10% had large-artery disease, 12% had embolic stroke, 59% had hypertensive arteriolopathy, and 19% had AH of undetermined etiology (Moulin et al. 1995). Another large-scale diagnostic evaluation reported that 47% of the cases were attributed to small-vessel disease, 11% to cardioembolism, and 7% to artery embolism (Gorman et al. 1998). A DWI study in 36 AH patients revealed that 50% had no visible lacunae and no significant correlation was observed between the infarct location and AH syndrome (De Reuck et al. 2008). Other studies reported that venous infarction can be the cause of AH following IS (Taly et al. 1988).

In summary, reported cases of AH are most frequently associated with lacunar infarction while large-scale studies have found no definite association between AH and lacunar infarction. New imaging techniques like DWI should be applied in these large-scale studies to settle this issue.

Diagnostic Testing for AH

A patient with acute onset AH should first be examined for acute IS. Brain CT or MRI, or both, should be performed immediately. In addition, electrocardiogram, chest X-rays, and laboratory blood testing should be performed to detect traditional risk factors like atrial fibrillation, diabetes mellitus, and hypercholesterolemia. DWI is the best method to precisely localize infarcts, while conventional MRI has limited

utility due to the difficulty in differentiating between acute and chronic lesions. In fact, previous AH studies using conventional MRI or CT have shown no causative lesions in 22–33% of cases (Colombo et al. 1986; Crespi et al. 1988; Moulin et al. 1995; Gorman et al. 1998; Arboix 2004). In a large-scale study of 100 patients who had confirmed AH associated with hemorrhage or infarction, 77% had localized infarction, but the remaining 23% had no visual lesion or had only leukoaraiosis (Moulin et al. 1995). Another large-scale study (Gorman et al. 1998) comprising 45 patients with AH showed MRI abnormalities in 24 of 30 patients and CT abnormalities in 20 of 45 patients. No single lesion was identified as the probable cause of AH in 15 of the 45 patients (33%), 9 of whom had undergone MRI. On the other hand, a DWI study of 29 AH patients showed that 96% had lesions (Hiraga et al. 2007). Thus, in studies not using DWI, small cortical or subcortical lesions or brainstem infarcts may have been overlooked. In other cases, acute lesions may not have been distinguished from chronic lesions.

However, it is clear that AH is not always associated with visible lacunar infarctions. Therefore, in selected patients, neurovascular imaging techniques such as MR angiography and cervical sonography, and cardiac evaluation using echocardiogram and Holter monitoring should be performed to detect the possibility of an underlying stroke mechanism. Of course, positron emission tomography or single-photon emission CT is not necessary for the diagnosis of AH. If AH is not associated with stroke, as suggested by the clinical course or additional symptoms, cerebrospinal fluid or serological studies should be performed as required. In addition, enhanced MRI/CT should be performed in selected cases. Multiple sclerosis and acute infection can mimic AH due to stroke, so these pathologies should be confirmed or eliminated.

Treatment for AH

The most effective treatment strategy will depend on the underlying etiology as AH can involve lacunar infarctions, cardioembolism, large-artery atherosclerosis, infection, trauma, or tumors. Anticoagulant therapy and antiplatelet therapy should be started if needed in AH cases due to IS. Recently, a case of AH due to basilar artery occlusion was successfully treated by tissue plasminogen activator (Cho et al. 2010). Another single case report suggested that the anticholinergic agent trihexyphenidyl may be useful in the amelioration of disabling ataxia and unilateral tremor in AH patients (Jabbari et al. 1983).

Prognosis of AH

Most studies have indicated a relatively positive prognosis for patients with AH following IS. In the original report, Fisher (1978) described an excellent outcome in his cases and, ever since, many case reports have mirrored his findings. In a study of 20 AH cases following IS, ICH, or other conditions, 11 demonstrated total recovery

and 9 of 20 cases showed residual deficits. The best outcomes were observed in patients with small infarction or hemorrhage, and a more complete improvement in paresis than in ataxia was observed (Taly et al. 1988). Hypesthetic AH due to thalamic infarction also showed good recovery, with recovery from motor and sensory disturbance occurring before the return of ataxia (Melo et al. 1992). Another follow-up report showed that of eight AH patients who were independent before stroke, seven resumed independent lifestyle 1 month after onset (Bamford et al. 1987). A larger-scale study involving 45 AH patients reported that 27% completely recovered, 50% had only mild residual abnormalities, 8% exhibited nil or only minimal improvement, 12% developed recurrent stroke, 4% died, and 4% developed poststroke epilepsy (Gorman et al. 1998). This report also suggested that the ataxic signs were more prominent and longer lasting than the corticospinal tract signs. Hiraga et al. (2007) reported that the mean modified Rankin Scale scores of AH patients at discharge were 1.7 for AH associated with corona radiata/internal capsule infarcts, 2.3 for AH involving the pons, and 1.3 for frontal cortex/subcortical infarcts. These results again indicate that many AH patients resume independent lifestyle. In summary, AH following IS has a relatively positive prognosis, although cerebellar-type ataxia may persist in a fraction of patients.

Conclusions and Future Directions

AH is usually caused by IS, especially lacunar infarction. The frequency of AH following cardioembolic stroke and large-artery atherosclerosis is also significant (10–20% of AH cases). AH can develop with or without lacunar infarctions. The most common loci for lesions leading to AH are the pons, internal capsule, lateral thalamus, and corona radiata. The diagnostic criteria for AH, especially the noncore symptoms, were not applied uniformly in many of the studies reviewed here. In addition, these studies employed the techniques available at the time when they were conducted, which may have limited some of their conclusions. For example, many did not use DWI, one of the most reliable methods to localize small acute lesions. It is clear from these inconsistencies that the diagnostic criteria for AH should be unified. Moreover, in this new era of high-resolution brain imaging, the exact lesion location should be reexamined in surviving patients by DWI. The frequency of AH in stroke patients, the exact frequency of AH due to IS and other causes, the clinical features (paresis pattern, sensory disturbance), and prognosis (especially recurrent stroke rate) should be examined in a nationwide or international study by using the latest imaging technology. Additionally, the assumption that a lacunar etiology is the most likely cause of AH should be reexamined. The underlying mechanisms of AH are subject to debate, especially the mechanism of homolateral ataxia. To examine the underlying mechanisms of AH, large-scale studies using SEPs, the cerebellar electrical stimulation method, and single-photon emission CT/positron emission tomography should be employed. Most importantly, novel treatment modalities are needed.

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