8 Symptoms and Signs of Hemorrhagic Stroke

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A patient with acute hemorrhagic stroke (HS) may present with a sudden onset of focal neurologic deficits and other symptoms due to increased intracranial pressure. Most HS patients visit an emergency center through a prehospital delivery system instead of an outpatient clinic. Because HS is very likely to worsen during the first 24 h, the physician in the emergency center who is in charge of such a patient should promptly suspect the possibility of HS on the basis of the patient's medical history and symptoms and immediately perform a brain computed tomography (CT). HS, which mainly presents as intracerebral hemorrhage (ICH) or subarachnoid hemorrhage (SAH), is caused by a rupture of cerebral arteries. Because the brain has the lowest tissue pressure in the human body, bleeding into the brain is likely to persist for more than several hours, which increases the risk of a poorer outcome. Therefore, the process of diagnosis and treatment for HS should begin as early as possible. The first step to a quicker HS diagnosis is an understanding of its symptoms and signs.

In fact, within emergency centers, the symptoms of HS that duty doctors should pay attention to are easily identifiable. In any case, the possibility of a stroke should be promptly taken into consideration, if a focal neurologic deficit develops suddenly, and brain CT should be performed immediately. If a headache is accompanied by other neurologic symptoms, the probability of HS is greatly increased. This chapter lists various symptoms and signs of HS and categorizes them for better understanding. It is true that, compared to the past, the necessity for understanding symptoms and signs of stroke is reduced, because of the remarkable developments in brain imaging. However, we should keep in mind that the more we know, the more likely it is that the patient will get faster and more appropriate care.

8.1 Symptoms and Signs of Intracerebral Hemorrhage

ICH is caused by a rupture of small penetrating or leptomeningeal arteries branching from the large intracranial arteries that run through the subarachnoid space. This rupture results in extravasation of blood, leading to brain tissue damage.

Two important facts help in understanding the symptoms of ICH. Firstly, the mass effect of the hematoma itself causes symptoms. Extravasated blood creates a space-occupying lesion in the limited space within the cranial cavity and subsequently increases intracranial pressure (ICP). This increased ICP typically causes headache, nausea, and vomiting. Secondly, most ICHs

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develop from "cerebral arteriolosclerosis," which is caused by vascular risk factors such as long-standing hypertension. Lacunar infarction, a subtype of ischemic stroke, is caused by small vessel occlusions, whose underlying pathological findings are almost identical to those of ICH. Simply stated, if cerebral arteriolosclerotic lesions suddenly block blood vessels, lacunar infarction will occur, and if they rupture, ICH will develop. In patients with small-sized ICHs, symptoms may not be indistinguishable from those of lacunar infarctions. Since ICH generally has a progressive nature, most ICH cases are more severe than lacunar infarctions. Cerebral amyloid angiopathy (one of the main causes of ICH, especially lobar hemorrhage) is pathophysiologically unlikely to cause lacunar infarctions. In lobar hemorrhages, symptoms may appear to be similar to those in territorial rather than in lacunar infarctions.

In summary, ICH can present with i) general symptoms such as decreased level of consciousness, nausea, vomiting, and headache, and ii) focal neurologic impairments according to the ICH location (Fig. 8.1). If the size of the hematoma is larger, symptoms such as loss of consciousness or seizures appear more frequently, neurological damage becomes more aggravated, and consequently, the prognosis of the patient may be worse.

8.1.1 Lobar Hemorrhage

Headache is the most common symptom in patients with lobar hemorrhage and is found in about 60–70% of the patients [1]. Nausea and/or vomiting is the second most common with about 30%, followed by seizures with about 20% [1]. Seizures occur more frequently in lobar hemorrhage than in other types of hemorrhage, which is directly related to the involvement of the cerebral cortex. At the initial stage, a serious depression in mental functions such as semicoma or coma is not common, but 10% of the patients show a decreased level of consciousness [1]. Lobar hemorrhages occur most frequently in the parieto-occipital area, and accordingly, hemisensory dysfunction, hemibody neglect, and visual field defects are frequently observed in these cases. Temporal lobar hemorrhage in the left side may cause sensory-dominant aphasia (Wernicke type) whereas that in the right side may arouse acute confusional states or agitated confusion. Frontal hemorrhage may cause hemibody weakness on the opposite side and may rarely induce mental status dysfunction such as abulia or apathy.

8.1.2 Basal Ganglia Hemorrhage

Basal ganglia hemorrhage, which occurs mainly in the putamen or the internal capsule, is the most common type of ICH. This is because of the anatomy of the lenticulostriate arteries, which supply the basal ganglia. As penetrating arteries, which directly branch from the internal carotid artery, they are vulnerable to high blood pressure. Basal ganglia hemorrhage can be asymptomatic if the hemorrhage is small in size and remains within the putamen. A hemorrhage, which expands into the surrounding area, especially the posterior limb of the internal capsule, causes in most cases hemibody weakness in the contralateral side. Hemisensory loss in the contralateral side may be seen in case of an expansion into the thalamus. Depending on the degree of involvement of the thalamus, eyeball gaze abnormalities, altered consciousness, and visual field defects may be present. When an intraventricular hemorrhage (IVH) or midline shift due to mass effect occurs, the patient's consciousness level may deteriorate, and the patient may even become comatose. Involvement of a lobar area can be accompanied by various forms of aphasia and mental state dysfunction. Taken together, the most common symptoms of basal ganglia hemorrhage are headache and contralateral weakness, but patients may present various neurological symptoms depending on the extent of expansion into the surrounding tissue.

Caudate hemorrhage usually shows very mild or even no distinct symptoms. Mild cognitive or behavioral abnormalities have been reported, but they are not common. Thus, caudate hemorrhages are usually identified as previous bleeding lesions such as slit-like lesions in magnetic resonance imaging. Because the caudate nucleus is directly in contact with the lateral ventricle, a sizable

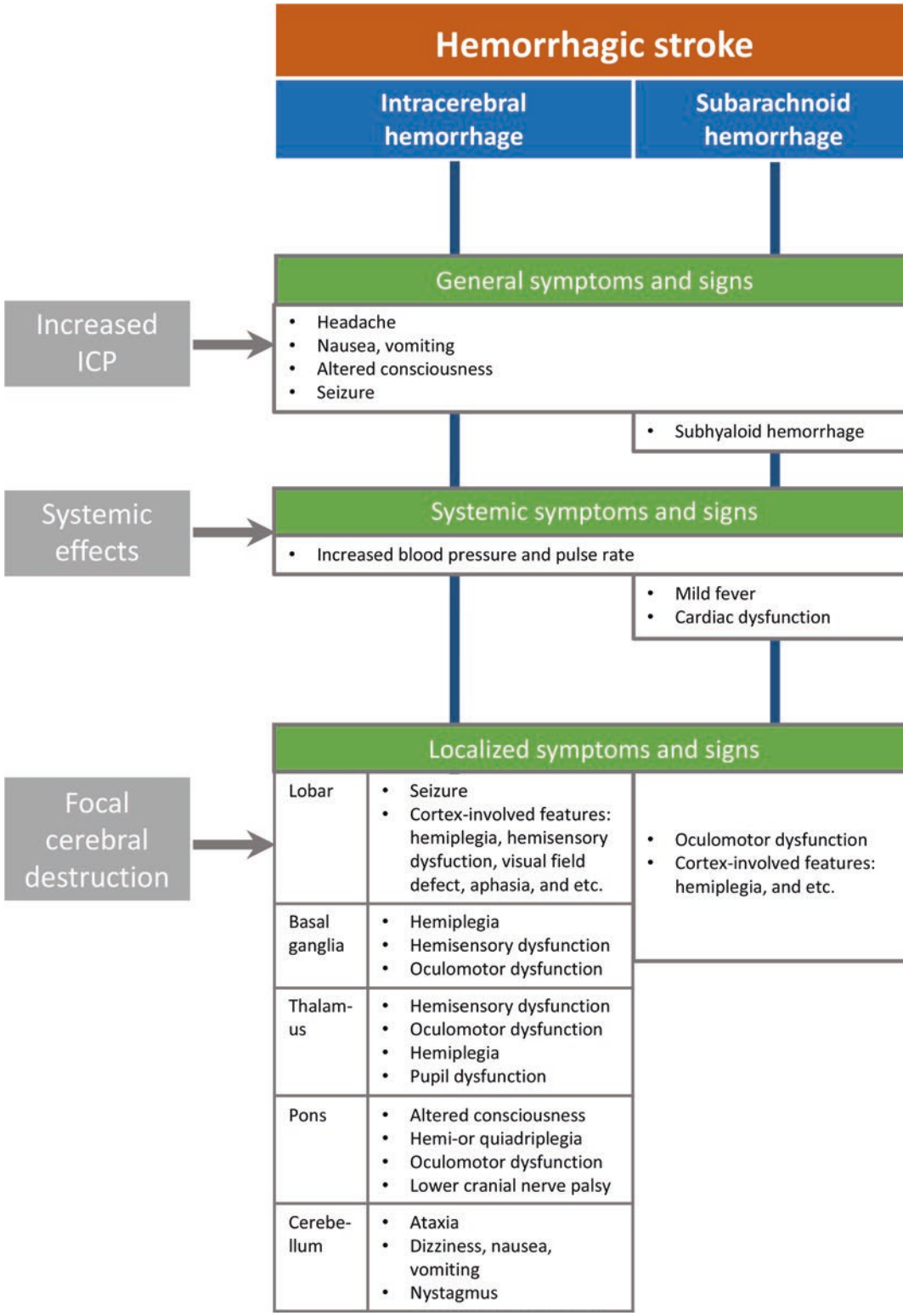

Fig. 8.1 Symptoms and signs of hemorrhagic stroke. *ICP* intracranial pressure

hemorrhage in this area is likely to cause bleeding into the lateral ventricle. The possibility of a caudate hemorrhage should be considered when primary IVH is detected without lesions of the brain parenchyma.

8.1.3 Thalamic Hemorrhage

Thalamic hemorrhages show symptoms similar to thalamic infarctions when their diameter is less than 2 cm [2]. Hemorrhages in the posterolateral portion of the thalamus are characterized mainly by hemisensory loss due to an involvement of ventral posterolateral, ventral posteromedial, and/or ventral lateral nuclei. These hemorrhages may be accompanied by vertical gaze abnormality and small, fixed pupils. The posterolateral portion is the most common site of thalamic hemorrhage. Hemorrhages in the anterior portion may result in decreased mental status and/or neuropsychiatric disturbances, and hemorrhages in the medial portion may cause vertical gaze abnormality, amnesia, and/or abulia. The vertical gaze limitation is known to occur in cases with involvement of the medial longitudinal fasciculus of the rostral interstitial nucleus, the vertical gaze center in the midbrain tegmentum. Involvement of the posterior part of the thalamus may be associated with visual field defects due to the destruction of the lateral geniculate body.

A relatively large thalamic hemorrhage (>2 cm) has four major symptoms: severe hemiparesis, hemisensory loss, vertical gaze abnormality, and constricted, fixed pupils [3]. These symptoms are not always present, but the symptoms and the prognosis worsen with an increase in the size of the bleed. Oculomotor dysfunction may present as skew deviation and conjugate eyeball deviation on the lesion side or opposite side to the lesion. Speech disturbances and neuropsychiatric symptoms such as amnesia and confusion may be present as well.

8.1.4 Pontine Hemorrhage

Pontine hemorrhage is not the most common ICH; its incidence is less than 5–10% of all cases of ICH [4]. However, it is the most severe hemorrhage type with the poorest prognosis having case fatality rate of 50% or more [4]. Patients with pontine hemorrhage often fall into deep coma due to disruption of the reticular activating system. In these cases, the prognosis is extremely poor due to the accompanying quadriplegia. Hemorrhages in the anterior portion of the pons involve the corticospinal tract leading to hemiparesis. Facial palsy, dysarthria, dysphagia, dizziness, vertigo, and deafness may be caused by the destruction of the nuclei and tracts of the 7th, 8th, 9th, and 10th cranial nerves. Involvement of the respiratory center may also result in apneustic or cluster breathing patterns. A variety of oculomotor dysfunctions may occur, such as unilateral or bilateral Horner syndrome, ocular bobbing, ocular dipping, ping-pong gaze, skew deviation, horizontal conjugate gaze palsy, and one-and-a-half syndrome.

8.1.5 Cerebellar Hemorrhage

Cerebellar hemorrhages usually develop around the dentate nucleus. In the case of expansion to the pontine tegmentum, early surgery may be needed for treatment or prevention of an obstructive hydrocephalus caused by compression of the fourth ventricle or its outlets – the lateral aperture (foramen of Luschka) and the median aperture (foramen of Magendie). Headache is relatively common in cerebellar hemorrhage, but impairment of intrinsic functions of the cerebellum may present as limb/truncal ataxia, dizziness, nausea, vomiting, and gaze-evoked nystagmus. If hemorrhage is confined to the cerebellum without involvement of the brain stem, hemibody weakness is usually absent.

8.2 Symptoms and Signs of Subarachnoid Hemorrhage

Subarachnoid hemorrhage is most commonly caused by traumatic brain injury, but about 80% of non-traumatic SAHs are due to a rupture of an intracranial aneurysm; about 10% are perimesencephalic SAHs [5]. Because non-traumatic SAHs show the poorest prognosis among the various subtypes of stroke, a better understanding of its

symptoms and signs is an essential prerequisite for rapid diagnosis and treatment (Fig. 8.1).

8.2.1 Symptoms of Subarachnoid Hemorrhage

8.2.1.1 Headache and Meningeal Irritation Signs

Headache is the most common and a characteristic symptom in patients with SAH. More than 70% of SAH patients complain of headache. Because the intracranial arteries that run in the subarachnoid space anatomically belong to the large arteries, sudden rupture of these vessels leads to bleeding into the intracranial space with arterial pressure. Meningeal distension and irritation due to elevated pressure of the cerebrospinal fluid cause severe headache. SAH patients often refer to it as the most severe headache they have experienced in their lifetime – "like a hammer hit," "like a bolt out of the blue," and so on. Headaches occur suddenly and become maximally severe within a few seconds to several minutes. Patients generally report generalized headaches, but the pain is often localized near the rupture site and occasionally involves the upper neck. Increased ICP is accompanied by nausea and vomiting in many cases, leading to severe convulsions and decreased consciousness. In addition to vomiting, meningeal irritation signs such as posterior neck discomfort, photophobia, and phonophobia may also be present. SAH is the worst type of stroke, and it is reported that about 10–12% of patients die before being transferred to the emergency center. If the SAH headache is not severe, it is often difficult to distinguish from other types of headache. Headaches in SAH patients may mimic vascular headaches such as migraine and tension-type headache or pain of muscular origin in the head and neck region. When the diagnosis is based only on the nature and severity of the headache, it is often difficult to differentiate headache due to SAH from other forms of headache. Brain CT is generally recommended for patients with the following conditions: (1) sudden headache in adults, (2) headaches accompanied by seizures or other neurological symptoms, and (3) severe headache that has not been experienced before.

8.2.1.2 Warning Leaks

The prodromal symptom of SAH, called "warning leaks" or "sentinel headache," refers to a situation in which a small extravasation of the blood from an aneurysm causes headaches before the incidence of an aneurysmal SAH. It is reported that these symptoms are present in 20–40% of SAH patients [6, 7] and are generally regarded as important clinical features to prevent of SAH. However, these findings were largely from retrospectively designed studies, a typical setting in which patients are exposed to a recall bias. In a prospectively designed study, only two of the 37 SAH patients were suspected of having actual warning leaks [6]. It is very difficult to distinguish real warning leaks because similar headaches are a common complaint in normal adults. In conclusion, warning leaks need to be suspected only in patients with known intracranial unruptured aneurysms, but their general value is quite limited in clinical practice.

8.2.1.3 Decreased Levels of Consciousness

Loss of consciousness is presented in about 50–70% of SAH patients [8] and mostly caused by aneurysmal SAH – rarely in perimesencephalic SAH. Behavioral symptoms such as confusion and agitation might occur together with altered consciousness, and seizures are common. Altered consciousness occurs mainly due to decreased global cerebral perfusion caused by increased ICP, which might result from accompanying cardiac arrhythmia or seizure.

8.2.1.4 Seizures

Seizures occur in about 10–20% of all SAH patients [9]. It is caused by blood-induced irritation of the cerebral cortex, so it mostly occurs in aneurysmal SAH and rarely in perimesencephalic SAH. Compared to other subtypes of stroke, the incidence of seizures in SAH is very high.

8.2.2 Signs of Subarachnoid Hemorrhage

8.2.2.1 Vital Signs

In most cases of SAH, vital signs are altered at the time of admission. Blood pressure and pulse

rate are typically increased. Body temperature may be normal or slightly elevated to about 38°C, and respiration is normal or slightly increased. High blood pressure at the time of admission may aggravate SAH status or induce rebleeding and therefore needs to be treated by parenteral administration of antihypertensive drugs. However, increased blood pressures is usually the result of a sudden increase in sympathetic tone, especially in previously normotensive patients, and continuous lowering of blood pressure may cause global cerebral ischemia. Accordingly, blood pressure control should be carefully performed in the early stages, with continuous monitoring of the blood pressure. Acute cardiac abnormality is common: troponin elevation occurs in 20–30% of SAH patients, and ECG changes including cardiac arrhythmia occur in more than 50% [10]. Left ventricular dysfunction is also common but usually transient.

8.2.2.2 Ocular Signs

A sudden increase in ICP due to SAH prevents venous outflow from the retina, resulting in venous hemorrhage, which is called subhyaloid hemorrhage. It occurs in about 10–20% of SAH survivors [11], who sometimes complain of scotoma due to retinal dysfunction at the bleeding site.

Oculomotor dysfunction, including third nerve palsy, sixth nerve palsy, and impaired vertical gaze (Parinaud's syndrome), is common in SAH patients. Third nerve palsy is the most important sign. It is indicative of an aneurysm rupture of the posterior communicating artery but is rarely caused by aneurysm ruptures of the superior cerebellar artery or the posterior cerebral artery. Third nerve palsy may occur without bleeding even in an unruptured aneurysm if the aneurysm expands rapidly. In this case, aneurysm ablation therapy should be performed immediately because it suggests an impending rupture of the aneurysm. Sixth nerve palsy may be caused by increased ICP and is often seen bilaterally. Parinaud's syndrome with vertical eye movement limitation implies a proximal dilatation of the aqueduct of Sylvius due to acute hydrocephalus, a state of pressing the vertical interstitial nucleus of the medial longitudinal fasciculus as the vertical gaze center.

8.2.2.3 Other Signs

Motor weakness may occur depending on the nature of SAH, for instance, when bleeding from an aneurysmal rupture directly destroys brain parenchyma or when SAH causes a severe, focal, chemical meningoencephalitis due to extravasated blood. Hemiparesis might be present as observed in cases of ischemic stroke, but it rarely occurs, with the exception of cases wherein an aneurysm of the middle cerebral artery ruptures. In some cases, monoparesis and paraparesis might be caused by the rupture of an aneurysm of the anterior communicating artery.

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