We are IntechOpen, the world’s leading publisher of Open Access books Built by scientists, for scientists

6,600
Open access books available

177,000
International authors and editors

195M
Downloads

154
Countries delivered to

TOP 1%
Our authors are among the most cited scientists

12.2%
Contributors from top 500 universities

WEB OF SCIENCE™
Selection of our books indexed in the Book Citation Index in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected. For more information visit www.intechopen.com
Intracerebral Hemorrhage: Influence of Topography of Bleeding on Clinical Spectrum and Early Outcome

Adrià Arboix1 and Elisenda Grivé2

1Cerebrovascular Division, Department of Neurology, Hospital Universitari del Sagrat Cor, Universitat de Barcelona, Barcelona, Spain
2Servei de Neuroradiologia, CRC, Hospital Universitari del Sagrat Cor, Barcelona, Spain

1. Introduction

Approximately 10-20% of strokes are due to intracerebral hemorrhage (ICH) [1]. Hospital admissions for ICH have increased by 18% in the past 10 years. ICH is a medical emergency. Rapid diagnosis and attentive management of patients with ICH is crucial because hematoma expansion and early deterioration is common in the first few hours after ICH onset. The clinical spectrum and outcome of a patient with ICH is directly related to the site of bleeding. The prognosis and treatment of ICH often depends on the areas affected by the hemorrhage [3-5]. Particular locations, such as the cerebral lobes, right putamen and cerebellum are relatively accessibile to surgical drainage, whereas other areas, such as the thalamus and the brainstem are inaccessible [6].

It is very difficult to determine whether the presenting neurological symptoms are due to cerebral ischemia or ICH based on the clinical characteristics alone. Vomiting, elevated systolic blood pressure (SBP) (>220 mmHg), severe headache, coma or decreased level of consciousness, and progression of neurological deficit over minutes or hours are suggestive of ICH, although none of these features are specific and, therefore, neuroimaging examination is mandatory. Neuroimaging data, particularly computed tomography (CT) is needed to rule out stroke mimics, to confirm the clinical diagnosis, and to distinguish ischemia from ICH [4,5].

The influence of the site of bleeding on the clinical spectrum and outcome in patients with ICH is still a poorly defined aspect of the disease. Factors associated with outcome in ICH have been evaluated in many studies but the findings are of limited utility because they have typically considered broad groups of patients with different etiologies or have otherwise employed univariate rather than factorial techniques for the analysis of data. Moreover, prognostic variables related to morbidity and mortality are of great importance but remain difficult to establish clearly because of methodological problems, including sample selection bias, timing of initial assessment, criteria for measuring outcome, and the role of other confounding factors. Although community-based studies and prospective stroke registries have provided data on the identification of prognostic factors in ICH...
patients, there is a scarcity of information on the differences across the clinical spectrum and outcome of hemorrhagic stroke according to the site of bleeding [5-8].

The aim of this chapter is to determine the influence of topography of hemorrhage on the clinical spectrum, in-hospital mortality, and early outcome in ICH patients according to data collected from a review of the literature and the authors’ experience based on a large hospital-based stroke registry (Sagrat Cor Hospital of Barcelona Stroke Registry) in Barcelona, Spain.

In relation to the site of bleeding, seven topographies were analyzed. These included the thalamus, internal capsule and basal ganglia, cerebral lobes, cerebellum, brainstem, multiple topographic involvement (when more than one of these areas was affected), and primary intraventricular hematoma. Secondary intraventricular blood expansion (evidence of intraventricular blood on CT and/or magnetic resonance imaging [MRI] scans) for each topography was also assessed.

In relation to localization of the hemorrhage in the Sagrat Cor Hospital of Barcelona Stroke Registry (Table 1), lobar ICH was the most frequent (33.2%) followed by hemorrhages in the

| Anatomic localization                          | No. patients (%) |
|------------------------------------------------|------------------|
| Lobar                                         | 76 (33.2)        |
| Frontal                                       | 8                |
| Parietal                                      | 23               |
| Temporal                                     | 14               |
| Occipital                                     | 13               |
| Frontoparietal                                | 3                |
| Temporoparietal                               | 6                |
| Temporo-occipital                             | 6                |
| Parieto-occipital                             | 2                |
| Frontoparietal temporal                       | 1                |
| Thalamus                                      | 31 (13.5)        |
| Cerebellum                                    | 15 (6.6)         |
| Brainstem                                     | 15 (6.6)         |
| Mesencephalon                                 | 2                |
| Pons                                          | 6                |
| Medulla oblongata                             | 1                |
| Pons and mesencephalon                        | 6                |
| Internal capsule                              | 7 (3.0)          |
| Basal ganglia                                 | 24 (10.5)        |
| Internal capsule and basal ganglia            | 18 (7.9)         |
| Multiple topographic involvement              | 34 (14.8)        |
| Thalamus, internal capsule/basal ganglia      | 18               |
| Lobar, internal capsule/basal ganglia         | 10               |
| Lobar, internal capsule/basal ganglia, thalamus| 5                |
| Brainstem, basal ganglia                      | 1                |
| Primary intraventricular hemorrhage           | 9 (3.9)          |

Table 1. Site of bleeding in 229 patients with hemorrhagic stroke in the Sagrat Cor Hospital of Barcelona Stroke Registry
Intracerebral Hemorrhage: 
Influence of Topography of Bleeding on Clinical Spectrum and Early Outcome

thalamus (13.5%), basal ganglia (10.5%), internal capsule and basal ganglia (7.9%), cerebellum (6.6%), and brainstem (6.6%). Multiple topographic involvement was found in 14.8% of the patients and primary intraventricular hemorrhage in 3.9%. In these series of 229 consecutive cases, the main cause of ICH was hypertension in 124 patients, arteriovenous malformations in 11, hematologic disorders in 9, and other causes in 12. In 73 patients, the cause of bleeding was not identified [5]. MRI and CT with angiographic studies (where relevant), MR angiography (MRA), MR venography or CT angiography (CTA), are reasonably sensitive at identifying secondary causes of hemorrhage, including arteriovenous malformations, aneurysms, cavernous malformations, tumors, moyamoya, vasculitis and dural venous thrombosis. Digital subtraction angiography (DSA) may be considered if clinical suspicion is high but noninvasive studies do not show a clear cause particularly in young, normotensive and surgical candidates. DSA remains the gold standard for the evaluation of vascular anomalies and allows endovascular treatment, however the use of non-invasive MRA or CTA may help to exclude unnecessary invasive DSA [6].

Risk factors and clinical variables associated with different topographic locations are shown in Tables 2 and 3. Sensory deficit was significantly associated with thalamic ICH; lacunar syndrome and hypertension with internal capsule/basal ganglia ICH; seizures and non-sudden stroke onset with lobar ICH; ataxia with hemorrhage in the cerebellum; cranial nerve palsy with brainstem ICH; and limb weakness, diabetes, and altered consciousness with multiple topographic involvement. On the other hand, hypertension and sensory deficit were inversely associated with lobar and cerebellar ICH, respectively.

The in-hospital mortality rate was 30.6% (n = 70). Causes of death included cerebral herniation in 44 patients, pneumonia in 8, sepsis in 8, sudden death in 3, myocardial infarction in 1, pulmonary thromboembolism in 1, and unknown cause in 5. Mortality at 3 months was 34% in a review of 586 patients with ICH from 30 centers. In other studies it was 31% at 7 days, 59% at 1 year, 82% at 10 years and more than 90% at 16 years [5].

According to the different sites of bleeding, in-hospital mortality rates were 16.3% in internal capsule/basal ganglia ICH, 20% in cerebellar ICH, 25% in lobar ICH, 25.8% in thalamic ICH, 40% in brainstem ICH, 44.4% in primary intraventricular hemorrhage, and 64.7% in multiple topographic involvement. Intraventricular extension of the hemorrhage was associated with a significantly higher in-hospital mortality rate in all ICH topographies except for lobar hemorrhage (presence of intraventricular hemorrhage vs. absence 41.1 vs. 0%, in thalamic ICH; 50 vs. 9.8%, in internal capsule/basal ganglia ICH; 66.7 vs. 8.3%, in cerebellar ICH; 100 vs. 25%, in brainstem ICH; and 87.5 vs. 44.4%, in multiple topographic involvement). Survivors were significantly younger than patients who died (mean age 69.07 [12.75] vs. 73.94 [10.32] years), for whom the overall mean survival time was 15 (23) days [5].

2. Internal capsule/basal ganglia

The commonest location of hypertensive ICH is the lateral basal-ganglionic-capsular region (classical deep subcortical intracerebral hemorrhages) [9,10]. Patients with small hematomas in this topography have a good outcome. Small hematomas of subcortical topography in the internal capsule, but also in the basal ganglia and more infrequently in the pons, may cause a lacunar syndrome [11]. In a clinical series hypertension and lacunar syndrome were significantly associated with internal capsule/basal ganglia ICH (Table 4). The selection of
| Variable | Thalamus (n=31) vs remaining ICH | Internal capsule basal ganglia (n=49) vs. remaining ICH | Lobar (n=76) vs remaining ICH | Cerebellum (n=15) vs remaining ICH | Brainstem (n=15) vs remaining ICH | Multiple sites (n=34) vs remaining ICH | Intraventricular (n=9) vs Remaining ICH |
|----------|----------------------------------|--------------------------------------------------------|-------------------------------|----------------------------------|---------------------------------|-------------------------------------|--------------------------------------|
| Hypertension | 77.6 vs. 55.5%*                  | 42.1 vs. 69.9†                                        |                               |                                  |                                 |                                     |                                      |
| Diabetes   |                                  | 35.3 vs. 11.8†                                       |                               |                                  |                                 |                                     |                                      |
| Heart valve disease |                               |                                                        |                               |                                  |                                 |                                     |                                      |
| Atrial dysrhythmia |                               |                                                        |                               |                                  |                                 |                                     |                                      |
| Previous cerebral hemorrhage | 6.0 vs. 1.3§                    |                                                        |                               |                                  |                                 |                                     |                                      |
| Anticoagulant treatment |                               |                                                        |                               |                                  |                                 |                                     | 22.2 vs. 0.5§                        |
| Sudden onset | 57.9 vs. 73.8†                   |                                                        |                               |                                  |                                 |                                     |                                      |
| Non-sudden onset | 38.1 vs. 23.5§                   |                                                        |                               |                                  |                                 |                                     |                                      |
| Dizziness  | 60.0 vs. 5.6†                    |                                                        |                               |                                  |                                 |                                     |                                      |
| Seizures   | 4.1 vs. 5.0§                     | 11.8 vs. 1.3§                                        |                               |                                  |                                 |                                     |                                      |
| Nausea, vomiting | 66.7 vs. 20.6†                  |                                                        |                               |                                  |                                 |                                     |                                      |
| Altered consciousness |                               |                                                        | 76.5 vs. 40.5†                |                                  |                                 |                                     |                                      |
| Limb weakness | 40.0 vs. 79.9†                  |                                                        |                               | 97.1 vs. 73.8†                   |                                 |                                     |                                      |
| Sensory deficit | 72.4 vs. 46.4*                  | 6.7 vs. 53.7†                                        |                               | 73.5 vs. 46.1*                   |                                 |                                     |                                      |
| Ataxia     | 0 vs. 11.1§                      | 86.7 vs. 3.7†                                        |                               |                                  |                                 |                                     |                                      |
| Cranial nerve palsy | 0 vs. 13.1§                     |                                                        |                               |                                  |                                 |                                     | 66.7 vs. 4.7†                        |
| Lacunar syndrome | 18.4 vs. 4.4†                    |                                                        |                               |                                  |                                 |                                     |                                      |

Data expressed as percentages; *P < 0.2; †P < 0.01; ‡P < 0.001; §P < 0.3; ††P < 0.07; †‡P < 0.08; †††P < 0.05; ‡‡‡P < 0.06.

Table 2. Frequency of vascular risk factors and clinical features according to site of bleeding in 229 patients with intracerebral hemorrhage (ICH) in the Sagrat Cor Hospital of Barcelona Stroke Registry.
### Table 3. Predictive value of different risk factors and clinical variables on the site of bleeding in the Sagrat Cor Hospital of Barcelona Stroke Registry

| Bleeding topography                  | β     | SE (β) | Odds ratio (95% CI) |
|--------------------------------------|-------|--------|--------------------|
| Thalamus                             | 1.1729| 0.4365 | 3.23 (1.37 to 7.60) |
| Sensory deficit                      |       |        |                    |
| Internal capsule/basal ganglia       | 1.3373| 0.5346 | 3.81 (1.33 to 10.86) |
| Lacunar syndrome                     | 0.8065| 0.3855 | 2.24 (1.05 to 4.77) |
| Hypertension                         | 2.4178| 0.8278 | 11.22 (2.21 to 56.84) |
| Seizures                             | 0.8045| 0.3344 | 2.24 (1.16 to 4.31) |
| Non-sudden stroke onset              | −1.0669| 0.3091 | 0.34 (0.19 to 0.63) |
| Hypertension                         | 5.7560| 1.1653 | 316.09 (32.2 to 3102.63) |
| Cerebellum                           |       |        |                    |
| Ataxia                               | 3.7108| 0.6393 | 40.89 (11.67 to 143.15) |
| Sensory deficit                      |       |        |                    |
| Brainstem                            | −4.0215| 1.5093 | 0.02 (0.009 to 0.35) |
| Cranial nerve palsy                  | 3.1078| 0.6393 | 40.89 (11.67 to 143.15) |
| Sensory deficit                      |       |        |                    |
| Multiple topography                  |       |        |                    |
| Limb weakness                        | 2.3628| 1.0662 | 10.62 (1.31 to 85.84) |
| Diabetes mellitus                    | 1.5992| 0.5099 | 4.95 (1.82 to 13.44) |
| Altered consciousness                | 1.3950| 0.4631 | 4.03 (1.63 to 10.0) |

Hypertension may be explained because blood supply of the putamen is derived predominantly from penetrating branches of the middle cerebral artery, which are the arterioles most frequently affected by hypertension [9,12]. This finding is consistent with other studies showing that the lateral ganglionic region is the most common topography of deep hypertensive ICH, and that a great proportion of hematomas found in the putaminal region are hypertensive [3,12] (Figures 1 and 2).

![Axial CT shows acute hypertensive putaminal hematoma with early peripheral edema.](https://www.intechopen.com)
Hemorrhage in the caudate nucleus accounts for approximately 7% of ICH. The symptoms and signs in caudate hemorrhage closely mimic SAH but the CT appearance of blood in the caudate and lateral ventricles is distinctive. Larger hemorrhages are more likely to rupture into the ventricle and have a much higher mortality than do small putaminal hematomas [4,12].

Fig. 2. Subacute intraparenchymatous hematoma in the right basal ganglia/external capsule. A) Axial T1WI MR showing peripheral hyperintensity and central isointensity related to different ICH temporal staging. Signal changes first occur peripherally and progress centrally; B) Axial T2WI MR; C) Axial MRA (3D TOF) MIP image shows no vascular malformation.
| Variable                                           | Lobar hemorrhage | Deep subcortical hemorrhage | P value |
|----------------------------------------------------|------------------|----------------------------|---------|
| **Demographic data**                               |                  |                            |         |
| Male sex                                           | 44 (45.4)        | 59 (64.1)                  | 0.007   |
| Age, years, mean (SD)                              | 70.3 (14.3)      | 71.8 (10.9)                | 0.414   |
| **Vascular risk factors**                          |                  |                            |         |
| Hypertension                                       | 41 (42.3)        | 64 (69.6)                  | 0.001   |
| Diabetes mellitus                                  | 10 (10.3)        | 13 (14.1)                  | 0.281   |
| Ischemic heart disease                             | 6 (6.2)          | 6 (6.5)                    | 0.579   |
| Atrial fibrillation                                | 10 (10.3)        | 11 (12)                    | 0.448   |
| Valvular heart disease                             | 5 (5.2)          | 1 (1.1)                    | 0.118   |
| Congestive heart failure                           | 3 (3.1)          | 2 (2.2)                    | 0.525   |
| Previous transient ischemic attack                 | 7 (7.2)          | 3 (3.3)                    | 0.188   |
| Previous cerebral infarction                       | 8 (8.2)          | 9 (9.8)                    | 0.454   |
| Previous intracerebral hemorrhage                  | 8 (8.2)          | 1 (1.1)                    | 0.021   |
| Chronic obstructive pulmonary disease              | 6 (6.2)          | 5 (5.4)                    | 0.537   |
| Chronic renal disease                              |                  |                            |         |
| Chronic liver disease                              | 8 (8.2)          | 2 (2.2)                    | 0.060   |
| Obesity                                            | 1 (1)            | 7 (7.6)                    | 0.027   |
| Alcohol consumption (> 80 g/day)                   | 3 (3.1)          | 6 (6.5)                    | 0.444   |
| Smoking (> 20 cigarettes/day)                      | 11 (11.3)        | 8 (8.7)                    | 0.360   |
| Hyperlipidemia                                     | 9 (9.3)          | 13 (14.1)                  | 0.208   |
| Anticoagulant treatment                            | 4 (4.1)          | 2 (2.2)                    | 0.367   |
| Peripheral vascular disease                        | 2 (2.1)          | 6 (6.5)                    | 0.123   |
| **Clinical features**                              |                  |                            |         |
| Sudden onset (min)                                 | 59 (60.8)        | 65 (70.7)                  | 0.102   |
| Acute onset (hours)                                | 28 (28.9)        | 20 (21.7)                  | 0.169   |
| Headache                                           | 45 (46.4)        | 27 (29.3)                  | 0.012   |
| Seizures                                           | 11 (11.3)        | 2 (2.2)                    | 0.012   |
| Nausea, vomiting                                   | 18 (18.6)        | 17 (18.5)                  | 0.569   |
| Decreased consciousness                           | 44 (45.4)        | 31 (37.5)                  | 0.068   |
| Motor deficit                                      | 66 (68)          | 77 (83.7)                  | 0.009   |
| Sensory deficit                                    | 36 (37.1)        | 55 (59.8)                  | 0.001   |
| Homonymous hemianopsia                             | 31 (32)          | 19 (20.7)                  | 0.055   |
| Aplasia, dysarthria                                | 35 (36.1)        | 32 (34.8)                  | 0.486   |
| Ataxia                                             | 3 (3.1)          | 3 (3.3)                    | 0.633   |
| Absence of neurological deficit at discharge       | 6 (6.2)          | 5 (5.4)                    | 0.537   |
| In-hospital mortality                              | 26 (26.8)        | 18 (19.6)                  | 0.158   |
| Respiratory complications                          | 7 (7.2)          | 10 (10.9)                  | 0.267   |
| Urinary complications                              | 13 (13.4)        | 15 (16.3)                  | 0.361   |
| Infectious complications                           | 15 (15.5)        | 24 (26.1)                  | 0.052   |
| Length of hospital stay, median (IQR)              | 17 (18)          | 20 (16)                    | 0.383   |

Table 4. Comparison between 97 patients with lobar hemorrhage and 92 patients with deep subcortical bleeding
3. Thalamic hemorrhage

Thalamic hematomas (Figures 3 and 4) is a subgroup of hemorrhagic stroke that accounted for 1.4% of all cases of stroke and 13% of intracerebral hemorrhages, a percentage in the range between 6% and 25.6% in the series of other authors [3,7,13].

![Axial CT reveals an acute anterior left thalamic hematoma.](image1)

![Axial CT shows a large thalamic hematoma with intraventricular rupture.](image2)

Sensory deficit was significantly associated with thalamic ICH [4,5]. It has been shown that 74.2% of patients with hemorrhage in the thalamus showed sensory deficit, which coincide in part with early observations emphasizing that the predominance of sensory deficit over motor is one cardinal feature of thalamic ICH [4]. Sometimes the contralateral limbs are
ataxic or have choreic movements. The commonest oculomotor abnormalities include paralysis of upward gaze, often with one or both eyes resting downward, and hyperconvergence of one or both eyes. These ocular abnormalities are due to direct extension of the hematoma to the diencephalic-mesencephalic junction or to compression of the quadrigeminal plate region.

The topography of thalamic lesion [14] was anterior in 6% of cases (behavioral abnormalities predominate), posteromedial in 24% (abnormalities of consciousness, papillary function and vertical gaze predominate), posterolateral in 48% (sensorimotor signs predominate), dorsal in 2% (slights sensorimotor signs usually transients and aphasia and behavioral are common) and affected all thalamic vascular territories in 20%. Intraventricular involvement was present in 42.6% of patients.

Thalamic hemorrhage is a severe clinical condition with in-hospital mortality rate of 19%, and symptom-free at discharge from the hospital documented in only 2.1% [15]. The mortality rate of thalamic hemorrhage ranges between 17% and 52% in the experience of different authors [14,16]. On the other hand, the mortality rate of thalamic hemorrhage is generally lower than that of brainstem hemorrhages or cerebral hemorrhages of multiple topographies, which show a very high in-hospital mortality rate usually greater than 40% [5]. The mortality rate in patients with thalamic hemorrhage, however, is higher than that of patient with capsular stroke [5,17]. Altered consciousness, intraventricular hemorrhage and age have been shown to be independent predictors of in-hospital mortality in patients with thalamic hematoma [15,17]. In summary, approximately one of each 10 patients with acute intracerebral hemorrhage had a thalamic hematoma. Patients with thalamic hemorrhage show a differential clinical profile than patients with internal capsule-basal ganglia ICH. Altered consciousness, intraventricular involvement and advanced age have been found to be independent predictors of in-hospital mortality.

4. Lobar hemorrhage

The frequency of lobar hemorrhage varies between 24% to 49% in the different clinical series reported in the literature [1,5,18]. In our experience, lobar bleeding was the most common ICH (33% of cases) [18]. The symptoms and signs in lobar hemorrhages are similar to cerebral infarctions. Seizures, non-sudden stroke onset, and hypertension were independent clinical factors related to the site of bleeding. Seizures occurred more frequently in lobar ICH than in the remaining ICH. Other studies have shown that seizures are more frequent in hemorrhagic than in ischemic stroke as well as more frequent in lobar than in deep hematomas mainly in the parietal and temporal lobes [19,20]. On the other hand, it has been generally considered that bleeding in ICH lasts only a few minutes. However, recent data show that substantial early hemorrhage growth in patients with ICH is common [21]. Explanation of the gradual onset of symptoms found in 38% of our patients with lobar ICH, suggests that the period of hematoma enlargement can extend for a number of hours from onset as a result of active bleeding, a phenomenon that is frequently but not always associated with clinical deterioration [4]. Lobar ICH was less commonly associated with hypertension than any of the remaining topographies. For this reason, non-hypertensive mechanisms of ICH including cerebral amyloid angiopathy (Figure 5), vascular malformations, sympathomimetic drugs, and bleeding disorders all have a tendency to cause predominantly subcortical lobar ICH with a lower frequency of deep hemispheric and brainstem hemorrhages [6,19].
Fig. 5. Lobar hemorrhage related to cerebral amyloid angiopathy. A) Axial T1WI MR shows subacute lobar hematoma in the left frontal lobe; in B) and C) axial T2*GRE MR, apart from the subacute lobar hematoma, superficial hemosiderosis, scattered microbleeds and a chronic right frontal cortical hematoma are shown.

In the comparative analysis between lobar hemorrhages and deep subcortical hemorrhages (Table 4), lobar hemorrhages were more common in women, in patients with previous ICH as well in those presenting with headache and seizures. In contrast, deep subcortical hemorrhages occurred more frequently in obese patients and were associated with motor and sensory deficits.

Lobar ICH is a severe disease, with in-hospital mortality (26.8%) and absence of neurological deficit at the time of hospital discharge being observed occasionally (6.2%). Lobar ICH may be considered a more benign condition as compared with brainstem hemorrhages or multiple topographic location [5,10] in which in-hospital mortality may be as high as 40%, but is more severe than capsular hemorrhages, which may even present as a lacunar syndrome, mainly pure motor hemiparesis or sensorimotor syndrome [11].

The lower frequency of hypertension in lobar ICH (42.3% vs. 69.6% in subcortical ICH) is a relevant clinical aspect that coincides with data reported in the literature [6], because high blood pressure has the lowest frequency as compared with other topographies of bleeding. However, other causes different from hypertension, such as arteriovenous malformations (8.5%), blood dyscrasias (5.5%) or anticoagulant iatrogenia (3%) are more common in lobar ICH. Because of the higher incidence of vascular malformations and other bleeding lesions in patients with lobar hematomas angiography is often indicated [6]. For patients presenting with lobar clots > 30 mL and with 1 cm of the surface, evacuation of supratentorial ICH by standard craniotomy might be considered [6].

In our experience of the Sagrat Cor of Barcelona Stroke Registry, chronic obstructive pulmonary disease (COPD), altered consciousness, previous cerebral infarction, chronic liver disease, female sex, seizures and headache were clinical variables independently associated with in-hospital mortality in the logistic regression analyses.

5. Cerebellar hemorrhage

This subgroup of hemorrhagic stroke account for 0.73% of total stroke and 6.9% of ICH, a percentage similar to 5–10% reported in the literature [4,9].

www.intechopen.com
Establishing the diagnosis of cerebellar hemorrhage is important because of the potentially serious outcome if not treated and the contrasting good prognosis after surgical treatment. Cerebellar hemorrhage usually originates in the region of the dentate nucleus, arising from distal branches of the superior cerebellar artery and the posterior inferior cerebellar artery (Figure 6). Characteristic presenting symptoms of cerebellar ICH include headache, vertigo, vomiting, and inability to stand and walk. Ataxia is an independent clinical factor associated with cerebellar ICH. In our experience, ataxia was found in 87% of patients and this finding agrees with the high frequency of cerebellar signs including gait ataxia, truncal ataxia, and ipsilateral limb ataxia reported by others [4,9].

Fig. 6. Right cerebellar hematoma in the region of the dentate nucleus. A) Axial CT shows acute hematoma with halo of surrounding edema; B) axial T1WI MR discloses the same hematoma in early subacute stage. Mass effect partially effaces the 4th ventricle.

Cerebellar hemorrhages are severe, with a high in-hospital mortality (21.4%) and functional deficit at hospital discharge in practically all patients. The in-hospital mortality of 21.4% observed in our patients [5] is lower than 39-47% observed in other studies [22-24] and similar to death rates (13% and 25%) reported by other authors [25,26]. Patients with larger cerebellar hematomas usually develop brainstem compression [25,28]. If the hematoma affects the caudal cerebellum, the medulla is the portion of the brainstem compressed and for this reason vasomotor disturbances and respiratory arrest may develop. Occasionally, patients with cerebellar hemorrhage present with symptoms and signs of hydrocephalus. In deteriorating patients with accessible lesions, surgery should not be delayed. Patients with cerebellar hemorrhage who are deteriorating neurologically or who have brainstem compression and/or hydrocephalus from ventricular obstruction should undergo surgical removal of the hemorrhage as soon as possible [6].

6. Pontine and brainstem hematomas

Hematomas affecting the pons and the brainstem are one of the topographies associated with a more severe clinical outcome [4]. Primary brainstem hemorrhages are located most
often in the pons. Midbrain and medullary hemorrhages are rare. Pontine hematomas (Figure 7) constitute a subgroup of hemorrhagic stroke that accounts for 0.36% of the total number of strokes and 3.4% of ICH, which is similar to 3–6% reported in most studies [4,8,9].

Fig. 7. Axial CT demonstrates a large pontine hemorrhage.

Early cranial nerve dysfunction was the independent clinical factor associated with brainstem ICH. Cranial nerve palsy found in 66.7% of our patients may be explained by involvement of the brainstem tegmentum by the hematoma either primarily or indirectly causing nuclear palsies and conjugate gaze abnormalities [4]. Pontine hematomas are severe, with a high mortality rate (50%). A small percentage of patients are symptom-free at the time of hospital discharge (7.1%) [5]. The 50% in-hospital mortality rate observed in our study [5] is lower than 55–60% reported by others [29,30] but higher than 31–47.5% of other series [31,32].

It should be noted that a pure motor hemiparesis, clinically indistinguishable from a lacunar infarction is an infrequent presenting form of pontine hematoma [11,33]. In this cases, there are two unilateral pontine hematomas localized in the basis pontis or at the union of basis pontis and tegmentum.

7. Multiple topographic involvement

In multiple topographic involvement with large-size hematomas (Figure 8), limb weakness, diabetes, and altered consciousness were independent clinical factors selected in the multivariate analysis. In relation to limb weakness (found in 97% of the cases), persistent hemiplegia is caused by involvement of the pyramidal tract fibers. It is well known that severity of hemiplegia is related to survival [4,9]. The state of alertness of the patient is a clinical feature that correlates with prognosis in ICH and, in general, in acute stroke patients. In the Lausanne Stroke Registry [35], 50% of ICH patients had some reduction in the level of consciousness, which is similar to 45.9% of our series. However, in patients with hemorrhage of multiple topographies, altered consciousness was found in 76.5% of cases. Reduced alertness in ICH is due to either a generalized increase in intracranial pressure, or
to compromise of both hemispheres, or to the reticular activating system bilaterally in the brainstem tegmentum [4].

![Fig. 8. A) and B) Multiple parenchymal hematoma in cerebral amyloid angiopathy](image)

In patients with ICH, diabetes was more frequently associated with multiple parenchymal hematoma (35.3%) [35]. Little is known about the influence of diabetes on the volume of damaged brain tissue in ICH patients. Diabetes is known to produce deleterious effects on the microvasculature that may result in increased bleeding risk. The ICH of multiple topography in patients with diabetes might be related to the specific angiopathy induced by diabetes in small vessels. The vasculopathy of perforating cerebral arteries, the walls of which are weakened by lipid and hyaline material (lipohyalinosis and fibrinoid necrosis), microaneurysms and/or microangiopathy may be a real risk for hematoma of multiple topography in diabetic patients [35]. These changes in cerebral vessels would perhaps make diabetics more prone to develop hemorrhages of large size than nondiabetics. More information, however, is needed on the cerebrovascular pathology whereby diabetes affects large and small blood vessels.

8. Primary intraventricular hemorrhage

Data regarding the frequency of primary intraventricular hemorrhage in the different hospital-based stroke registries are scarce. In our experience, primary intraventricular hemorrhage (Figure 9) is a rare subgroup of haemorrhagic stroke that accounted for 0.31% of all cases of stroke and 3.3% of intracerebral hemorrhages [5]. The clinical syndrome closely mimics subarachnoid hemorrhage, with sudden headache, stiff neck, vomiting and lethargy [36]. In childhood ventricular bleeding usually arises from small subependymal arteriovenous malformations. In adults, most intraventricular hemorrhages are due to ventricular spread of primary hypertensive bleeds into periventricular structures. Primary intraventricular hemorrhage is a severe clinical condition with an in-hospital mortality rate, in the present study, of 41.7%, and only one patient (8.3%) was symptom-free at discharge [5]. In other series, the mortality rate ranged between 33.3% and 43% [36-38].

www.intechopen.com
Fig. 9. Axial CT showing hemorrhage within the 3rd and both lateral ventricles with small layering fluid-fluid levels.

In conclusion, different topographies of ICH have an influence on the clinical spectrum and early outcome of patients with hemorrhagic stroke. Sensory deficit is frequently associated with ICH in the thalamus, lacunar syndrome and hypertension with internal capsule/basal ganglia ICH, seizures and non-sudden stroke onset with lobar ICH, ataxia with hemorrhage in the cerebellum, cranial nerve palsy with brainstem ICH, and limb weakness, diabetes, and altered consciousness with multiple topographic involvement. In-hospital mortality rates are also different according to the site of bleeding, varying from 16% in patients with internal capsule/basal ganglia hematomas, 20% in those with cerebellar hemorrhage and 25% for lobar and thalamic hematomas. Brainstem, primary intraventricular hemorrhage, and multiple topographic involvement are very severe conditions, with in-hospital mortality rates ranging between 40% to 65%. The morbidity and mortality associated with ICH remain high despite recent advances in our understanding of the clinical course of ICH. Rapid recognition and diagnosis of ICH as well as identification of early prognostic indicators are essential for planning the level of care and avoiding acute rapid progression during the first hours. Aggressive treatment of hypertension is essential in the primary and secondary prevention of ICH.

9. Acknowledgements

We thank M. Balcells, MD, for valuable participation in the study and Marta Pulido, MD, for editing the manuscript and editorial assistance. This study was supported in part by a grant from the Fondo de Investigación Sanitaria (FIS PI081514). Instituto de Salud Carlos III, Madrid, Spain.

10. References

[1] Rosenow F, Hojer CH, Meyer-Lohmann CH, Hilgers RD, Mühlhofer H, Kleindienst A, et al. Spontaneous intracerebral hemorrhage. Prognostic factors in 896 cases. Acta Neurol Scand 1997;96:174–182.
Intracerebral Hemorrhage: Influence of Topography of Bleeding on Clinical Spectrum and Early Outcome

291

[2] Lisk DR, Pasteur W, Rhoades H, Putnam RD, Grotta JC. Early presentation of hemispheric intracerebral hemorrhage: prediction of outcome and guidelines for treatment allocation. Neurology 1994;44:133–139.

[3] Juvela S. Risk factors for impaired outcome after spontaneous intracerebral hemorrhage. Arch Neurol 1995;52:1193–1200.

[4] Kase CS, Mohr JP, Caplan LR. Intracerebral hemorrhage. In: Mohr JP, Choi DW, Grotta JC, Weir B, Wolf PhA, eds. Stroke. Pathophysiology, diagnosis, and management. Churchill Livingstone, Philadelphia, 2004, pp 327-376.

[5] Arboix A, Comes E, García-Eroles L, Massons J, Oliveres M, Balcells M, et al. Site of bleeding and early outcome in primary intracerebral hemorrhage. Acta Neurol Scand 2002;105:282–288.

[6] Morgenstern LB, Hemphill JC 3rd, Anderson C, Becker K, Broderick JP, Connolly ES Jr, et al. Guidelines for the Management of Spontaneous Intracerebral Hemorrhage. A Guidelines for Healthcare Professionals from the American Heart Association/American Stroke Association. Stroke 2010;4:2108–2129.

[7] Tatu L, Moulin Th, Mohamad RE, Vuillier F, Rumbach L, Czorny A. Primary intracerebral hemorrhages in the Besançon Stroke Registry. Initial clinical and CT findings, early course and 30-day outcome in 350 patients. Eur Neurol 2000;43:209–214.

[8] Qureshi A, Mendelow AD, Hanley DF. Intracerebral haemorrhage. Lancet 2009;373:1632–1644.

[9] Kase CS. Subcortical hemorrhages. In: Donnan G, Norrving B, Bamford J, Bogousslavsky J, eds. Subcortical stroke (2 edition). Oxford Medical Publications, Great Britain, 2002, pp 347–377.

[10] Arboix A, Martinez-Rebollar M, Oliveres M, García-Eroles L, Massons J, Targa C. Acute isolated capsular stroke: a clinical study of 148 cases. Clin Neurol Neurosurg 2005;107:88–94.

[11] Arboix A, García-Eroles L, Massons J, Oliveres M, Balcells M. Hemorrhagic pure motor stroke. Eur J Neurol 2007;14:219–223.

[12] Miyai I, Suzuki T, Kang J, Volpe BT. Functional outcome in patients with hemorrhagic stroke in putamen and thalamus compared with those with stroke restricted to the putamen or thalamus. Stroke 2000;31:1365–1369.

[13] Kumral E, Kocaer T, Ertuhey NO, Kumral K. Thalamic hemorrhage. A prospective study of 100 patients. Stroke 1995;26:964–970.

[14] Chung CS, Caplan LR, Han W, Pessin MS, Lee KH, Kim JM. Thalamic hemorrhage. Brain 1996;119:1973–1886.

[15] Arboix A, Rodriguez-Aguilar R, Oliveres M, Comes E, García-Eroles L, Massons J. Thalamic haemorrhage vs internal capsule-basal ganglia haemorrhage: clinical profile and predictors of in-hospital mortality. BMC Neurol 2007;7:32, doi: 10.1186/1471-2377-7-32.

[16] Mori S, Sadoshima S, Ibayashi S, Fujishima M. Impact of thalamic hematoma on six-month mortality and motor and cognitive outcome. Stroke 1995;26:620–626.

[17] Shah SA, Kalita J, Misra UK, et al. Prognostic predictors of thalamic hemorrhage. J Clin Neurosci 2005;12:559–561.

[18] Arboix A, Manzano C, García-Eroles L, Massons J, Oliveres M, Parra O, et al. Determinants of early outcome in spontaneous lobar cerebral hemorrhage. Acta Neurol Scand 2006;114:187–192.
[19] Hårdemark HG, Wesslén N, Persson L. Influence of clinical factors, CT findings and early management on outcome in supratentorial intracerebral hemorrhage. Cerebrovasc Dis 1999;9:10–21.

[20] Broderick J, Brott Th, Tomsick Th, Leach A. Lobar hemorrhage in the elderly. The undiminishing importance of hypertension. Stroke 1993;24:49–51.

[21] Brott T, Broderick J, Kothari R, Barsan W, Tomsick T, Sauerbeck L, et al. Early hemorrhage growth in patients with intracerebral hemorrhage. Stroke 1997;28:1–5.

[22] Van der Hoop RG, Vermeulen M, van Gijn J. Cerebellar hemorrhage: diagnosis and treatment. Surg Neurol 1988;29:6–10.

[23] Melamed N, Satya-Murti S. Cerebellar hemorrhage. A review and reappraisal of benign cases. Arch Neurol 1984;41:425–428.

[24] Dunne JW, Chakera T, Kermode. Cerebellar hemorrhage. Diagnosis and treatment: a study of 75 consecutive cases. Q J Med 1987;64:739–754.

[25] González-García J, Gelabert-González M, García-Allut A, Fernández-Villa JM, López-García E, García-Pravos A. Hematomas de cerebro: un ictus con tratamiento quirúrgico. Rev Neurol 2000;31:1119–1126.

[26] Toyoda K, Okada Y, Ibayashi S, Inoue T, Yasumori K, Fukui D, et al. Antithrombotic therapy and predilection for cerebellar hemorrhage. Cerebrovasc Dis 2007;23:109–116.

[27] Turgut M, Özcan OE, Ertürk Ö, Saribas O, Erbengi A. Spontaneous cerebellar strokes. Clinical observations in 60 patients. Angiology 1996;47:841–848.

[28] Arboix A, Rennie M. Clinical study of 28 patients with cerebellar hemorrhage. Med Clin (Barc) 2009;132:665–668.

[29] Wijdicks EFM, Louis ES. Clinical profiles predictive of outcome in pontine hemorrhage. Neurology 1997;49:1342–1346.

[30] Chung CS, Park CH. Primary pontine hemorrhage: a new CT classification. Neurology 1992;42:830–834.

[31] Wessels T, Möller-Hartmann W, Noth J, Klötzsch C. CT findings and clinical features as markers for patient outcome in primary pontine hemorrhage. Am J Neuroradiol 2004;25:257–260.

[32] Murata Y, Yamaguchi S, Kajikawa H, Yamamura K, Sumioka S, Nakamura S. Relationship between the clinical manifestations, computed tomographic findings and the outcome in 80 patients with primary pontine hemorrhage. J Neurol Sci 1999;167:107–111

[33] Arboix A. Clinical study of 14 patients with pontine hemorrhage. Med Clin (Barc) 2008;130:339–341.

[34] Bogousslavski J, Van Melle J, Regli F. The Lausanne Stroke Registry: analysis of 1.000 consecutive patients with first stroke. Stroke 1988;19:1083–1092.

[35] Arboix A, Massons J, Garcia-Eroles L, Oliveres M, Targa C. Diabetes is an independent risk factor for in-hospital mortality from acute spontaneous intracerebral hemorrhage. Diabetes Care 2000;23:1527–1532.

[36] Angelopoulos M, Gupta SR, Hia AB. Primary intraventricular hemorrhage in adults: clinical features, risk factors, and outcome. Surg Neurol 1995;44:433–436.

[37] Passero S, Ulivelli M, Reale F. Primary intraventricular haemorrhage in adults. Acta Neurol Scand 2002;105:115–119.

[38] Verma A, Maheshwari MC, Bhargava S. Spontaneous intraventricular haemorrhage. J Neurol 1987;234:233–236.
Neuroimaging methodologies continue to develop at a remarkable rate, providing ever more sophisticated techniques for investigating brain structure and function. The scope of this book is not to provide a comprehensive overview of methods and applications but to provide a 'snapshot' of current approaches using well established and newly emerging techniques. Taken together, these chapters provide a broad sense of how the limits of what is achievable with neuroimaging methods are being stretched.

How to reference
In order to correctly reference this scholarly work, feel free to copy and paste the following:

Adrià Arboix and Elisenda Grivé (2012). Intracerebral Hemorrhage: Influence of Topography of Bleeding on Clinical Spectrum and Early Outcome, Neuroimaging - Methods, Prof. Peter Bright (Ed.), ISBN: 978-953-51-0097-3, InTech, Available from: http://www.intechopen.com/books/neuroimaging-methods/intracerebral-hemorrhage-influence-of-topography-of-bleeding-on-clinical-spectrum-and-early-outcome
