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Intracerebral Hemorrhage: Influence of Topography of Bleeding on Clinical Spectrum and Early Outcome

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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 accesible to surgical drainage, whereas other areas, such as the thalamus and the brainstem are inaccesible [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 tipically 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 then 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
Frontoparietotemporal	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

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	Intrave-ntricular (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							22.2 *vs.* 0.5‡
Atrial dysrhythmia							44.4 *vs.* 9.5*
Previous cerebral hemorrhage			6.0 *vs.* 1.3§				
Anticoagulant treatment							22.2 *vs.* 0.5§
Suddent 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

Bleeding topography	β	SE (β)	Odds ratio (95% CI)
Thalamus			
Sensory deficit	1.1729	0.4365	3.23 (1.37 to 7.60)
Internal capsule/basal ganglia			
Lacunar syndrome	1.3373	0.5346	3.81 (1.33 to 10.86)
Hypertension	0.8065	0.3855	2.24 (1.05 to 4.77)
Lobar			
Seizures	2.4178	0.8278	11.22 (2.21 to 56.84)
Non-sudden stroke onset	0.8045	0.3344	2.24 (1.16 to 4.31)
Hypertension	–1.0669	0.3091	0.34 (0.19 to 0.63)
Cerebellum			
Ataxia	5.7560	1.1653	316.09 (32.2 to 3102.63)
Sensory deficit	–4.0215	1.5093	0.02 (0.009 to 0.35)
Brainstem			
Cranial nerve palsy	3.7108	0.6393	40.89 (11.67 to 143.15)
Sensory deficit	–4.0215	1.5093	0.02 (0.009 to 0.35)
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)

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

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).

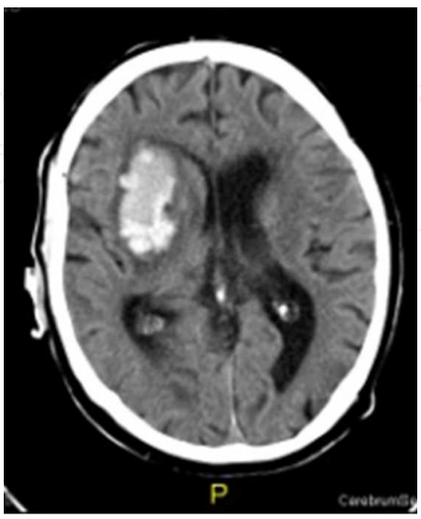
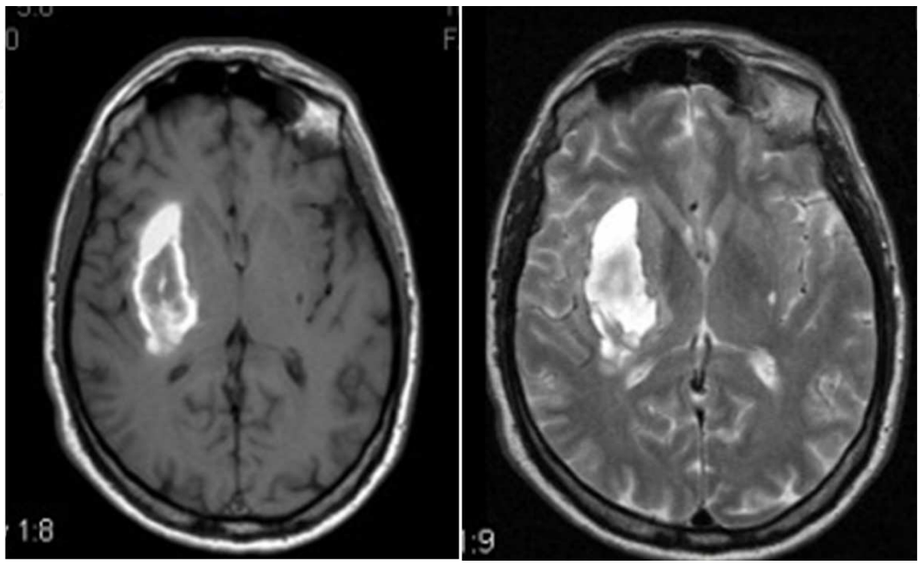
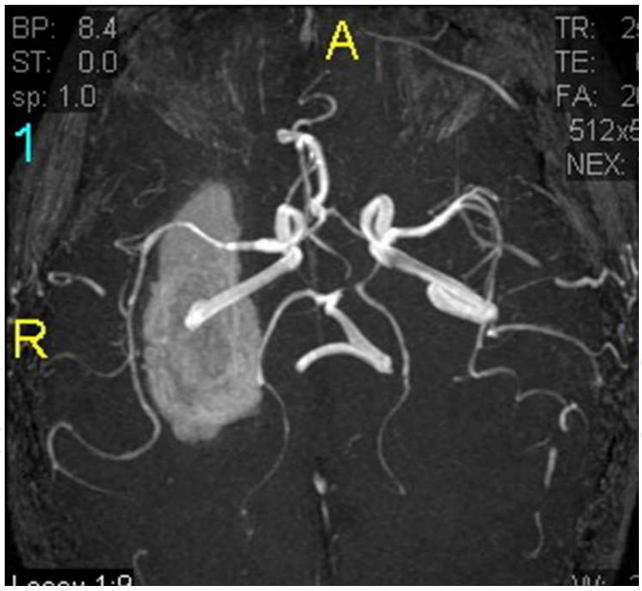


Fig. 1. Axial CT shows acute hypertensive putaminal hematoma with early peripheral edema.

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].



(a) (b)



(c)

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		2 (2.2)	0.236
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			
Suden 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].

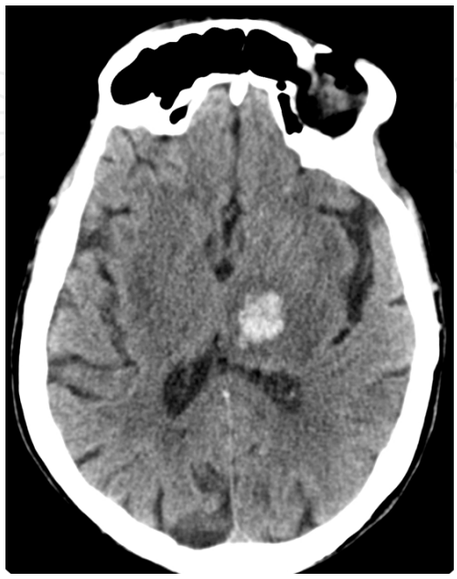


Fig. 3. Axial CT reveals an acute anterior left thalamic hematoma.

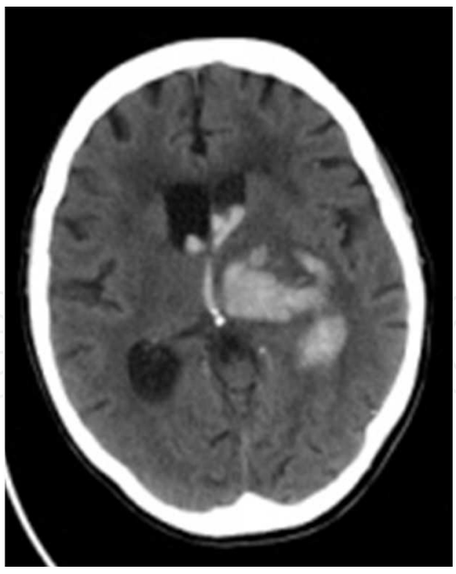


Fig. 4. Axial CT shows a large thalamic hematoma with intraventricular rupture.

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

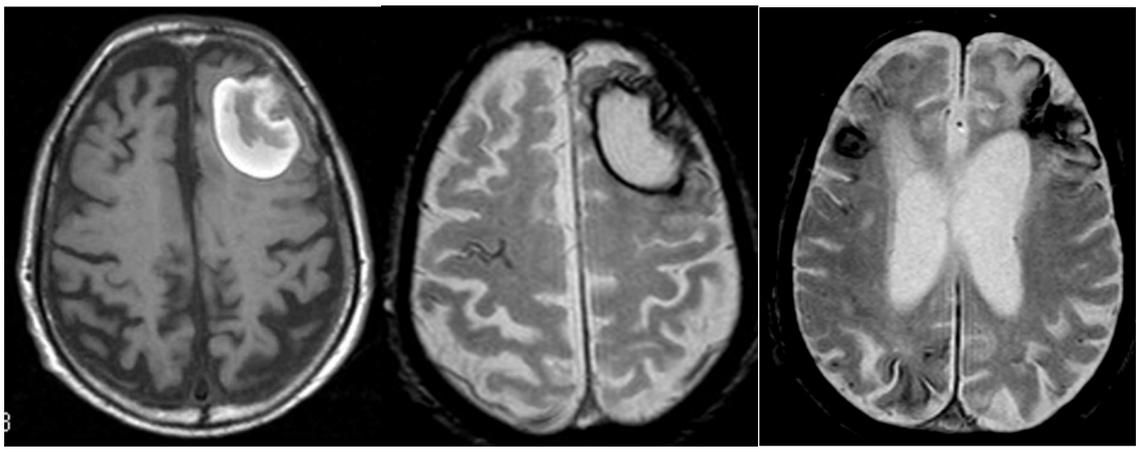
ataxic or have choreic movements. The commonest oculomotor abnormalities include paralysis of upward gaze, often with one or both eyes resting downward, and hyperconvergence of one or both eyes. These ocular abnormalities are due to direct extension of the hematoma to the diencephalic-mesencephalic junction 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].



(a) (b) (c)

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 mutiple 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].

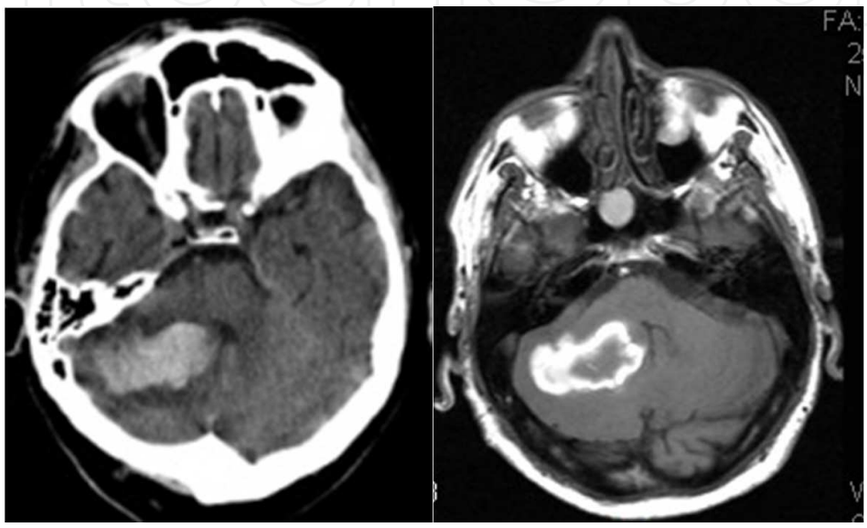
The lower frequency of hypertension in lobar ICH (42.3% *vs.* 69.6% in subcortical ICH) is a relevant clinical aspect that coincides with datas reported in the literature [6], because high blood pressure has the lowest frequency as compared with other topographies of bleeding. However, other causes different 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].

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].



(a) (b)

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 ussually 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. Ocassionally, patients with cerebellar hemorrhage present with symptoms and signs of hydrocephalus. In deteriorating patients with accesible 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].

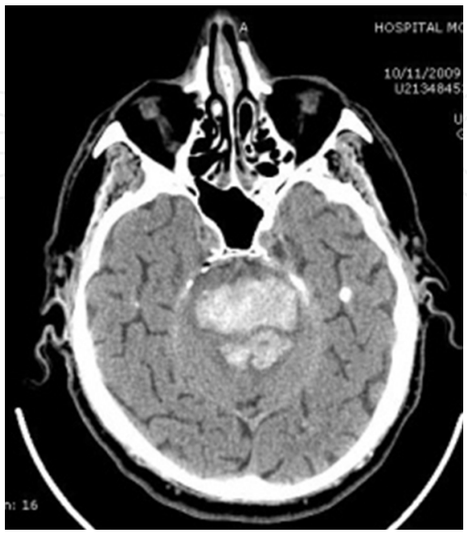


Fig. 7. Axial CT demonstrates a large pontine hemorrhage.

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].

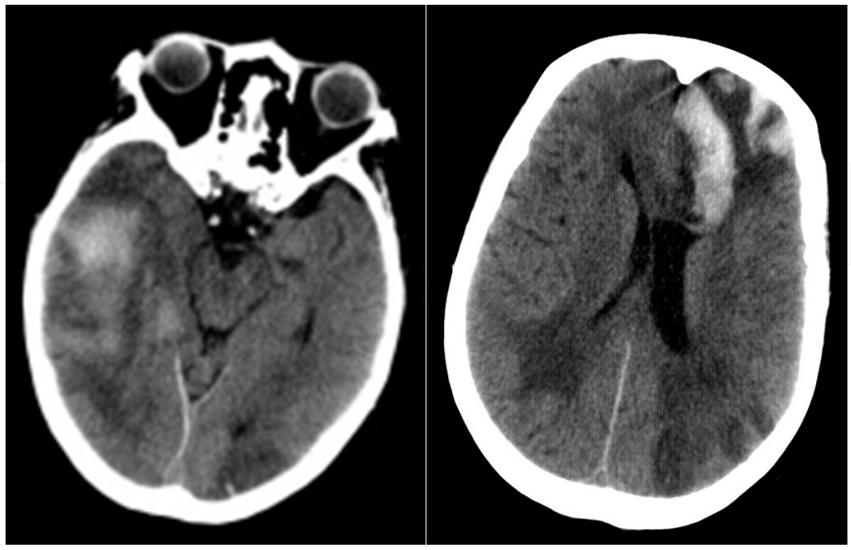
Pontine hematomas are severe, with a high mortality rate (50%). A small percentage of patients are symptom-free at the time of hospital discharge (7.1%) [5]. The 50% in-hospital mortality rate observed in our study [5] is lower than 55–60% reported by others [29,30] but higher than 31–47.5% of other 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].



(a) (b)

Fig. 8. A) and B) Multiple parenchymal hematoma in cerebral amyloid angiopathy

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].

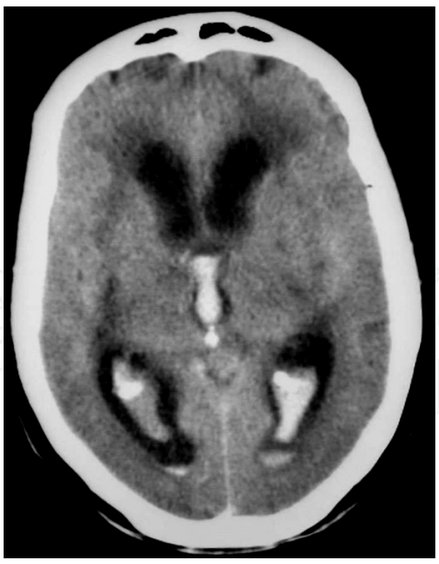


Fig. 9. Axial CT showing hemorrhage within the 3rd and both lateral ventricles with small layering fluid-fluid levels.

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.

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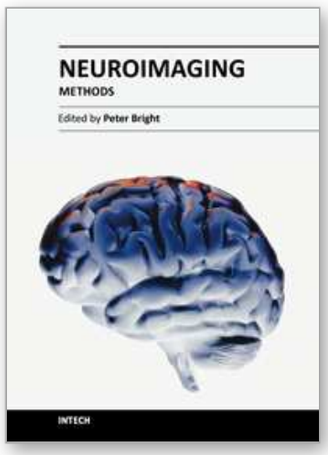

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Neuroimaging methodologies continue to develop at a remarkable rate, providing ever more sophisticated techniques for investigating brain structure and function. The scope of this book is not to provide a comprehensive overview of methods and applications 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.

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