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# Lobar Intracerebral Haematomas: A Review

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**Abstract**

***The occurrence of lobar intracerebral haematomas is mainly linked to the presence of cerebral amyloid angiiopathy. The clinical presentation depends on the extend and the location of the haematoma. Cerebral amyloid angiopathy frequently occurs in patients with Alzheimer disease. Although the latter is the main cause of cognitive decline leading to dementia also isolated cerebral amyloid angiopathy can have influence on cognition. Magnetic resonance imaging is the most valuable neuroimaging technique as it is able not only to locate the lesions, but also to determine the age of the haematomas. Acute surgical evacuation of the haematoma is still the most used treatment.***

***Keywords:*** ***Lobar intracerebral haematoma (LCH); Intracerebral haematoma (ICH); Cerebral amyloid angiopathy (CAA); Alzheimer disease (AD); Magnetic resonance imaging (MRI); Computed tomography (CT).***

## 1. Introduction

Spontaneous cerebral haemorrhages account for 10 to 15% of all strokes and are associated with a high mortality rate. The most common causes are arterial hypertension, cerebral amyloid angiopathy (CAA) and the use of anticoagulant or anti-platelet drugs [1]. Lobar intracerebral haematomas (LCHs) are mainly linked to CAA [2]. They represent one third of all the cerebral haemorrhages [3]. The coexistence of arterial hypertension and drugs acting on blood coagulation increase the risk to induce an LCH [4]. However, LCHs can also occur in brains of elderly patients not displaying CAA [5].

CAA-induced LCHs can be suspected during life only by biopsy examination of the surgically removed haematoma and the adjacent cerebral parenchyma [6], [7]. A definite diagnosis of CAA can, however, only been made by post-mortem examination of the brain [8]. The clinically proposed Boston criteria for CAA have already been validated by a small post-mortem study of patients with LCHs [9].

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Computed tomography (CT) and magnetic resonance imaging (MRI) are the best imaging techniques for evaluation of suspected acute cortical haemorrhages. However, MRI alone is saving time and costs and is even more reliable than CT [10]. MRI is also more suitable for identification of small and chronic lesions and for screening of additional ischemic lesions and cortical micro-bleeds [11].

## 2. Incidence

CAA is not observed in brains during ageing in persons with normal cognition [12]. It is frequently observed in patients with Alzheimer disease (AD) [13]. However, LCHs occur more frequently in "pure" CAA patients without AD symptoms than in those with this neurodegenerative disease [14]. LCHs appear only in the severe forms of CAA in AD brains. Although CAA is also found in 45% of brains with Lewy body disease and in 21% of those with progressive supranuclear palsy no LCHs are observed in these disease entities [15].

## 3. Clinical Aspects

The clinical presentation of an LCH depends on the dimensions of the haematoma and its location [3]. Also, a large part of the symptoms can be due to the additional AD features [16]. In contrast to hypertensive haemorrhages, CAA-related LCHs occur mainly during the night and without elevated blood pressure at onset [17]. They are mainly observed in relatively young patients and less in the old ones [18]. The LCHs in CAA brains predominate in the temporal, parietal and occipital lobes [19], [20], while in the non-CAA brains they tend to be more frequent in the frontal lobes [21]. Cerebellar lobar haematomas are rarely related to CAA [22]. The majority of these types of lesions are mainly due to hypertensive small vessel disease [23], [24].

One of the main questions remains whether CAA and LCHs can be responsible for cognitive decline in the absence of AD features [8]. There is a substantial risk of incident dementia in dementia-free survivors of spontaneous intracerebral haemorrhage (ICH), suggesting that underlying CAA can be a contributing factor for the occurrence of new-onset dementia [25]. Also, CAA is sometimes associated with cognitive decline before the appearance of a symptomatic LCH [26]. Compared to patients with other causes of ICH, those with CAA-associated LCHs have a lower mortality rate [27]. However, the rate of recurrence in CAA is among the highest of all stroke types [28].

## 4. Neuroimaging

MRI is the most efficient neuroimaging technique, using diffusion weighted imaging and apparent diffusion coefficient [29]. The distinctive patterns of the lesions and the absence of an ischaemic stroke are the hallmark features of hyper-acute LCHs [30]. There is a progressive shift of the intensity of the hypo-signal from the haematoma core in the acute stage to the boundaries later on. During the residual stages the MRI hypo-signal mildly decreases in the boundaries with the appearance of superficial siderosis and the collaps of the haematoma core (Fig. 1). Brains with CAA related LCHs are associated to more severe white matter changes and more frequent cortical micro-infarcts and micro-bleeds compared to CAA brains without haemorrhages [21].

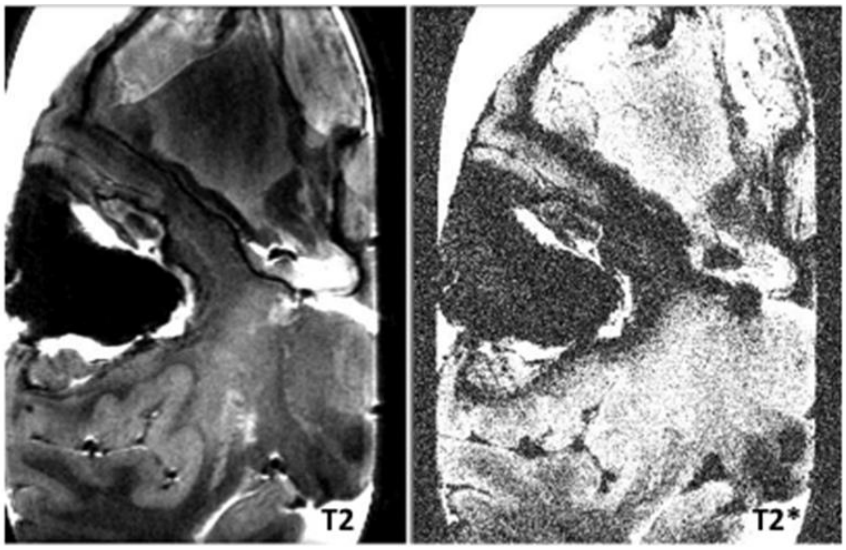


**Fig. 1.** Post-mortem T2 and T2* 7.0-tesla MRI imaging of a coronal section of a cerebral hemisphere with two lobar haematomas: a hyperacute one with a homogenous hypo-intense core and a one month old one with only hypo-intensities in the boundaries.

Five distinct stages of haemorrhage can be defined: hyper-acute with long T1 and T2, acute with long T1 and short T2, early sub-acute with short T1 and long T2 and chronic with short T2 [31]. Apparent diffusion coefficient is decreased in acute haemorrhagic lesions as well as in acute non-haemorrhagic ones (0-3 days). However, mild hypo-intense areas are seen on T2-weighted MR images in haemorrhagic strokes compared to the normal or to the increase of the hypo-intensities in the non-haemorrhagic ones [32]. During the chronic stage the apparent diffusion coefficient remains decreased in the haemorrhagic lesions compared to the increase in the non-haemorrhagic ones, from day 31 on [33].

## 5. Treatment

Today, no specific treatment is able to prevent the bleeding and the eventual cognitive decline [34]. Although definitive evidence favouring surgical intervention is lacking, there is a good theoretical rationale for early surgical intervention [1]. The STICH II trial confirms that early surgery does not increase the rate of death or disability at six months and may have a small but clinically relevant advantage for patients with LCHs without intra-ventricular bleeding [35].

## 6. Discussion

No evidence of involvement by CAA is found in the basal ganglia with and without deep haematomas [36]. One post-mortem study argues that CAA cases with all types of ICHs are less frequent than those without haemorrhages [5]. However, a later article from the same research group demonstrates a significantly higher frequency of LCHs in patients with severe degrees of CAA compared to those with low-grade ones. The higher incidence of LCHs is mainly observed in significantly older patients [37].

Strictly lobar cerebral and cerebellar micro-bleeds are considered as the NMR hallmarks of CAA, whereas their presence in the central gray matter is more frequently found in patients with severe arterial hypertension [38]. Also, the combination of cortical and deep micro-bleeds suggests a hypertensive angiopathy [39]. The incident cortical micro-bleeds in brains with LCHs are associated with haemorrhagic biomarkers. Ischaemic burden, on the other hand, is increased in non-LCH brains [40].

CAA-associated micro-bleeds and macro-bleeds comprise distinct entities. Increased vessel wall thickness may predispose to formation of micro-bleeds relative to LCHs [41]. Deep ischaemic white matter changes in brains with CAA-related LCHs are thought to be relatively rare and probably related to another unknown pathogenetic mechanism [42]. Superficial siderosis on MRI can not only be sequels of LCHs but also of cortical micro-bleeds and be included in the Boston criteria for CAA [43], [44].

## 7. Conclusions

LCHs are mainly related to the presence of CAA. The clinical presentation depends of the extension and the location of haematoma and the eventual association to Alzheimer's disease. Acute surgical evacuation is still the most proposed treatment.

## 8. Disclosure

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