



DR. SILJA RÄTY (Orcid ID : 0000-0002-6921-0597)

DR. HANNE SALLINEN (Orcid ID : 0000-0001-7133-8189)

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OCCIPITAL INTRACEREBRAL HEMORRHAGE – CLINICAL CHARACTERISTICS, OUTCOME, AND POST-ICH EPILEPSY

Running title: Occipital Intracerebral Hemorrhage

Silja Rätty, MD¹; Hanne Sallinen, MD¹; Pekka Virtanen, MD²; Elena Haapaniemi, MD, PhD¹; Teddy Y Wu, FRACP³, PhD; Jukka Putaala, MD¹, PhD; Atte Meretoja, MD, FRACP, PhD¹; Turgut Tatlisumak, MD, PhD⁴; Daniel Strbian, MD, PhD¹

¹HUS Neurocenter, Neurology, University of Helsinki and Helsinki University Hospital, Helsinki, Finland

²HUS Medical Imaging Center, Radiology, University of Helsinki and Helsinki University Hospital, Helsinki, Finland

³New Zealand Brain Research Institute and Department of Neurology, Christchurch Hospital, Christchurch, New Zealand

⁴Department of Clinical Neurosciences/Neurology, Institute of Neurosciences and Physiology, Sahlgrenska Academy at University of Gothenburg and Department of Neurology, Sahlgrenska University Hospital, Gothenburg, Sweden

Correspondence: Silja Rätty, HUS Neurocenter, Neurology, Helsinki University Hospital,

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Haartmaninkatu 4, 00290, Helsinki, Finland. E-mail: silja.raty@hus.fi.

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Conflict of interest

SR, HS, PV, TW, EH, AM, and DS have no disclosures. TT has received advisory board honoraria from Boehringer Ingelheim, Bayer, Lumosa Pharm, BMS, and Portola Pharm and has worked as a national principle investigator for trials sponsored by Bayer and BMS. JP has received advisory board honoraria from Boehringer-Ingelheim, Bayer, Portola, BMS-Pfizer, and Abbott/St. Jude Medical, speaker's honoraria from Boehringer-Ingelheim, Bayer, BMS-Pfizer, and editor's honorary from Terve Media, grants from BMS-Pfizer, Abbott/St. Jude Medical, and Business Finland, and Amgen, has worked in research collaboration with Vital Signum, Nokia Technologies, Bittium, and BcB Medical, and is a stock owner of Vital Signum. He has participated in the European Stroke Organisation's guideline working groups on Post-stroke hyperglycemia and Secondary prevention in patients with AF, as well as in the Finnish Duodecim Society's guideline working group on Ischemic stroke and TIA.

Data availability statement

The data supporting the findings of the study are available from the corresponding author upon reasonable request.

ABSTRACT

Objectives: Posterior location affects the clinical presentation and outcome of ischemic stroke, but little is known about occipital intracerebral hemorrhage (ICH). We studied non-traumatic occipital ICH phenotype, outcome, and post-ICH epilepsy.

Materials and methods: Occipital ICH patients were retrospectively identified from the Helsinki ICH Study registry of 1013 consecutive ICH patients treated in our tertiary center in 2005–2010. They were compared to non-occipital ICH patients to evaluate the effect of location on functional outcome at discharge (dichotomized modified Rankin Scale, mRS), 3- and 12-month mortality, and incidence of epilepsy.

Results: We found 19 occipital ICH patients (5.3% of lobar and 1.9% of all ICH). Compared to non-occipital lobar ICHs, they were younger (median age 63 vs 71 years, $P = .007$) and had lower National Institutes of Health Stroke Scale on admission (1 vs 8, $P < .001$), smaller hematoma volume (6.3 vs 17.7 ML, $P = .008$), and more frequently structural etiology underlying the ICH (26% vs 7%, $P = .01$). Mortality at both 3 and 12 months was 6%, whereas 84% reached favorable outcome (mRS 0–2) at discharge. Occipital location was associated with favorable outcome at discharge in lobar ICH (OR 11.02, 95% CI 1.55–78.20). Incidence of post-ICH epilepsy (median follow-up 2.7 years) was 18%, equaling to that of non-occipital lobar ICH.

Conclusions: Occipital ICH patients are younger, have less severe clinical presentation, smaller hematoma volume, more often structural etiology, and better outcome than other ICH patients. They exhibit a similar risk of epilepsy as non-occipital ICHs.

Keywords: Cerebral hemorrhage, Epilepsy, Occipital lobe, Outcome, Visual fields

INTRODUCTION

The incidence of intracerebral hemorrhage (ICH) is approximately 25/100 000/y¹, contributing to a cumulative lifetime risk of 8.2%². Although the acute and 1-year mortality of ICH reaches 40% and 50%, respectively^{1,3}, lobar location of bleeding seems to accompany a better prognosis⁴. Other factors associated with outcome include hematoma volume, severity, age, infratentorial origin, presence of intraventricular hemorrhage (IVH), and etiology⁵⁻⁸.

In ischemic stroke, the location of the lesion impacts the severity⁹, outcome⁹, and risk of post-stroke epilepsy¹⁰. However, similar topological data on hemorrhage are scarce. In one study, isolated occipital hemorrhages were rare (4.6% of lobar ICH) and had better outcome than other lobar hemorrhages¹¹, whereas another study did not find any occipital areas associating with lower acute mortality¹².

This retrospective, observational, registry-based study identifies occipital hemorrhages among consecutive spontaneous ICH patients treated in a single center. Our goal was to investigate the effect of occipital location on short-term functional outcome, short- and long-term mortality, and incidence of post-ICH epilepsy. Additionally, we characterized the clinical features, recovery, and vision-related disability.

MATERIALS AND METHODS

The study population is derived from the retrospective Helsinki ICH Study (HICHS) registry, consisting of 1013 consecutive ICH patients treated in Helsinki University Hospital between January 2005 and March 2010, described in more detail elsewhere^{7,13}. The study has been approved by the Helsinki University Hospital institutional review board as an observational registry study with no study-related patient contact. Therefore, no patient consent was required.

The hemorrhage location was evaluated from the primary computed tomography or magnetic resonance imaging scan, and all affected anatomical structures were labelled as ‘supratentorial lobar’, ‘supratentorial deep’, ‘infratentorial’, or ‘IVH’. ICH extending to both cortical and deep supratentorial structures were categorized according to the assumed origin by a neuroradiologist. If the origin was unidentifiable, the hematoma was ‘mixed’. Multifocal hemorrhages were classified as ‘lobar’ if each hematoma met the criteria. The neuroradiologist further defined the occipital location based on established anatomical landmarks. Exclusively occipital hemorrhages were classified as ‘occipital’; ICHs with a parietal or temporal extension were ‘non-occipital’. ICH volumes were measured by semi-automatic planimetry using Analyze 12.0 software^{14,15}.

The HICHS registry combines data retrieved from province-wide electronic medical records and mortality data from Statistics Finland⁸. All patients had been evaluated by a neurologist on admission. Glasgow Coma Scale (GCS) and National Institutes of Health Stroke Scale (NIHSS) were either recorded or constructed from medical notes. The etiology of ICH was categorized according to SMASH-U classification⁸. Modified Rankin Scale (mRS) at discharge and mortality at 3 and 12 months were determined. Seizures occurring within the first week after ICH onset were classified as acute and those occurring later as epileptic late seizures^{7,13}. Patients with pre-ICH epilepsy were excluded from the analysis of post-ICH epilepsy incidence.

In addition to the registry data, we reviewed medical records of occipital ICH patients for details on visual symptoms, recovery, and vision-related disability, including habitation, return to work, and ability to drive a car. Based on these data, we defined the patients’ outcome as favorable (mRS 0–2) or unfavorable (mRS 3–6) at 3 and 12 months. Data were collected from the neurological, neurosurgical, and ophthalmological outpatient visits, from physiotherapists’, occupational therapists’, and neuropsychologists’ evaluations, and from contacts to other

specialties and to primary care. Initial visual field defect (VFD) was assessed by confrontation perimetry on admission and residual defect either by standard automated perimetry (SAP) after the acute phase or confrontation perimetry at discharge or at follow-up visits.

Statistical analyses

We tested normality of the data with Shapiro-Wilk and Kolmogorov-Smirnov tests, and due to non-normal distribution of the continuous variables, reported their median and interquartile range. We used Pearson X^2 or Fisher's exact test to compare dichotomous variables and Mann-Whitney U test for continuous variables. To assess the independent association of occipital location on functional outcome and mortality, we ran a multivariable binary logistic regression analysis. Other covariates were selected based on previous prognostic data⁵⁻⁸. All covariates were available for all patients, except for 13 (3.6%) patients without either mortality or planimetric data who were excluded from the multivariable analysis. Statistical significance was set at 0.05 (two-sided). We performed all analyses using SPSS 25 (IBM Corp, Armonk, NY).

RESULTS

Clinical characteristics

Our search yielded 19 patients with occipital hemorrhages, accounting for 5.3% of lobar and 1.9% of all ICH patients. Median age was 63, and 68% were men. The patients with occipital ICH were younger than the other ICH patients (lobar 71 years, $P = .007$; all 68 years, $P = .04$) but did not differ in other baseline characteristics (Table 1).

The admission NIHSS of the occipital ICH patients was lower and the GCS higher compared to the other lobar (median NIHSS 1 vs 8, $P < .001$; median GCS 15 vs 14, $P < .001$) and all ICH patients (NIHSS 11, $P < .001$; GCS 14, $P < .001$) (Table 1). Up to 74% presented with visual symptoms, including 63% who had no other focal deficits. VFD was observed in 79% of the patients. During the diagnostic evaluation, 42% of the occipital ICH patients underwent magnetic resonance imaging and 58% underwent angiography: magnetic resonance angiography (11%), computed tomography angiography (47%), or digital subtraction angiography (5%). The selection of imaging modality was based on the clinician's judgement with an input from a neuroradiologist or neurosurgeon when needed. Median volume was 6.3 ML in occipital ICH and 17.7 ML in other lobar hemorrhages ($P = .008$). Repeat imaging (median time interval 2 d, IQR 1–28 d) was performed in 84% of the patients, revealing no hematoma growth (median -18%).

The most common etiology of occipital ICH was amyloid angiopathy (53%), followed by structural lesions (26%), three of which were arteriovenous malformations (AVM) (Table 2).

Structural lesions were more prevalent in occipital ICH than in non-occipital lobar ICH (7%, $P = .01$).

Short-term functional outcome and 3- and 12-month mortality

Only one (5%) patient with occipital ICH died during the acute hospital stay, which was due to severe bleeding diathesis and herniation. The median mRS score of the occipital ICH patients at discharge was 2, and 84% of them achieved mRS 0–2. In comparison, the patients with non-occipital lobar ICH (median mRS 4, mRS 0–2 in 24%) and all non-occipital ICH (median mRS 5, mRS 0–2 in 17%) had much worse outcomes ($P < .001$) (Table 3).

One of the occipital and 28 of non-occipital ICH patients were lost to follow-up after discharge due to out-of-province residence. Of the 17 remaining occipital ICH patients, no one died within a year, accounting for a mortality rate of 6% at 3 and 12 months. This differed significantly from the non-occipital lobar ICH patients at 12 months (34%, $P = .01$) and from all non-occipital ICH patients at 3 and 12 months (33% and 37%, $P = .02$ and $P = .006$) (Table 3). When adjusted for strong predictors (age, sex, NIHSS on admission, ICH volume, presence of IVH, and structural etiology), occipital location remained independently associated with favorable outcome (mRS 0–2) at discharge (OR 11.02, 95% CI 1.55–78.20) (Table 4). However, it did not predict mortality at 3 (OR 2.70, 95% CI 0.29–24.8) or 12 months (OR 0.97, 95% CI 0.11–8.33).

Vision-related disability

Eight occipital ICH patients suffered from residual visual impairments at follow-up visits (Table 5). Six patients underwent a single and four patients repeated SAPs within 1–7 months: studies revealed six stroke-related VFDs, only two of which were a quadrant or larger in the final perimetry. Out of seven patients not examined with SAP, six displayed normal confrontation perimetry at discharge or at follow-up visits. At one year, 16 patients lived at home and one had moved to a nursing home. Nine patients regained their driving license, whereas two received a permanent driving ban. Out of six patients working before ICH, five returned to work (Table 5). Altogether, 14 occipital ICH patients achieved favorable outcome at 3 and 12 months. Functional outcome was not attainable for two patients at the former and for three patients at the latter time point.

Seizures and epilepsy

Two (11%) of the occipital ICH patients experienced early seizures, compared to 20% of the non-occipital lobar ($P = .55$) and 11% of the non-occipital ICH patients ($P > .99$). Post-ICH epilepsy (median follow-up 2.7 years) affected 18% of alive patients, with equal occurrence among the non-occipital lobar ICH patients (16%, $P = .74$). Epilepsy was less frequent among all non-occipital ICH patients (9%), but the difference was non-significant ($P = .19$).

DISCUSSION

In this retrospective study, the prevalence of occipital ICH was low (5.3% of lobar and 1.9% of all ICH). Compared to non-occipital lobar ICHs, the hematomas were smaller, more frequently of structural etiology, and afflicted younger patients with often sole visual symptoms. The occipital ICH patients had better functional outcome, but the occurrence of post-ICH epilepsy did not differ between the occipital and non-occipital lobar ICH patients.

The prevalence and volume of occipital ICH in our study are equal to the findings of Gerner *et al.* who discovered occipital location to be the rarest, even when adjusted for the size of the lobes¹¹. The infrequency is even more pronounced considering that the occipital lobe is the preferred location of amyloid angiopathy¹⁶. Moreover, they reported that the occipital lobe harbored the smallest hematoma with the least growth¹¹, which was hypothesized to stem from a higher gray/white-matter (GW) ratio¹⁷ and lower arterial pressure gradient¹⁸. Other possible reasons for the lack of hematoma growth in our study include the lower baseline volume and blood glucose, as well as the later arrival and low frequency of imaging within the first 24 hours¹⁹. The latter two probably reflect the frequency of patients with isolated visual symptoms and their long prehospital delay, previously reported in occipital ischemia²⁰.

Structural etiology was more prevalent among the occipital than non-occipital ICH patients, potentially contributing to the younger age and better outcome²¹. AVM-associated hemorrhage has lower in-hospital mortality and more favorable outcome at discharge than an alternative etiology²¹. In a cohort of 343 AVM patients, the occipital lobe was only the third most frequent lobar location with an occurrence of 20%²², which is in line with our cohort where 19% of the lobar AVM-related ICHs were occipital. Thus, the relative frequency of structural etiology in occipital ICH seems to reflect the smaller absolute number of other etiologies, not occipital predisposition of AVM. It may also stem from the more extensive imaging workup compared to the other ICH patients.

Previous studies have reported improvement in 48 to 67% and full recovery in 9 to 39% of stroke-related VFD²³⁻²⁵. In our study, 9 out of 15 patients with initial VFD experienced either partial or complete recovery based on normal or improved SAP or later normal confrontation perimetry. Although confrontation perimetry cannot exclude VFD²⁶, our results are in line with the previous

data.

The outcome of the occipital ICH patients was distinctly more encouraging than that among the other ICH patients and in previous studies that report mRS 0–2 in 33–42% of ICH survivors at 6 months and in 17–25% at 12 months³. Our results agree with Gerner *et al.* who found occipital location to predict independently favorable outcome at 3 months, with over 80% achieving mRS 0–3¹¹. Studies on posterior cerebral artery infarcts report almost identical outcomes: 6-month mRS 0–3 in 83%²⁷ and short- and long-term mortalities of 0–8%^{27,28} and 4–10%^{27,29}, respectively.

Why is occipital location associated with a better prognosis? First, mRS emphasizes mobility over cognitive and social function³⁰, potentially leading to underestimation of vision-related impairments. Nevertheless, it does not explain differences in mortality or the high rate of independence of occipital ICH patients. The smaller hematoma volume and growth are probably major contributors^{5,6,8}, even though the better short-term outcome also appear in the adjusted results. However, we considered only baseline volume as a confounder in our analyses due to the inconsistent rate and timing of the control imaging. Thus, we cannot rule out the effect of hematoma growth on the better outcome of the occipital ICH patients.

Cortical location was previously shown to elevate the risk of post-ICH epilepsy in this cohort¹³, but the data on the relationship between the affected lobe and incidence of epilepsy are scarce. A study of 68 patients found no association between occipital location and seizures³¹. In our study, late seizures were equally prevalent among the occipital and non-occipital lobar ICH patients. The possible explanations are contradictory: the occipital ICH patients were younger but had smaller hematoma volume — the former associated with a lower and the latter with a higher risk of epilepsy^{13,32,33}. The greater GW ratio in the occipital and temporal lobe¹⁷ could be a predisposing factor for seizures. Furthermore, focal occipital seizures may sometimes remain unrecognized or be misdiagnosed as migraine.

Strengths and limitations

We studied occipital ICH in a consecutive, well-characterized cohort. The main limitations of the study are the retrospective methodology, small sample size collected in one center, and missing functional outcome of the non-occipital ICH patients at 3 and 12 months. Moreover, due to the retrospective design, the imaging work-up of ICH varied, as well as the diagnostics of VFD. Other

visual functions were not assessable, unless they were addressed in neuropsychological tests or reported by patients. Finally, we defined hematoma location qualitatively, in contrast to studies that have used quantitative voxel-wise methods¹².

In conclusion, our study showed that occipital ICH patients have good prognosis, and most of them return to independent daily activities. This appears to be attributed to occipital location, although we were only able to show this in short-term functional outcome. Further multicenter studies with larger sample sizes are required to confirm our findings.

REFERENCES

1. van Asch CJ, Luitse MJ, Rinkel GJ, van der Tweel I, Algra A, Klijn CJ. Incidence, case fatality, and functional outcome of intracerebral haemorrhage over time, according to age, sex, and ethnic origin: A systematic review and meta-analysis. *Lancet Neurology* 2010;9(2):167-176.
2. Feigin VL, Nguyen G, Cercy K, et al. Global, regional, and country-specific lifetime risks of stroke, 1990 and 2016. *N Engl J Med* 2018;379(25):2429-2437.
3. Poon MTC, Fonville AF, Al-Shahi Salman R. Long-term prognosis after intracerebral haemorrhage: Systematic review and meta-analysis. *J Neurol Neurosurg Psychiatry* 2014;85(6):660-667.
4. Sreerishnan A, Dearborn JL, Greer DM, et al. Intracerebral hemorrhage location and functional outcomes of patients: A systematic literature review and meta-analysis. *Neurocrit Care* 2016;25(3):384-391.
5. Hemphill JC, Bonovich DC, Besmertis L, Manley GT, Johnston SC. The ICH score: A simple, reliable grading scale for intracerebral hemorrhage. *Stroke* 2001;32(4):891-897.
6. Sembill JA, Gerner ST, Volbers B, et al. Severity assessment in maximally treated ICH patients: The max-ICH score. *Neurology* 2017;89(5):423-431.
7. Meretoja A, Strbian D, Putaala J, et al. SMASH-U: A proposal for etiologic classification of intracerebral hemorrhage. *Stroke* 2012;43(10):2592-2597.
8. Rost NS, Smith EE, Chang Y, et al. Prediction of functional outcome in patients with primary intracerebral hemorrhage: The FUNC score. *Stroke* 2008;39(8):2304-2309.
9. Wu O, Cloonan L, Mocking SJT, et al. Role of acute lesion topography in initial ischemic stroke severity and long-term functional outcomes. *Stroke* 2015;46(9):2438-2444.
10. Heuts-van Raak L, Lodder J, Kessels F. Late seizures following a first symptomatic brain infarct are related to large infarcts involving the posterior area around the lateral sulcus. *Seizure* 1996;5(3):185-194.
11. Gerner ST, Kuramatsu JB, Moeller S, et al. Specific lobar affection reveals a rostrocaudal gradient in functional outcome in spontaneous intracerebral hemorrhage. *Stroke* 2017;48(3):587-595.
12. Lee J, King C, Stradling D, et al. Influence of hematoma location on acute mortality after intracerebral hemorrhage. *J Neuroimaging* 2014;24(2):131-136.
13. Haapaniemi E, Strbian D, Rossi C, et al. The CAVE score for predicting late seizures after

intracerebral hemorrhage. *Stroke* 2014;45(7):1971-1976.

14. Wu TY, Sobowale O, Hurford R, et al. Software output from semi-automated planimetry can underestimate intracerebral haemorrhage and peri-haematoma oedema volumes by up to 41.

Neuroradiology 2016;58(9):867-876.

15. Wu TY, Yassi N, Shah DG, et al. Simultaneous multiple intracerebral hemorrhages (SMICH).

Stroke 2017;48(3):581-586.

16. Vinters HV, Gilbert JJ. Cerebral amyloid angiopathy: Incidence and complications in the aging brain. II. The distribution of amyloid vascular changes. *Stroke* 1983;14(6):924-928.

17. Allen JS, Damasio H, Grabowski TJ, Bruss J, Zhang W. Sexual dimorphism and asymmetries in the gray-white composition of the human cerebrum. *Neuroimage* 2003;18(4):880-894.

18. Martin PJ, Evans DH, Naylor AR. Transcranial color-coded sonography of the basal cerebral circulation. Reference data from 115 volunteers. *Stroke* 1994;25(2):390-396.

19. Broderick JP, Diringer MN, Hill MD, et al. Determinants of intracerebral hemorrhage growth: An exploratory analysis. *Stroke* 2007;38(3):1072-1075.

20. Rätty S, Silvennoinen K, Tatlisumak T. Prehospital pathways of occipital stroke patients with mainly visual symptoms. *Acta Neurol Scand.* 2018;137(1):51-58.

21. Murthy SB, Merkler AE, Omran SS, et al. Outcomes after intracerebral hemorrhage from arteriovenous malformations. *Neurology* 2017;88(20):1882-1888.

22. Crawford PM, West CR, Chadwick DW, Shaw MD. Arteriovenous malformations of the brain: Natural history in unoperated patients. *J Neurol Neurosurg Psychiatry.* 1986;49(1):1-10.

23. Gray CS, French JM, Bates D, Cartlidge NE, Venables GS, James OF. Recovery of visual fields in acute stroke: Homonymous hemianopia associated with adverse prognosis. *Age & Ageing* 1989;18(6):419-421.

24. Tiel K, Kolmel HW. Patterns of recovery from homonymous hemianopia subsequent to infarction in the distribution of the posterior cerebral artery. *Neuro-Ophthalmology* 1991;11(1):33-39.

25. Zhang X, Kedar S, Lynn MJ, Newman NJ, Biouesse V. Natural history of homonymous hemianopia. *Neurology* 2006;66(6):901-905.

26. Pandit RJ, Gales K, Griffiths PG. Effectiveness of testing visual fields by confrontation. *Lancet* 2001;358(9290):1339-1340.

27. Ntaios G, Spengos K, Vemou AM, et al. Long-term outcome in posterior cerebral artery stroke. *Eur J Neurol* 2011;18(8):1074-1080.

28. Cals N, Devuyst G, Afsar N, Karapanayiotides T, Bogousslavsky J. Pure superficial posterior cerebral artery territory infarction in The Lausanne Stroke Registry. *J Neurol* 2002;249(7):855-861.
29. Kumral E, Bayulkem G, Ataç C, Alper Y. Spectrum of superficial posterior cerebral artery territory infarcts. *Eur J Neurol* 2004;11(4):237-246.
30. de Haan R, Limburg M, Bossuyt P, van der Meulen J, Aaronson N. The clinical meaning of Rankin 'handicap' grades after stroke. *Stroke* 1995;26(11):2027-2030.
31. De Reuck J, Hemelsoet D, Van Maele G. Seizures and epilepsy in patients with a spontaneous intracerebral haematoma. *Clin Neurol Neurosurg* 2007;109(6):501-504.
32. Merkler AE, Gialdini G, Lerario MP, et al. Population-based assessment of the long-term risk of seizures in survivors of stroke. *Stroke* 2018;49(6):1319-1324.
33. Yang T, Lin W, Chang W, et al. Predictors and outcome of seizures after spontaneous intracerebral hemorrhage. Clinical article. *J Neurosurg* 2009;111(1):87-93.

Table 1. Clinical variables of ICH patients.

	Occipital ICH (n=19)	Non-occipital lobar ICH (n=337)	P- value†	Non-occipital ICH (n=994)	P- value‡	Missing data (occipital/non- occipital lobar/non- occipital)
Demographics						
Age, y	63 (55–69)	71 (60–79)	.007	68 (58–78)	.04	0/0/0
Men	13 (68)	180 (53)	.20	569 (57)	.33	0/0/0
Hypertension	10 (53)	186 (55)	.83	627 (63)	.35	0/0/0
Diabetes	3 (16)	44 (13)	.73	139 (14)	.74	0/0/0
Coronary heart disease	3 (16)	57 (17)	>.99	125 (13)	.73	0/6/12
Atrial fibrillation	4 (21)	47 (14)	.50	139 (14)	.34	0/6/14
Dyslipidemia	7 (37)	61 (18)	.69	190 (19)	.08	0/6/13
Previous ICH	1 (5)	26 (8)	>.99	53 (5)	>.99	0/8/18
Pre-ICH mRS	0 (0–0)	0 (0–0)	.39	0 (0–0)	.43	0/0/0
Prehospital route						
Presentation delay, d	1 (0–2)	0 (0–1)	.006	0 (0–1)	<.001	0/0/0
Use of EMS	7 (37)	274 (83)	<.001	859 (88)	<.001	0/6/20
Clinical variables on arrival						
NIHSS score	1 (1–3)	8 (3–18)	<.001	11 (4–20)	<.001	0/0/0
GCS score	15 (15–15)	14 (10–15)	<.001	14 (10–15)	<.001	0/0/0
Systolic blood pressure, mmHg	165 (141–176)	165 (144–187)	.37	171 (149–193)	.11	0/9/25
Diastolic blood pressure, mmHg	96 (89–100)	87 (74–102)	.07	90 (77–103)	.23	0/11/29

Glucose, mmol/l	5.9 (5.4–6.9)	7.5 (6.3–9.2)	.003	7.3 (6.2–9.1)	.005	2/32/95
Hemoglobin, g/l	147 (132–157)	137 (127–148)	.06	139 (128–150)	.11	1/16/41
Platelet count, E9/l	206 (171–249)	204 (164–253)	.90	209 (171–253)	.95	1/17/47
Radiological variables						
Imaging within 24 h of onset	9 (47)	233 (69)	.048	757 (76)	.01	0/0/0
Follow-up imaging	16 (84)	222 (66)	.10	615 (62)	.047	0/0/0
MRI	8 (42)	72 (21)	.047	144 (15)	.004	0/0/0
Any angiography	11 (58)	111 (33)	.03	246 (25)	.002	0/0/0
CTA	9 (47)	103 (31)	.13	228 (23)	.02	0/0/0
MRA	2 (11)	8 (2)	.09	18 (2)	.05	0/0/0
DSA	1 (5)	5 (2)	.28	8 (1)	.16	0/0/0
IVH	3 (16)	92 (27)	.27	409 (41)	.03	0/0/0
ICH volume at baseline, ML	6.3 (4.1–11.7)	17.7 (5.4–39.5)	.008	9.9 (3.7–28.0)	.21	0/4/19
ICH growth to second imaging, %	-18 (-89–0.6)	0 (-50–41)	.08	0 (-31–46)	.03	5/136/424
Hospital stay						
Deterioration within 72 h	4 (21)	136 (40)	.09	415 (42)	.07	0/1/3
ICH evacuation	1 (5)	34 (10)	.71	60 (6)	>.99	0/0/2
Length of stay, d	7 (3–14)	8 (3–14)	.35	8 (3–14)	.36	0/0/0

N (%) and median (IQR) are reported. Fisher's exact test, Pearson X^2 test, or Mann–Whitney U test were used. mRS, modified Rankin Scale; EMS, emergency medical service; NIHSS, National Institutes of Health Stroke Scale; GCS, Glasgow Coma Scale; MRI, magnetic resonance imaging; CTA, computed tomography angiography; MRA, magnetic resonance angiography; DSA, digital subtraction angiography; IVH, intraventricular hemorrhage. †*P*-value for occipital ICH vs non-occipital lobar ICH; ‡*P*-value for occipital ICH vs all non-occipital ICH

Table 2. Etiology of ICH according to SMASH-U classification.

SMASH-U classification	Occipital ICH	Non-occipital lobar	<i>P</i> -value†	Non-occipital ICH	<i>P</i> -value‡
	(<i>n</i> =19)	ICH (<i>n</i> =337)		(<i>n</i> =994)	
Structural lesion	5 (26)	22 (7)	.01	46 (5)	.002
Arteriovenous malformation	3 (16)	13 (4)	.046	16 (2)	.004
Anticoagulation	2 (11)	52 (15)	.75	140 (14)	.74
Amyloid angiopathy	10 (53)	193 (57)	.69	197 (20)	.002
Hypertension	0	0	.	350 (35)	.001
Systemic/Other disease	1 (5)	25 (7)	>.99	48 (5)	.61
Undetermined	1 (5)	45 (13)	.49	213 (21)	.15

N (%) are reported. †*P*-value for occipital ICH vs non-occipital lobar ICH; ‡*P*-value for occipital ICH vs all non-occipital ICH

Table 3. Outcome of occipital, non-occipital lobar, and all non-occipital ICH.

	Occipital ICH (n=19)	Non-occipital lobar ICH (n=337)	P-value†	Non-occipital ICH (n=994)	P-value‡	Missing data (occipital/non- occipital lobar/non- occipital)
In-hospital mortality	1 (5)	70 (21)	.14	243 (24)	.06	0/0/0
mRS at discharge	2 (1–2)	4 (3–5)	<.001	5 (3–5)	<.001	0/0/0
mRS 0–2 at discharge	16 (84)	81 (24)	<.001	173 (17)	<.001	0/0/0
Mortality at 3 months§	1 (6)	90 (27)	.05	316 (33)	.02	1/8/28
Mortality at 12 months§	1 (6)	111 (34)	.01	357 (37)	.006	1/8/28

N (%) and median (IQR) are reported. †*P*-value for occipital ICH vs non-occipital lobar ICH; ‡*P*-value for occipital ICH vs all non-occipital ICH; §Patients lost to follow-up were excluded from the analysis

Table 4. Multivariable binary logistic regression analysis for outcome of lobar ICH.

Covariates	Favorable outcome (mRS 0–2) at discharge†			3-mo mortality‡			12-mo mortality‡		
	OR (95% CI)	P-value	Wald	OR (95% CI)	P-value	Wald	OR (95% CI)	P-value	Wald
Occipital location	11.02 (1.55–78.20)	.02	5.8	2.70 (0.29–24.8)	.38	0.8	0.97 (0.11–8.33)	.98	0.0
Age per year	0.93 (0.90–0.96)	<.001	16.9	1.06 (1.03–1.10)	.001	11.0	1.07 (1.04–1.10)	<.001	18.2
NIHSS on admission per 1 point	0.80 (0.72–0.88)	<.001	21.3	1.14 (1.09–1.19)	<.001	34.0	1.11 (1.07–1.15)	<.001	28.1
Baseline volume per ml ³	0.86 (0.81–0.92)	<.001	21.7	1.04 (1.02–1.05)	<.001	18.0	1.03 (1.01–1.04)	<.001	12.2
IVH	0.18 (0.03–1.04)	.06	3.7	1.33 (0.60–2.93)	.49	0.5	1.39 (0.68–2.84)	.36	0.8
Male sex	1.50 (0.71–3.17)	.29	1.1	3.11 (1.41–6.87)	.005	7.9	2.51 (1.31–4.81)	.006	7.6
Structural etiology	1.37 (0.36–5.20)	.65	0.2	0.00	>.99	0.0	0.15 (0.02–1.01)	.05	3.8

†missing data 4/358 (1.1%); ‡missing data 13/358 (3.6%)

Table 5. Recovery and residual impairments of occipital ICH patients.

	Occipital ICH (<i>n</i>=17)
Residual visual symptoms	8 (47)
Standard automated perimetry	10 (59)
Hemianopia/partial hemianopia†	2 (20)
Quadrantanopia/partial quadrantanopia†	2 (20)
Scotoma†	2 (20)
Normal†	4 (40)
Driving	
Permitted to drive	9 (53)

Permanent driving ban	2 (12)
No previous driving license	2 (12)
Not assessed	4 (24)
Occupational therapy evaluation	10 (59)
Visual impairment†	6 (60)
Occupational therapy†	2 (20)
Neuropsychological evaluation	6 (35)
Visual impairment‡	5 (83)
Neuropsychological therapy‡	1 (17)
Neurological/neurosurgical control	12 (71)
Habitation at 12 months	
Home	16 (94)
Nursing home	1 (6)
Work	
Returned to work	5 (29)
Disability pension	1 (6)
Retired (before ICH)	9 (53)
Other	2 (12)

N (%) are reported. One patient was lost to follow-up due to out-of-province residence and one died during hospital stay. †*n*=10, ‡*n*=6