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HYDROCEPHALIE AIGUË ET INFARCTUS CÉRÉBRAUX RETARDÉS DANS LES SUITES D'HÉMORRAGIE SOUS ARACHNOIDIENNE ANÉVRISMALE

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HYDROCEPHALIE AIGUË ET INFARCTUS CÉRÉBRAUX RETARDÉS DANS LES SUITES D'HÉMORRAGIE SOUS ARACHNOIDIENNE ANÉVRISMALE

Résumé

Introduction : Les infarctus cérébraux retardés compliquant les suites d'hémorragies sous-arachnoïdiennes anévrismales sont une cause majeure de morbi-mortalité, et les mécanismes physiopathologiques responsables ne sont pas encore totalement élucidés.

Objectif : Nous avons émis l'hypothèse que l'hydrocéphalie aiguë pourrait être une des causes d'ischémie cérébrale retardée, indépendamment de la survenue et de la sévérité du vasospasme.

Méthodes : Les données cliniques et radiologiques des patients pris en charge pour hémorragie sous arachnoïdienne anévrismale dans notre centre entre 2017 et 2020 ont été rétrospectivement analysées. L'infarctus cérébral retardé a été défini comme tout infarctus visible à l'imagerie dans les 6 semaines suivant la rupture anévrismale. L'hydrocéphalie aiguë était retenue sur l'imagerie d'admission dès que l'index bicaudé relatif était supérieur à 1. Le vasospasme cérébral était diagnostiqué sur l'analyse du diamètre des artères intra-crâniennes. Nous avons utilisé des modèles uni- et multivariés pour vérifier les associations entre ces variables.

Résultats : Sur les 164 patients inclus, le vasospasme était présent chez 58 patients (35.4%), et l'infarctus cérébral retardé chez 47 (28.7%). L'hydrocéphalie aiguë était diagnostiquée chez 85 patients (51.8%) sur l'imagerie d'admission. Aucune relation n'a été démontrée entre l'hydrocéphalie aiguë et la survenue d'infarctus cérébral retardé dans notre analyse multivariée (OR ajusté : 1.20 95% CI [0.43– 3.37]; p =0.732). Seule la survenue de vasospasme était indépendamment associée à l'infarctus cérébral retardé (OR ajusté : 10.97 95% CI [4.60 – 26.01]).

Conclusion : Notre étude n'a pas démontré d'association entre l'hydrocéphalie aiguë et l'infarctus cérébral retardé, même après ajustement en fonction de la survenue et de la sévérité du vasospasme.

Mots-clés : Infarctus cérébral retardé, hydrocéphalie aiguë, hémorragie sous arachnoïdienne anévrismale, vasospasme.

ACUTE HYDROCEPHALUS AND DELAYED CEREBRAL INFARCTION AFTER ANEURISMAL SUBARACHNOID HEMORRHAGE

Abstract

Background: Delayed cerebral infarction (DCIn) following aneurysmal subarachnoid hemorrhage (aSAH) is a major cause of morbi-mortality, yet the causes for DCIn remain incompletely understood.

Objective: We tested the hypothesis that acute hydrocephalus could be related to the occurrence of DCIn, independently of the occurrence and severity of vasospasm.

Methods: Radiological and clinical data of patients treated at a single large volume academic center for aSAH between 2017 and 2019 were retrospectively analyzed. DCIn was defined as imaging stigma of cerebral infarction visible on 6 weeks imaging follow-up after aSAH. Hydrocephalus was defined on baseline imaging as a relative bicaudate index above 1. Cerebral vasospasm was defined by reduction of arteries diameter in comparison with initial diameter. We used uni- and multivariable models to test the associations between these variables, hydrocephalus and DCIn.

Results: Of 164 included patients**,** vasospasm occurred in 58 patients (35.4%), and DCIn in 47 (28.7%). Acute hydrocephalus was present in 85 patients (51.8%) on baseline CT. No relation was found between acute hydrocephalus and delayed cerebral infarction in our multivariate analysis (adjusted OR: 1.20 95% CI [0.43– 3.37]; p =0.732). Only vasospasm occurrence was independently associated with DCIn (adjusted OR: 10.97 95% CI [4.60 – 26.01]).

Conclusion: Our study did not show an association between acute hydrocephalus and DCIn after aSAH, after adjustment for the presence and severity of cerebral vasospasm.

Keywords: Delayed cerebral infarction, acute hydrocephalus, aneurysmal subarachnoid hemorrhage, vasospasm.

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En présence des Maîtres de cette Faculté,

de mes chers condisciples
et selon la tradition d'Hippocrate,
je promets et je jure d'être fidèle aux lois de l'honneur et de la probité dans l'exercice de la Médecine.

Je donnerai mes soins gratuits à l'indigent,
et n'exigerai jamais un salaire au-dessus de mon travail.

Admis dans l'intérieur des maisons, mes yeux
ne verront pas ce qui s'y passe, ma langue taira
les secrets qui me seront confiés et mon état ne servira pas à corrompre les mœurs ni à favoriser le crime.

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Table of contents

List of acronyms

SAH: subarachnoid hemorrhage
aSAH: aneurysmal subarachnoid hemorrhage
DCI: delayed cerebral ischemia
DCIn: delayed cerebral infarction
CT: computed tomography
MRI: magnetic resonance imaging
WFNS: world federation of neurosurgical surgery
BCI: bicaudate index
rBCI: relative bicaudate index
VPS: ventricule-peritoneal shunting
EVD: external ventricular drainage

Introduction

Delayed cerebral ischemia (DCI) is a major cause of morbi-mortality in patients with aneurysmal subarachnoid hemorrhage (aSAH) [1]. DCI is a clinical-imaging syndrome consisting of focal neurological and/or cognitive deficits and/or cerebral infarction on CT/MRI, occurring in approximately one third of patients between 3–15 days following the initial hemorrhage [2], resulting from seemingly complex micro- and macrovascular secondary brain insults [3,4]. The causes of DCI are still to be elucidated, currently believed to be diverse [5]. Indeed, while cerebral vasospasm has been consistently associated with DCI [6], other factors such as cerebral oedema, cerebral autoregulation impairment, inflammation and oxidative stress, cortical spreading depression and microthrombosis are likely important but understudied factors [7]. The suspected role of these factors is substantiated by the paradoxical results of many previous DCI targeted therapies, that demonstrated a significant improvement on vasospasm, but failed to improve the rates of DCI and favorable outcomes [8].

Amongst potential factors, acute hydrocephalus remains poorly explored [9] while this frequent phenomenon following aSAH has been linked to various direct and indirect mechanisms that may be at play to explain DCI occurrence variations amongst individuals [10,11]. The association between acute hydrocephalus and DCI has been only scantly studied despite the pathophysiological rationale: by increasing intracranial pressure, acute hydrocephalus could reduce cerebral perfusion and, if left non treated, leading to cerebral ischemia [12,13]. Further, as vasospasm [14], DCI and acute hydrocephalus are largely determined by the abundance of the initial bleed [15-17], investigating their inter-relationship is of importance to shed light on the mechanisms for DCI [18-20].

In a retrospective cohort of patients with aSAH, we tested the hypothesis that acute hydrocephalus may be related to the occurrence of DCIn, adjusting for the occurrence of numerous factors, especially vasospasm and his severity.

Patients and Methods

Patients' selection

Patients admitted at our center following aSAH between January 2017 and January 2020 were screened for inclusion. They were analyzed if they presented within 7 days of symptoms onset, compatible with SAH due to an intracranial aneurysm rupture, as demonstrated using CT and CT-Angiography or MR and MR-Angiography.

Patients with other etiologies of SAH, patients who died in the first 10 days following hemorrhage, mostly due to massive hemorrhage leading to global cerebral oedema and ischemia, and patients with missing data due to an early interfacility transfer were excluded.

Data collection and variables' definitions

For each patient, surgical clipping or endovascular coiling was performed as soon as possible following admission. The initial clinical severity was assessed using the World Federation of Neurosurgical Societies grading scale [21] (WFNS). Aneurysmal SAH was diagnosed by the presence of blood on CT or MRI. The amount of cisternal blood on initial exam was graded according to the Fisher [22] and Hijdra [23] scales. Baseline demographics as well as past medical history were collected using health electronic records.

Delayed cerebral ischemia is defined as a focal neurologic impairment lasting at least 1 hour, not apparent immediately following aneurysm repair and not attributed to other causes, or a decrease of at least 2 points on the Glasgow Coma Scale, or the presence of a cerebral infarction on MRI or CT performed within 6 weeks following the aSAH [24]. We decided to focus on delayed cerebral infarction (DCIn), a more objective and reproducible criterion: DCIn was defined in our study by a new hypodense lesion on CT or a new hyperintensity on DWI or FLAIR, that was not visible on admission or on the immediate postoperative CT, corresponding to a vascular territory not attributable to other causes such as surgical clipping, endovascular treatment, ventricular catheter placement, or intraparenchymal hematoma.

Ventricular enlargement on the admission imaging was quantified by measuring the bicaudate index (BCI) [25]. The BCI is defined as the width of the frontal horns at the level of the caudate nuclei divided by the corresponding diameter of the brain, as shown in Figure 1. To calculate age-adjusted relative sizes, the BCIs were divided by the corresponding upper limit (95th percentile) per age group (rBCI). The measurements of the bicaudate index were done blinded to the subsequent occurrence of DCIn.

Hydrocephalus was defined as a rBCI greater than 1. External ventricular shunting was decided when acute hydrocephalus was associated with neurological deterioration.

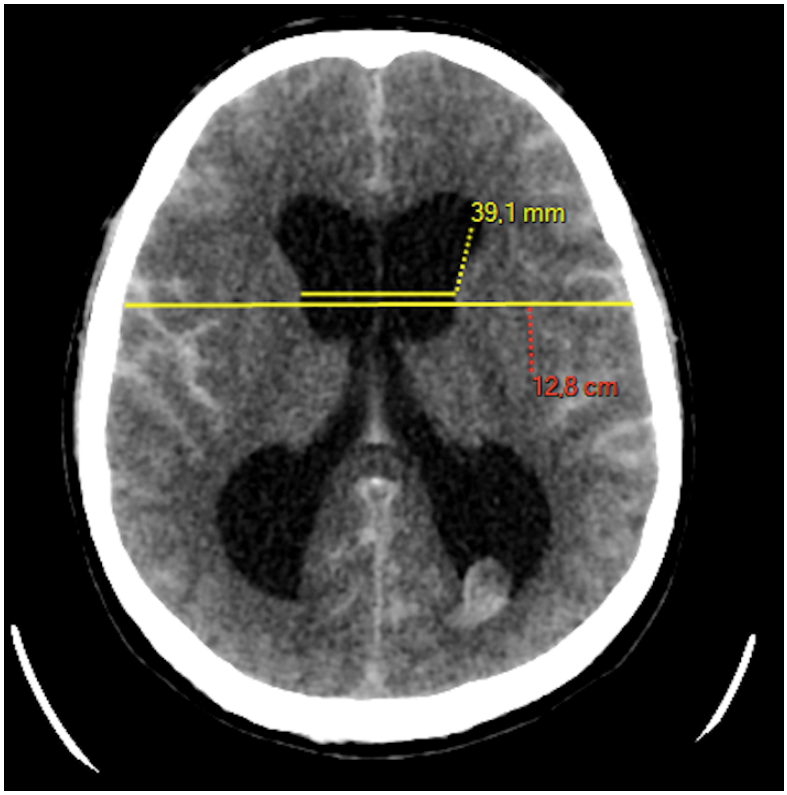

Figure 1: Illustration of the method for measuring bicaudate index. The width of the frontal horns at the level of the caudate nuclei = 3.9 cm. The diameter of the brain at the same level = 12.8 cm. BCI = 3.9/12.8 = 0.30. Patient was 61. rBCI = 1.6

Vasospasm was defined as an objective continuous measure of the intracranial artery diameter on follow-up CT Angiogram performed for neurological deterioration, or when doppler velocities increased over 130 cm/s during daily TCD monitoring. Its prevention consisted of nimodipine administration during the first 21 days. When vasospasm occurred, endovascular treatment was discussed for each patient. Vasospasm was rated as severe when narrowing of the arterial diameter was greater than 50% compared to the admission CT [26]. When CT perfusion was performed, data were analyzed.

MRI and CT were analyzed by two neuroradiologists regarding DCIn and arterial diameter in vasospasm, and dissensus were resolved by consensus.

Statistical analyses

Frequency counts are presented as percentage. Continuous and ordinally scaled variables were tested for normal distribution using the Kolmogorov–Smirnov test and are presented as mean ± standard deviation (SD) and/or as median with interquartile range (IQR). Continuous parameters are compared among patients by either Student's t-test in case of a normal distribution or by the Mann-Whitney U-test in case of a non-normal distribution. Contingency analyses for categorical variables were performed using the Chi-square test. We performed multivariable analysis for DCIn occurrence using a binary logistic regression model. In multivariable models, we adjusted for factors with a p-value < 0.1 in univariate analysis. A 2-sided P-value < 0.05 was considered significant. We performed all statistical analysis using the SPSS software (version 22; IBM Corp.).

Ethics

In accordance with French legislation on the re-use of health data acquired as part of routine care, patients were informed they could oppose the use of their health-related data for research purposes, and written informed consent waived.

Results

Patients' selection and study sample

Between January 2017 and January 2020, 213 patients with SAH were admitted at our institution. 49 patients were excluded. Figure 2 presents a detailed flowchart of patients' selection.

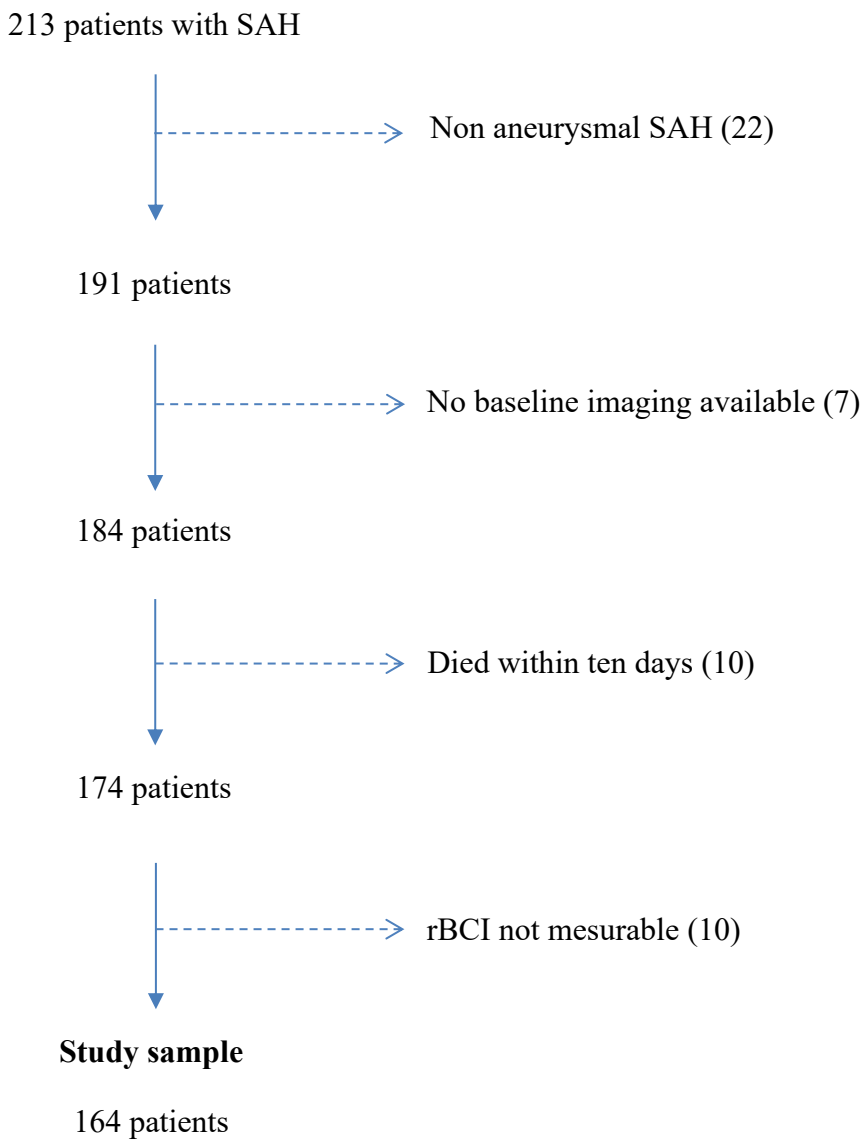

Figure 2: Study flow charts

The final analysis included 164 patients with aSAH with a CT scan performed within 7 days after the ictus.

Median age was 53.8 years [45.2 – 61.8] and 107 patients (65.2%) were women. 40.9 percent of patients had history of smoking. 50 patients (30.5%) suffered of high blood pressure.

At the admission, 84 patients (51.2%) were WFNS I, 17.7% were WFNS II, 1.2% were WFNS III, 13.4% were WFNS IV and 16.5% were WFNS V.

On admission CT scan, the median Hijdra score was 18 [9 – 25]. The ruptured aneurysm was located in the anterior circulation in 151 patients (92.1%).

141 patients (86%) were treated with endovascular embolization and 23 patients (14%) underwent surgical clipping. Cerebral infarction or contusion attributed to a complication from endovascular treatment or surgical clipping was diagnosed for 32 patients (19.5%).

Baseline characteristics of included patients are summarized in Table 1.

Table 1: Patient's characteristics

Variables	n =164
Baseline characteristics	
Age (years)	53,8 [45,2 – 61,8]
Female sex	107 (65.2%)
Active smoking	67 (40,9%)
Excess alcohol consumption	10 (6,1%)
Previous SAH	1 (0,6%)
Familial SAH history	4 (2,4%)
High Blood Pressure	50 (30,5%)
Dyslipidemia	16 (9,8%)
Diabetes mellitus	3 (1,8%)
Ischemic heart disease	2 (1,2%)
Antiplatelet treatment	6 (3,7%)
Anticoagulant treatment	4 (2,4%)
SAH characteristics and management	
Anterior location aneurysm	151 (92,1%)
Fisher grade	
I	3 (1,8%)
II	9 (5,5%)
III	8 (4,9%)
IV	144 (87,8%)
WFNS grade	
1	84 (51,2%)
2	29 (17,7%)
3	2 (1,2%)
4	22 (13,4%)
5	27 (16,5%)
Hijdra Scale (total)	18 [9 - 25]
Baseline rCBI	1 [0,8 - 1,1]
Treatment modality	
Clipping	23 (14%)
Coiling	141 (86%)
Acute hydrocephalus	85 (51,8%)
Requiring EVD	62 (37,8%)
Requiring VPS	26 (15,9%)
Vasospasm	58 (35,4%)
> 50%	44/58 (76%)
Delayed Cerebral Ischemia	47 (28,7%)

Hydrocephalus occurrence and determinants

Acute hydrocephalus occurred in 85 patients (51.8 %) and 62 (37.8 % of the 164 patients) were treated by external ventricular drain (EVD) within 3 days following admission. 26 patients (15.9 %) were treated by Ventricule-Peritoneal Shunting. The distribution of the rBCI at admission is shown in Figure 3.

Hydrocephalus was more frequent in patients with higher Hijdra scale scores (mean 22,5 ± SD 9,5 in patients with hydrocephalus, versus 12,2 ± 8 in those without; $p < 0.01$), with higher Fisher scale grades (4 ± SD 0,2 versus 3,6 ± 0,8; $p < 0.01$), higher WFNS grades (3 ± SD 1,6 vs 1,5 ± 1,1; $p < 0.001$). Other patient or SAH related variables did not influence the risk for acute hydrocephalus.

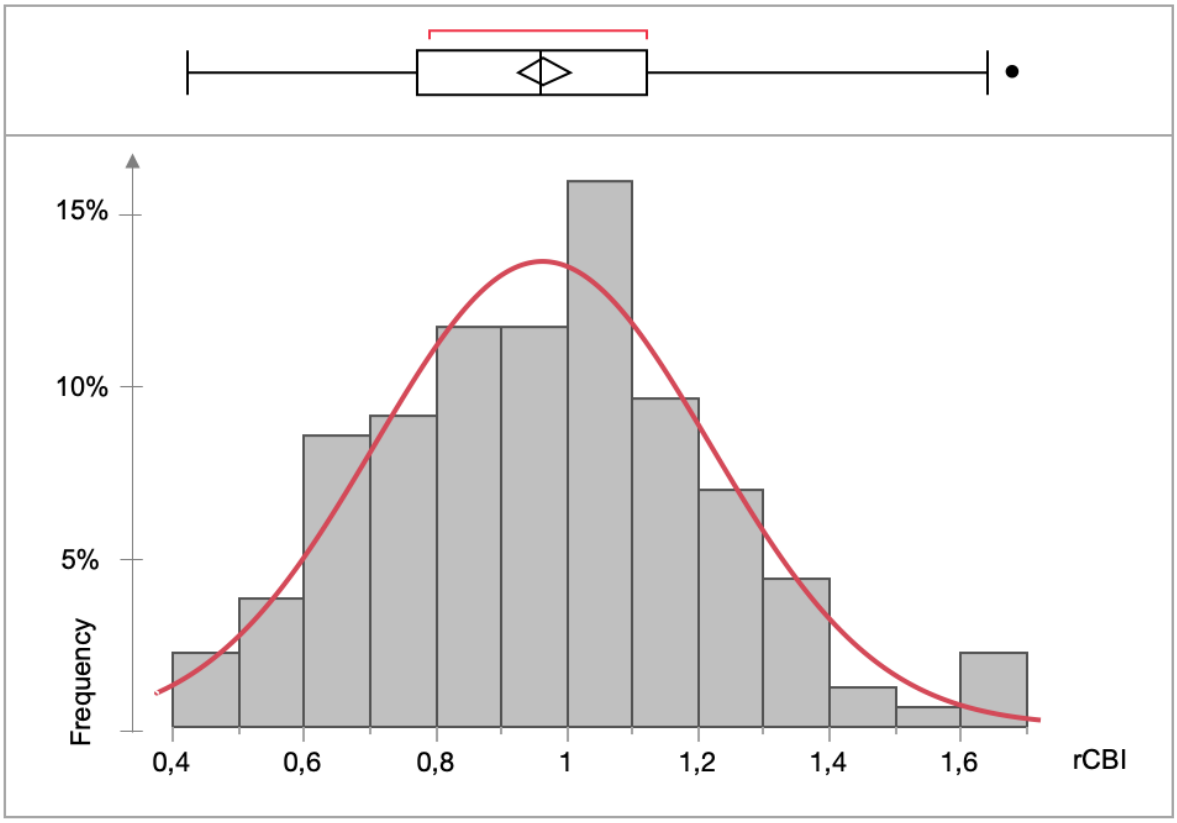

Figure 3: rBCI distribution at the admission. Histogram plot (bottom), box plot (top) and normogram (line) displaying the distribution of rBCI on admission CT.

Vasospasm occurrence

Vasospasm occurred in 58 patients (35.4%) and 44 of them (76% of the 58 patients) developed severe vasospasm defined by narrowing of arterial diameter greater than 50%.

Table 2: Univariable analysis of DCIn determinants

Variables	DCIn (n=47)	No DCIn (n=117)	p value
Age (years)	54,3 ± 13,2	53,7 ± 13	0.801
Female sex	31 (66%)	76 (65%)	1.000
Previous aSAH	0 (0%)	1 (2,1%)	0.288
Family history of	0 (0%)	4 (3,4%)	0.579
Antiplatelet treatment	4 (8,5%)	2 (1,7%)	0.059
Dyslipidemia	6 (12,8%)	10 (8,5%)	0.397
Diabetes Mellitus	1 (2,1%)	2 (1,7%)	0.748
Active smoking	20 (42,6%)	47 (40,2%)	0.861
High blood pressure	15 (31,9%)	35 (29,9%)	0.852
Alcoholism	9 (19,1%)	1 (0,9%)	0.352
Fisher Grade	3,8 ± 0,7	3,8 ± 0,6	0.993
WFNS scale	3,1 ± 1,7	1,9 ± 1,4	<0.001
Total Hijdra scale	21,5 ± 10,4	15,9 ± 9,7	<0.001
Baseline rBCI	1 ± 0,3	0,9 ± 0,2	0.051
rBCI < 1	19 (40,4%)	69 (59%)	0.038
Treatment modality			
Surgery	8 (17%)	15 (12,8%)	0.467
Embolization	39 (83%)	102 (87,2%)	0.467
Vasospasm	36 (76,6%)	22 (18,8%)	<0.001
Hydrocephalus	32 (68,1%)	53 (45,3%)	<0.001
EVD placement	27 (57,4%)	35 (29,9%)	0.001

DCIn occurrence and determinants

DCIn occurred in 47 patients (28.7%) and was found to be more frequent in patients under antiplatelet treatment (8,5% vs 1,7%; p=0.059), in patients with higher WFNS grades (mean

3,1 ± SD 1,7 versus 1,9 ± 1,4; p <0.001), higher total Hijdra scale (21,5 ± 10,4 vs 15,9 ± 9,7; p < 0.001), with hydrocephalus (68,1% vs 45,3%; p <0.001) and with EVD placement (57,4% vs 29,9%; p=0.001). Details are presented in Table 2.

In the multivariable model, adjusting for Hijdra scale, WFNS score, Vasospasm occurrence, EVD placement and hydrocephalus, only vasospasm occurrence remained independently associated with DCIn (adjusted OR: 10.97 95% CI [4.60 – 26.01]). Hydrocephalus was not associated with DCIn (adjusted OR: 1.20 95% CI [0.43– 3.37]; p = 0.732).

Table 3: DCIn occurences per range of rBCI

rBCI range	n patients (total, 164)	Occurrence of DCIn	p value
< 1	88	19 (21,6%)	0.04
1 - 1.2	48	16 (33,3%)	0.45
1.2 - 1.5	23	10 (43,5%)	0.13
>1.5	5	2 (40%)	0.65

Discussion

In this retrospective analysis of prospectively registered patients, we found no independent association between acute hydrocephalus and DCIn. While we found an univariate association between these variables, it was not maintained after adjustment for confounding factors, and most notably the Hijdra and WFNS scale, both reflecting the abundance of initial bleeding which likely confounded the univariable association [27-29].

Our results corroborate a previous study by Bakker & al [13] that also showed that acute hydrocephalus was not a risk factor for the occurrence of DCIn. In this work that included 321 patients within 4 days of aSAH, the authors had not adjusted their analyses for the occurrence or severity of vasospasm, and their work was hence not designed to address this specific question. From a pathophysiological standpoint, it is well established that brain hypoperfusion during SAH is at least partially explained by raised intracranial pressure resulting from hydrocephalus, amongst other factors [30], substantiating our working hypothesis by which hydrocephalus may contribute to DCIn [31-33].

There's been reports of both neurological symptoms and perfusion defects reversal following aggressive CSF diversion for acute hydrocephalus in patients with aSAH and symptomatic cerebral vasospasm [34,35]. In turn, we consider the possibility that the association between hydrocephalus and DCIn in our sample may have been dimmed by an adequate management of acute hydrocephalus by EVD, reducing the delays until intracranial pressure normalization and potentially minimizing the secondary injury mechanisms that may underlie an association with DCIn.

Importantly, as early evacuation of subarachnoid blood has been reported to be the most important strategy to reduce the risk of future shunt dependent hydrocephalus [36], it may have an additional role in limiting hypoperfusion and resulting DCIn.

Also, as shown by van Asch et al [11], the impact of hydrocephalus is a more local than a global process and the effect seems to be more pronounced in the vicinity of the ventricles than in more remote sites with hypoperfusion mostly localized in deep grey matter and periventricular white matter. As we did not investigate the infarcts patterns, we are unable to substantiate this hypothesis.

An other interesting point is that hydrocephalus may indirectly contribute to DCI by releasing natriuretic factor and favouring natriuretic peptid secretion syndrome [18,37], possibly due to the enlargement of the third ventricle, which could interfere with hypothalamic function. Nowadays, this syndrome is correctly treated with sodium and fluid restoration and inhibition of natriuresis with fludrocortisone acetate, which would notably reduce the risk of vasospasm and therefore of DCI [38].

Of note, acute hydrocephalus treatment is not subject of consensus and EVD placement happens to be at the center's discretion. In our center, we treat symptomatic acute hydrocephalus and practice "wait and see" for the asymptomatic ones. As our study didn't show any link between hydrocephalus and DCIn, we could think that's a correct care policy, trying to avoid complications from EVD (infection, rebleeding) [39] but keep reducing intracranial pressure and increase cerebral perfusion by EVD placement when hydrocephalus is symptomatic, to avert resulting complications of prolonged ventricle enlargement [40].

Our study has several limitations.

First, it is a retrospective mono centric study, leading to limited external validity and potential biases.

Second, acute hydrocephalus was either symptomatic and treated by EVD or clinically silent. In our sample we did not investigate the longitudinal effects of increased ventricular volume on cerebral perfusion and the occurrence of DCIn.

Third, DCIn has been radiologically defined as every new cerebral infarction, with inevitable difficulties in assessing whether they were delayed infarctions, related to the procedure, or related to hematoma resorption or the presence of material. We aimed to minimize this bias by a doubled assessment with dissensus resolved in consensus.

Importantly, there was no prespecified timing for follow-up CT for the hydrocephalus evolution, and imaging was obtained at the attending's discretion, based on clinical evolution.

Strengths include a well phenotyped series of patients with aSAH, including notably a quantitative assessment of vasospasm severity, which is recognized in itself as a predictor of DCI and DCIn.

Conclusion

In a retrospective sample of patients with aSAH, there was no association between acute hydrocephalus occurence or its severity, and the occurrence of DCIn.

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Vu, le Directeur de Thèse

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Introduction : Les infarctus cérébraux retardés compliquant les suites d'hémorragies sous-arachnoïdiennes anévrismales sont une cause majeure de morbi-mortalité, et les mécanismes physiopathologiques responsables ne sont pas encore totalement élucidés.

Objectif : Nous avons émis l'hypothèse que l'hydrocéphalie aiguë pourrait être une des causes d'ischémie cérébrale retardée, indépendamment de la survenue et de la sévérité du vasospasme.

Méthodes : Les données cliniques et radiologiques des patients pris en charge pour hémorragie sous arachnoïdienne anévrismale dans notre centre entre 2017 et 2020 ont été rétrospectivement analysées. L'infarctus cérébral retardé a été défini comme tout infarctus visible à l'imagerie dans les 6 semaines suivant la rupture anévrismale. L'hydrocéphalie aiguë était retenue sur l'imagerie d'admission dès que l'index bicaudé relatif était supérieur à 1. Le vasospasme cérébral était diagnostiqué sur l'analyse du diamètre des artères intra-crâniennes. Nous avons utilisé des modèles uni- et multivariés pour vérifier les associations entre ces variables.

Résultats : Sur les 164 patients inclus, le vasospasme était présent chez 58 patients (35.4%), et l'infarctus cérébral retardé chez 47 (28.7%). L'hydrocéphalie aiguë était diagnostiquée chez 85 patients (51.8%) sur l'imagerie d'admission. Aucune relation n'a été démontrée entre l'hydrocéphalie aiguë et la survenue d'infarctus cérébral retardé dans notre analyse multivariée (OR ajusté : 1.20 95% CI [0.43– 3.37]; p =0.732). Seule la survenue de vasospasme était indépendamment associée à l'infarctus cérébral retardé (OR ajusté : 10.97 95% CI [4.60 – 26.01]).

Conclusion : Notre étude n'a pas démontré d'association entre l'hydrocéphalie aiguë et l'infarctus cérébral retardé, même après ajustement en fonction de la survenue et de la sévérité du vasospasme.

Mots-clés : Infarctus cérébral retardé, hydrocéphalie aiguë, hémorragie sous arachnoïdienne anévrismale, vasospasme.

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Membres du Jury :
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