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Retrospective Study of Spontaneous Subarachnoid Haemorrhage: Vasospasm and Other Complications (Part III)

S. Bakanauskaitė*
J. Grigaitė**
R. Danilevičienė**
G. Lizaitienė*
L. Piliponis***
J. Ščerbak****
D. Jatužis**
J. Valaikienė**

**Faculty of Medicine, Vilnius University, Lithuania*

***Centre of Neurology, Faculty of Medicine, Vilnius University, Lithuania*

****Faculty of Medicine, Lithuanian University of Health Sciences, Lithuania*

*****Centre of Neurosurgery, Vilnius University Hospital Santaros Klinikos, Lithuania*

Summary. *Background.* Acute subarachnoid haemorrhage (SAH) is a dangerous condition: roughly 50% of patients die within 30 days, nearly 25% develop significant neuropsychological and cognitive deficits, about 10% suffer from other complications, and only around 15% recover fully. One of the main causes of mortality is cerebral vasospasm (CVS), a cause of delayed cerebral ischaemia (DCI), which can lead to secondary cerebral infarction. Thus, early detection of CVS is crucial for improving patient outcomes.

The aim of this study was to analyse the demographic and clinical (including complications) data of patients diagnosed with SAH, to evaluate the diagnostic methods, and to investigate the prevalence and treatment of complications.

Materials and methods. This retrospective study included 102 patients diagnosed with spontaneous SAH, aged at least 18 years, who were treated in the Vilnius University Hospital Santaros Klinikos (VUL SK) in 2014-2017. The obtained VUL SK data were selected by the I60 ICD-10-CM code and depersonalized. Head computerized tomography (CT), CT angiography (CTA), digital subtraction angiography (DSA), transcranial color-coded duplex sonography (TCCS), and magnetic resonance angiography (MRA) were used to evaluate the cerebral arteries.

Results. In 102 patients (mean age 58±16 years; 55% female), the most common cause of spontaneous SAH was ruptured aneurysm, the most common arterial sites were the middle cerebral artery (36%), anterior cerebral artery and/or communicating artery (together 33%), and internal carotid artery (20%). In-hospital mortality was as high as 21%. All patients underwent head CT, 78% CTA, 33% DSA, 8% TCCS, and 1% MRA. 71% patients experienced SAH complications: 66% acute, 27% chronic. The most common acute complications were cerebral oedema (34%), rebleeding (29%), and acute ischaemia (23%), the most common chronic complications hydrocephalus (11%), DCI (9%), and CVS (8%).

Conclusions. CVS was diagnosed considerably less frequently in this study compared to other studies. While the diagnosis rates of ischaemia and hyponatremia were likewise significantly lower in this study compared to those in others, they are in line with the CVS diagnosis rate. Aside from the CVS and CVS-related complications, all the other SAH complications were roughly within the range of the other studies.

Keywords: subarachnoid haemorrhage, intracranial aneurysm, diagnostics, complication, vasospasm.

INTRODUCTION

Acute subarachnoid haemorrhage (SAH) is a dangerous condition in which blood exudes into the subarachnoid space [1]. Even though the most common cause of SAH is trauma, SAH caused by a ruptured aneurysm occurs in approximately 9.7-14.5 per 100,000 people worldwide (in

Address:
Simona Bakanauskaitė
Faculty of Medicine, Vilnius University
M. K. Čiurlionio St. 21, LT-03101 Vilnius, Lithuania
E-mail: sbakanauskaite@gmail.com

Lithuania, 2.7 per 100,000) [2–4]. Around half of SAH patients are under the age of 55 [2]. Aside from intracerebral aneurysm rupture, other causes of spontaneous SAH include arteriovenous malformation (AVM) / fistula, vasculitis, intracerebral artery dissection, amyloid angiopathy, and drug use [5]. SAH mortality ranges from 8% to 67% [3]. Despite advances in the diagnosis and treatment of SAH, approximately 50% of patients die within 30 days, nearly 25% develop significant neuropsychological and cognitive deficits, about 10% suffer from other complications, and only around 15% recover fully [2, 6, 7]. The clinical prognosis of SAH depends on those complications that can cause severe disability and lethal outcomes [5]. The complications of SAH are categorised by the time of development into acute (the first 3 days after the event) and chronic (after the third day). The most common acute complications are rebleeding, acute hydrocephalus, and cerebral oedema [8]. Cerebral infarction, which is one of the most serious SAH complications, forms in ~30% of patients [5]. The most common chronic complications are chronic hydrocephalus and cerebral vasospasm (CVS) [8]. CVS is one of the main causes of mortality and delayed cerebral ischaemia (DCI), which can cause a secondary ischaemic stroke [9]. A DCI-related cerebral infarct is a potentially avoidable cause of mortality and morbidity after experiencing SAH [6]. The risk of DCI occurring increases depending on the grade of angiographically confirmed CVS; the more severe the grade, the higher the risk. It is noteworthy that CVS does not always cause DCI, which can have other causes, for example, arterial occlusion/damage during surgical intervention, thromboembolism, microclot, etc. [3, 10, 11]. CVS is linked to a high rate of morbidity and mortality and may require multiple treatments when refractory, which leads to higher treatment costs and longer hospital stays [12]. Thus, early detection of CVS and other aforementioned complications is crucial for improving patient outcomes. The aim of this study was to analyse the demographic and clinical (including complications) data of patients diagnosed with SAH and treated in the Vilnius University Hospital Santaros Klinikos (VUL SK) in 2014-2017, to evaluate the diagnostic methods, and to investigate the prevalence and treatment of complications.

Table 1. **General clinical characteristics, functional status, neuroimaging and in-hospital mortality of patients**

Variables	All (n=102) (%)
Female	56 (55%)
Male	46 (45%)
World Federation of Neurosurgical Societies (WFNS) Grading System for SAH	
I	40 (39%)
II	6 (6%)
III	14 (14%)
IV	8 (8%)
V	19 (19%)
Data unavailable	15 (15%)
Hunt-Hess scale	
0	3 (3%)
1	19 (19%)
2	22 (22%)
3	13 (13%)
4	4 (4%)
5	20 (20%)
Data unavailable	21 (21%)
Glasgow coma scale (GCS)	
3-8	19 (19%)
9-12	10 (10%)
13-15	61 (60%)
Data unavailable	12 (12%)
Modified Rankin scale (mRS)	
0	8 (8%)
1	19 (19%)
2	12 (12%)
3	12 (12%)
4	15 (15%)
5	12 (12%)
6	24 (24%)
Head CTA	80 (78%)
Head DSA	32 (33%)
Head MRA	1 (1%)
Post-operation head CT	64 (63%)
Pre-release head CT	39 (38%)
TCCS	8 (8%)
Deaths	22 (21%)

METHODS

The Lithuanian Bioethics Committee of Vilnius Region on 04-12-2018 issued Permit no. 158200-18/12-1076-577 for the study "Acute and Chronic Complications of Patients Who Experienced SAH: Prognostic Factors and Clinical Outcomes in VUL SK in 2014-2017", which analysed demographic and clinical data of patients who experienced SAH from 01-01-2014 to 31-12-2017. The study data were obtained from the VUL SK electronic health record system, selected by I60 ICD-10-CM code and depersonalised. Only pre-discharge patient health data was available. Any personal data in the questionnaire was encrypted to ensure confidentiality. The study included patients diagnosed with spontaneous SAH who were at least 18 years old and had been treated in the VUL SK in 2014-2017. Patient sociodemographic data, including age and gender, were collected. Specific parameters were analysed using the World Federation of Neurological Surgeons Grading System (WFNS) SAH grading scale, the Hunt-Hess scale, the Glasgow coma scale (GCS), and the aneurysm treatment method (surgical or endovascular), as well as by the aneurysm location and the in-hospital mortality rate. Functional status was evaluated using the modified Rankin scale (mRS) at the discharge of the patient and categorised into favourable (mRS 0-2) and unfavourable (mRS 3-6) outcomes. Data were also collected on the need for additional

neurosurgical treatment, i.e., external ventricular drains (EVD) for acute and/or symptomatic hydrocephalus.

The statistical data were analysed using MS Excel. The quantitative data that conformed to the normal distribution are presented as averages, and the data that did not conform are presented as median and minimal and maximal values. The categorical data are presented as numerical values and percentages.

The pathologies of the cerebral arteries were evaluated using head computerized tomography (CT), CT angiography (CTA), digital subtraction angiography (DSA), transcranial Doppler ultrasound (TCD), transcranial color-coded duplex sonography (TCCS), and magnetic resonance angiography (MRA).

RESULTS

Overall, 114 patients experienced SAH, of which 12 patients were not included in this study because the cause of their SAH was trauma. Table 1 shows the clinical characteristics of the analysed patients, including data on their functional status; Figure 1 presents the distribution of patients by age. The average patient age was 58 years with a standard deviation of ±16 years. The majority (55%) of the patients were female.

58% of patients had primary arterial hypertension (PAH), 8% were smokers (5% non-smokers and 87% unclassified), 25% experienced sentinel pain due to SAH, although overall 57% complained just of a headache, 40% had positive meningeal symptoms, 33% experienced loss of consciousness, 33% had limb paresis, 28% had motor aphasia, and 27% had cranial nerve damage. The patients evaluated their own pain with an average score of 8.23 using the Visual Analogue Scale (VAS).

All patients diagnosed with spontaneous SAH were treated in the intensive care unit. Each of them underwent head CT, 78% CTA, 33% DSA, and 1% MRA. In addition, one patient underwent cerebral angiography during brain death examination and one patient underwent aortography during coronary angiography. CVSs were mostly diagnosed by DSA (4%), less commonly by CT/CTA (2%), TCCS (1%), or only clinically (1%). One patient developed both acute ischaemia and CVS, but no patients developed both DCI and CVS. After treatment, arterial flow was assessed using TCCS in 8% of patients and using extracranial colour-coded duplex sonography (ECCS) in 8% of patients (Table 1).

Table 2. **Location of the aneurysms**

Location of the aneurysms	N=64 (%)
Internal carotid artery (ICA):	13 (20%)
ICA cavernous segment	2 (3%)
ICA clinoid segment	3 (5%)
ICA bifurcation	8 (13%)
Posterior communicating artery (PComm)	3 (5%)
Anterior communicating artery (AComm)	16 (25%)
Anterior cerebral artery (ACA)	5 (8%)
Middle cerebral artery (MCA):	23 (36%)
MCA proximal segment	9 (14%)
MCA insular segment	14 (22%)
Posterior cerebral artery (PCA):	2 (3%)
PCA proximal segment	1 (2%)
PCA distal segment	1 (2%)
Basilar artery (BA):	2 (3%)
BA bifurcation segment	1 (2%)
BA brainstem	1 (2%)

Of the 88% of patients who underwent angiography (CTA, DSA, MRA, etc.), 70% (64 patients) of SAH involved a ruptured aneurysm, 5% AVM or other blood vessel malformation, 5% a complication of endovascular intervention, 4% a typical perimesencephalic SAH, 3% complications of intravenous thrombolysis, 1% microangiopathy, 1% bleeding diathesis, 1% other secondary bleeding, and 11% different causes of bleeding. In 3%, the aneurysm was diagnosed by head CT instead of angiography. 12% also had an unruptured aneurysm. Among 70% of patients with ruptured aneurysms, the most common sites of aneurysms were the middle cerebral (MCA) artery (36%), anterior cerebral artery (ACA) and/or communicating artery (AComm) (together 33%), and internal carotid artery (ICA) (20%) (Table 2).

43% of patients with spontaneous SAH were treated by aneurysm clipping, 8% by coiling. 65% of all patients were given medication. 58% of patients received antihypertension drugs, 30% calcium channel blockers, 25% osmotic di-

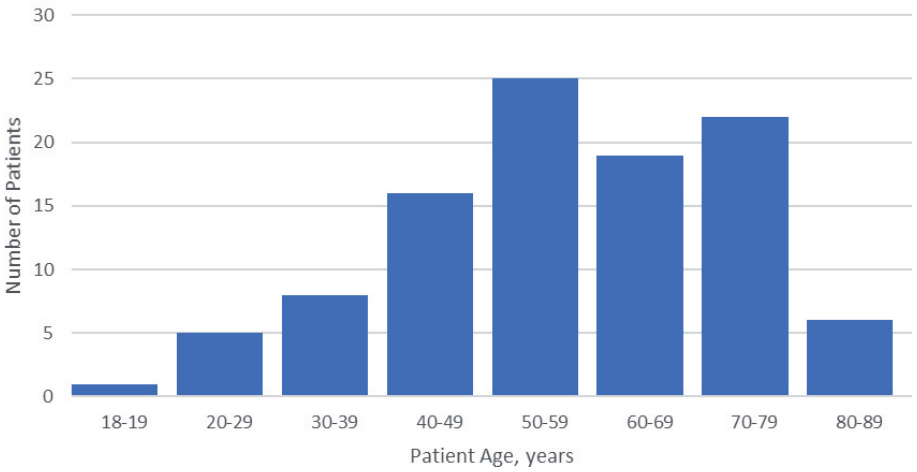

Fig. 1. **Patient distribution by age**

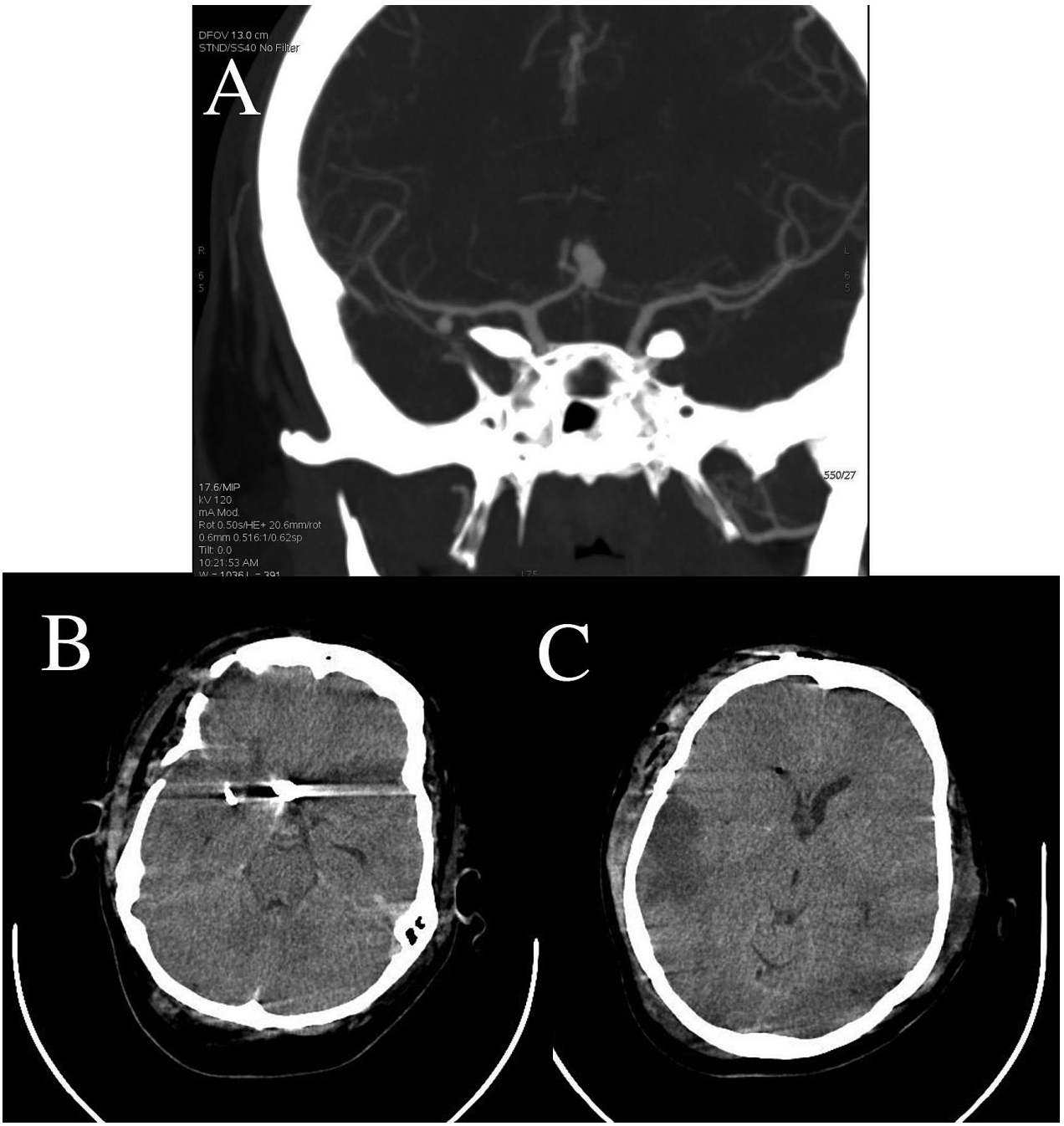

Fig. 2. **A clinical case – an example of SAH with subsequent cerebral ischaemia**
A 52-year-old female arrived at the emergency room after an episode of sudden-onset numbness in all four limbs and speech impairment that lasted up to an hour. The neurological examination was unremarkable. The patient was diagnosed with a panic attack and was discharged with no further investigation.A year later she was admitted after being found unconscious. She complained of a very intense headache and nausea. A neurological examination revealed a stiff neck. An urgent head CTA showed a SAH and two aneurysms at the right AComm and MCA (Fig. 2a). A pterional craniotomy was performed, both the ruptured AComm and unruptured MCA aneurysms were found and clipped (Fig. 2b). After the operation, the patient developed delirium, which persisted until discharge. No focal neurological signs were observed. The subsequent head CT revealed an ischaemic zone in the right temporal area (Fig. 2c) and the subsequent DSA confirmed a CVS.

uretics, 18% triple-H therapy (hypertension, hypervolemia, haemodilution), and 4% inotropic drug infusions. 21% of patients had EVD for symptomatic hydrocephalus.

40% of patients had a favourable functional status and 60% had an unfavourable status. In-hospital mortality was 21% (Table 1). 71% experienced SAH complications: 66% acute, 27% chronic (Fig. 2). 24% of patients had no complications and for 8% the situation was unknown. The most common acute complications were cerebral oedema (34%), rebleeding (29%), and acute ischaemia (23%), while the most common chronic complications were hydrocephalus (11%), DCI (9%), and CVS (8%) (Table 3).

Table 3. **SAH complications**

Complications	Number of patients (%)
Acute complications:	67 (66%)
Cerebral oedema	35 (34%)
Rebleeding	30 (29%)
Acute ischaemia	23 (23%)
Acute hydrocephalus	20 (20%)
Hyponatremia	13 (13%)
Intracerebral haemorrhage	11 (11%)
Hypokalaemia	10 (10%)
Infection	10 (10%)
Intraventricular haemorrhage	8 (8%)
Transient ischaemic attack	5 (5%)
Hypernatremia	2 (2%)
Headache	1 (1%)
Seizures	1 (1%)
Delirium	1 (1%)
Other complications (stress cardiomyopathy / neurogenic pulmonary oedema)	1 (1%)
Chronic complications:	28 (27%)
Chronic hydrocephalus	11 (11%)
Delayed cerebral ischaemia	9 (9%)
Cerebral vasospasm	8 (8%)
Persistent delirium	5 (5%)
Hyponatremia	2 (2%)

DISCUSSION

SAH complications are an important subject, as similar studies have shown, since they affect survival rate and functional prognosis, as do other factors such as gender, age, PAH, smoking, and positive family history [2, 8, 13–17], which also figure in this study: the average age was 58, the majority were female (55%), and most had PAH (58%). Even though smoking and a positive family history are significant risk factors, the study database contained no information on them for the vast majority of patients, presumably because the data compilers had no access to the relevant medical history files.

In this study, as in other SAH studies, a ruptured aneurysm was the main cause of spontaneous SAH, 70% and 85%, respectively [2, 14, 15, 18, 19]. The most common aneurysm location in this study was MCA (36%), although in other studies it was ACA [18, 19].

The most serious complication of acute SAH is rebleeding, which is associated with a higher mortality rate [8, 14, 16, 17]. For this reason, it is recommended to start rebleeding prevention before a specific ruptured aneurysm treatment [17]. In other studies, 7-26% (average 13%) of patients experienced rebleeding [8, 14, 16, 17] compared to 28% in this study.

For patients with SAH, one of the main causes of mortality is CVS, which can be identified using TCD/TCCS (average maximum blood flow rate in excess of 120 cm/s) [9, 20]. This complication usually occurs 3-14 days (peaking at 7-10) after SAH, but ultrasound changes can be seen from the second day [2, 8, 14, 15, 20]. While CVS can present with symptoms such as focal neurologic deficit, agitation, and confusion, it can also be asymptomatic up to 70% of the time [8, 14]. Therefore, the diagnosis should be confirmed angiographically, for which some use TCD as a first-line diagnostic tool [8]. In other studies, CVS was confirmed in 30-75% of patients with SAH [12, 15, 21], but only in 7% of patients in this study.

Acute ischaemia is a complication that manifests during the first 24-72 hours after SAH [20, 22]. It can be indicated by cerebral oedema, which is linked to loss of consciousness at ictus [22]. In this study, acute ischaemia was diagnosed radiologically in only 23% of patients, but in 66% in other studies [23]. After the first 72 hours, DCI can occur, which manifests as focal impairment or a 2-point decrease in the GCS score persisting for at least 1 hour, if other causes are ruled out [5, 24–26]. This complication poses a higher risk for younger (<55 years) patients and those who smoke. Even in serious cases, it can be silent [27]. DCI is diagnosed radiologically in 40-60% of patients, but in this study, in only 9% of patients, who had an average mRS of 4 [5, 14, 24, 26].

Cerebral oedema is a complication, which can cause diffuse cerebral cortex damage, possibly lethal, within a couple of hours of SAH [8]. This complication is an independent predictor of death or severe disability in the first 3 months after SAH, even after taking into account other prognostic factors such as age, aneurysm size, and neurological status upon arrival at the hospital [8, 28]. Sudden diffuse cerebral oedema can form regardless of the severity of the primary haemorrhaging, i.e., even after minor haemorrhaging [8]. In this study, cerebral oedema occurred in 34% of patients, a more common complication than in other studies (15-20%) [15, 28].

Acute obstructive hydrocephalus, a sudden dilatation of the ventricular system caused by mechanical obstruction of the cerebrospinal fluid flow, which causes an increase in intracranial pressure, is another complication that worsens the prognosis of SAH [8]. Acute hydrocephalus presents clinically as a progressively worsening mental status and increased intracranial pressure (headache, nausea, vomiting, eye stasis, arterial hypertension, bradycardia, alteration or cessation of respiration), and radiologically as a dilatation of the ventricles [29]. Chronic hyporesorptive hydrocephalus, which is also diagnosed using head CT, is due to the impaired permeability of the arachnoid granulations, indirectly caused by the dissolution of blood clots; this prevents the normal reabsorption of cerebrospinal fluid and also causes dilatation of the ventricular system. A typical clinical manifestation is the Adam and Hakims triad, i.e., gait ataxia, urinary incontinence, and cognitive disorders (usually disorientation and confusion) [8]. In this study, 20% of patients had acute hydrocephalus and 11% had chronic hydrocephalus, which corresponds to the 20-30% incidence rate in other studies [8, 14, 16, 17].

Hyponatremia is a water and electrolyte imbalance, i.e., a decrease in the plasma Na^+ concentration below 135 mmol/L, that usually manifests neurologically due to the movement of liquids into the brain's cells which causes cerebral oedema, altered mental status, and seizures [30, 31]. This can complicate the patient's course of treatment, cause irreversible brain damage, and increase mortality. It is also thought to be one of the causes of CVS after SAH [30]. In this study, only 15% of patients had hyponatremia (13% acute and 2% chronic), but in other studies it was at least 30% [30–32].

The most common acute SAH complications in this study, cerebral oedema (34%), rebleeding (28%), and acute ischaemia (23%), have similar occurrence rates found in other studies [8, 14–17], but the most common chronic complications in this study, chronic hydrocephalus (11%) and DCI (9%), fail to correspond to the most common rates in other studies: CVS (30-75%) and DCI (40-60%) [8, 17].

For decades, the standard diagnostic tool for cerebral blood vessel haemorrhaging has been conventional angiography, which is nowadays usually performed using DSA. While the American Heart Association and the American Stroke Association do recommend its use with 3D reconstruction before any anticipated SAH treatment for a ruptured aneurysm (level IB), the American College of Radiology in its 2020 Appropriateness Criteria states that DSA and CTA are equal diagnostic tools, but both are invasive, expensive, and time consuming compared to TCD/TCCS [33].

TCD/TCCS are non-invasive, inexpensive, and repeatable (due to non-ionising radiation) diagnostic tools for evaluating intracranial haemodynamics in many cerebral blood vessel diseases [34]. They can assess clinically significant indicators such as linear blood flow rate, pulsatility index, and blood vessel diameter, and diagnose CVS before any brain damage or neurological deficit occurs [34, 35]. The present results show that in real practice, TCCS was not used frequently enough, i.e., only for 8% of patients after SAH; furthermore, of the 8% of patients diagnosed with CVS, only 1% were diagnosed using TCCS. Daily use of TCD/TCCS is recommended, especially for younger patients, in order to capture elevated and increasing (depending on the elapsed time) blood flow velocities as soon as possible [7, 17, 20, 26, 36–39].

This study has several limitations. First, the retrospective nature of the analysis means that the results may not reflect the current situation, missing data cannot be recovered, and data collection errors cannot be corrected in real time. Second, the limitation of the study to a single centre means the sample size is very small; adding more centres would better reflect the overall situation. Third, the lack of summarised data on SAH complications and their occurrence rates affects the ability to draw relevant conclusions. Fourth, only pre-discharge patient health data were available, which means the final rates of complications and mortality may differ from those reported in this study.

CONCLUSIONS

CVS was diagnosed significantly less frequently in this study compared to other studies. Increased diagnosis rates might be achieved using TCCS, a non-invasive tool that can be used repeatedly. Although the diagnosis rates of ischaemia and hyponatremia, i.e., CVS-related complications, were also significantly lower in this study compared to those in others, they are in line with the CVS diagnosis rate. Aside from the CVS and CVS-related complications, all the other SAH complications were roughly within the range of the other studies.

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S. Bakanauskaitė, J. Grigaitė, R. Danilevičienė, G. Lizaitienė, L. Piliponis, J. Ščerbak, D. Jatužis, J. Valaikienė

SPONTANINIŲ SUBARACHNOIDINIŲ HEMORAGIJŲ RETROSPEKTYVINIS TYRIMAS: VAZOSPAZMAS IR KITOS KOMPLIKACIJOS (III DALIS)

Santrauka

Įvadas. Ūminė subarachnoidinė hemoragija (SAH) yra gyvybei grėsminga būklė: beveik 50 % asmenų, sergančių SAH, miršta per 30 dienų, maždaug 25 % pacientų lieka su reikšmingais neuropsichologiniais ir pažinimo sutrikimais, apytiksliai 10 % stebimos kitos komplikacijos ir tik apie 15 % visiškai pasveiksta. Viena iš pagrindinių mirštamumo priežasčių yra radiologinis cerebrinis vazospazmas, kuris yra viena iš vėlyvos smegenų išemijos priežasčių, galinti sukelti antrinį išeminį insultą. Tad, siekiant pagerinti SAH patyrusių ligonių klinikines išeitis, ypač svarbu laiku diagnozuoti vazospazmą.

Tyrimo tikslas – išanalizuoti pacientų, patyrusių SAH, demografinius ir klinikinius duomenis, įskaitant komplikacijas, įvertinti jų dažnį, diagnostiką ir gydymą.

Metodika. Į šį retrospektyvinį tyrimą buvo įtraukti 102 pilnamečiai pacientai, kuriems buvo diagnozuota spontaninė SAH ir kurie buvo gydyti Vilniaus universiteto ligoninės Santaros klinikose (VUL SK) 2014–2017 m. Iš VUL SK surinkti duomenys pagal I60 TLK-10-AM kodą ir nuasmeninti. Galvos smegenų arterijoms įvertinti buvo naudojami šie tyrimai: galvos kompiuterinė tomografija (KT), KT angiografija (KTA), skaitmeninė subtrakcinė angiografija (SSA), transkranijinė spalvinė duplekssonografija (TKSS) ir magnetinio rezonanso angiografija (MRA).

Rezultatai. 102 pacientams (amžiaus vidurkis – 58 ± 16 m., 55 % – moterys) dažniausia spontaninės SAH priežastis buvo aneurizmos plyšimas, dažniausia plyšusios aneurizmos lokalizacija – vidurinė smegenų arterija (36 %), priekinė smegenų arterija ir priekinė jungiančioji arterija (kartu sudarė 33 %), vidinė miego arterija (20 %). Mirštamumas ligoninėje siekė net 21 %. Visiems pacientams buvo atlikta galvos KT, 78 % – KTA, 33 % – SSA, 8 % – TKSS, 1 % – MRA. 71 % įvyko komplikacijos po SAH: 66 % – ankstyvos komplikacijos, 27 % – vėlyvos. Dažniausios ankstyvos komplikacijos – smegenų edema (34 %), pakartotinis kraujavimas (29 %), ankstyva išemija (23 %). Tuo tarpu dažniausios vėlyvos komplikacijos – hidrocefalija (11 %), vėlyva galvos smegenų išemija (9 %), vazospazmas (8 %).

Išvados. Vazospazmas tiriamiems pacientams diagnozuotas daug rečiau, nei kituose publikuotuose tyrimuose. Nors išemijos ir hiponatremijos atvejų buvo nustatyta mažiau, palyginti su kitų autorių duomenimis, jos šiame tyrime diagnozuotos panašiu dažniu kaip ir vazospazmas. Kitų SAH komplikacijų dažnis maždaug atitiko tarptautinių tyrimų rezultatus.

Raktažodžiai: subarachnoidinė hemoragija, intrakranijinė aneurizma, diagnostika, komplikacijos, vazospazmas.

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