Vertigo: Could this Symptom Indicate the Existence of an Unruptured Intracranial Aneurysm?

Grigorios GKASDARIS^{a, b}, Pedram TABATABAEI^c, Harry KOURTOPOULOS^{c, d},
Theodossios BIRBILIS^a

^aDepartment of Neurosurgery, University Hospital of Alexandroupolis, Alexandroupolis, Greece

^bDepartment of Neurosurgical Oncology and Vascular Neurosurgery, Pierre Wertheimer Neurological and Neurosurgical Hospital, Hospices Civils de Lyon, Lyon, France

^cDepartment of Neurosurgery, University Hospital of Umeå, Sweden

^dDepartment of Neurosurgery, University Hospital of Larissa, Greece

ABSTRACT

Background: Unruptured intracranial aneurysms (UIAs) can be presented with various symptoms, including atypical headaches and cranial nerve deficits. Vertigo is often referred in the literature as a coexisting symptom. Our aim was to investigate the importance of vertigo in the UIA symptomatology and present a possible explanation for its existence.

Methods: We conducted a retrospective observational multicenter study concerning patients with surgically treated intracranial aneurysms. During a period of 10 years, 1 085 patients with cerebral aneurysms underwent surgery. There were 812 patients with ruptured intracranial aneurysms (RIA) and 273 with UIA. The medical records for each of the 273 patients were analyzed.

Results: After the implementation of exclusion criteria, 89 (32.6%) of UIA patients were selected in the study, from which 71 (79.8%) were females and 18 (20.2%) males. The mean age was 56.9 ($\pm$ 12.876) years old. Vertigo existed in 72 (80.9%), headache in 41 (46.1%) and visual symptoms in 21 (23.6%) patients. No significant correlation ($p > 0.05$) was demonstrated between gender, age or aneurysm location in correlation with vertigo, headache or visual symptoms, apart from a negative significant correlation between age and vertigo ($p = 0.031$).

Conclusions: Vertigo is an alarming symptom that could indicate the existence of an UIA. The pathophysiological mechanism could be explained by the formation of an aneurysmal vortex that projects into the parent artery, leading to disturbances in the laminar flow and formation of an irregular/turbulent flow, which potentially affects the cerebral autoregulation and by consequence, the central processing of movement.

Keywords: intracranial aneurysm, unruptured, vertigo, hemodynamics, turbulent flow, cerebral flow, cerebral autoregulation.

Address for correspondence:

Grigorios Gkasdaris M.D.

Department of Neurosurgery, University Hospital of Alexandroupolis, Greece

Department of Neurosurgical Oncology and Vascular Neurosurgery, Pierre Wertheimer Neurological and Neurosurgical Hospital, Hospices Civils de Lyon, Lyon, France

email: grgkasdaris@gmail.com

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INTRODUCTION

An intracranial aneurysm (IA) is a focal vascular defect usually presented as an outpouching of the cerebral artery wall, mostly referring to saccular aneurysms. An IA can be discovered at imaging obtained for unrelated causes, detected at imaging obtained for symptoms possibly related to an unruptured intracranial aneurysm (UIA), or it can be diagnosed as subarachnoid hemorrhage (SAH) at the time of the aneurysmal rupture (1). The reported prevalence of UIAs remains at 2-6% (2-4). The incidence of rupture is estimated at 10-15/100,000/year (5,4). Intracranial aneurysms are responsible for 75% of SAH, which is correlated with severe neurologic disabilities and mortality at 30-50% (2,5). However, most of the UIAs remain silent, or may be presented with non-specific symptoms (1).

These symptoms are very diverse and heterogeneous. The most usual symptoms described in the literature are new-onset acute headaches, isolated cranial nerve palsy or even ischemic stroke (2). Interestingly, over the last decades, numerous articles have mentioned vertigo as an initial/coexisting symptom of UIAs (6-14). However, in the literature vertigo is not yet established as a characteristic symptom on the background of an UIA, neither there is an explanation for the mechanism of its creation.

In our clinical practice, we observed that a significant percentage of patients with symptomatic UIA complained for vertigo and thus, the aim of our study was to examine the importance of this symptom along with all the other symptoms in relation to gender, age and aneurysm location. Furthermore, we propose a pathophysiological theory in an attempt to explain the mechanism of vertigo. The initial hypothesis stated that the tested parameters would present statistically significant correlations especially between vertigo and aneurysm location. □

MATERIAL AND METHODS

Patients

We conducted a retrospective observational multicenter study based on data collected during a period of 10 years from our hospitalized patients who underwent surgery due to the existence of one or more, ruptured or unruptured IAs.

FIGURE 1. Digital subtraction angiography showing a giant aneurysm (white asterisk) of the cavernous segment of the left internal carotid artery (lateral view)

We found that 1 085 patients with IAs had been treated surgically. From them, 812 patients presented with ruptured intracranial aneurysms (RIA) and 273 with UIA. The medical records of each of the 273 patients were analyzed with respect to history, physical examination, operative protocol, and imaging (CT/CTA, MRI/MRA, DSA) (Figure 1).

Exclusion parameters included the presence of at least one of the following criteria: patients initially treated for a ruptured aneurysm with an unruptured one identified during the primary DSA investigation, patients who had earlier undergone any neurosurgical treatment, patients who were totally asymptomatic when the aneurysm was found, dissecting aneurysm or aneurysm with arteriovenous malformation. The study was approved by the institutional review board. Patient consent form was not necessary for this type of study.

Clinical parameters

We searched individual patient files for the presenting symptoms and focused on registration of vertigo (by either a single word or phrases used to describe the symptom) and additional written information from each patient file in order to objectify the presence of vertigo. Only patient files with the term "vertigo" or the periphrastic description

of it, and the absence of an obvious vertiginous etiology, were taken into consideration. Additionally, acute onset headache and visual symptoms (impaired vision, ophthalmoplegia, etc) were assessed as parameters in patients' symptomatology. The remaining complaints represented only a minority of all symptoms.

The existence of the aneurysms was analyzed according to the location in reference to the circle of Willis (anterior circulation vs posterior circulation) and in accordance with the specific aneurysm location, including PCoA (posterior communicating artery), MCA (middle cerebral artery), ACoA (anterior communicating artery), OphA (ophthalmic artery), basilar bifurcation, PICA (posterior inferior cerebellar artery), cavernous ICA (internal carotid artery), AChA (anterior choroidal artery), supraclinoid-ICA, ICA-bifurcation, SCA (superior cerebellar artery), PCA (posterior cerebral artery), basilar trunk and VA (vertebral artery). (15)

Statistical analysis

Data were thereafter gathered and statistically analyzed using the IBM SPSS Statistics (Version 23.0. Armonk, NY: IBM Corp, 2015). Correlation analyses amongst gender, circle of Willis, aneurysm location and vertigo, headache, visual symptoms were performed using the chi-square test. Samples created by estimating the distribution of frequencies of the presence or absence of each symptom across the age spectrum and they were examined for normality using the Kolmogorov-Smirnov and Shapiro-Wilk tests. The Independent-sample t-test was used for the analysis of normally distributed variables and Mann-Whitney U test for that of non-normally distributed variables. The level of statistical significance was set at $p < 0.05$. □

RESULTS

From the 1 085 patients with surgically treated IAs, 812 (74.8%) subjects had RIAs and 273 (25.2%) UIAs. Eighty-nine (32.6%) of UIAs patients were eligible to be included in the study. From them, 71 (79.8%) were females and 18 (20.2%) males. The mean age was 56.9 years old (21-78 years, SD 12.876).

Vertigo was present in 72 (80.9%) patients, headache in 41 (46.1%), visual symptoms in 21 (23.6%) and other symptoms in 23 (25.8%). Seventy aneurysms (78.7%) were found in the an-

FIGURE 2. Graph presenting the percentage of each aneurysm site found in our population, in an ascending scale

terior portion of the circle of Willis and the remaining ones 19 (21.3%) in the posterior circulation. The majority of aneurysms were discovered in the ACoA [24 (27.0%)] and PCoA [22 (24.7%)] (Figure 2).

From the 72 patients (80.9%) presented with vertigo, 54 had aneurysms in the anterior circulation (77.1% of the anterior circulation group) and

TABLE 1. Descriptives of patients with vertigo with regard to gender and circle of Willis

Gender		Vertigo		Total
		No vertigo	Vertigo	
Female	Circle of Willis Anterior circulation	14	41	55
		25.5%	74.5%	100.0%
	Posterior circulation	1	15	16
		6.3%	93.8%	100.0%
	Total	15	56	71
Male	Circle of Willis Anterior circulation	2	13	15
		13.3%	86.7%	100.0%
	Posterior circulation	0	3	3
		0.0%	100.0%	100.0%
	Total	2	16	18
Total	Circle of Willis Anterior circulation	16	54	70
		22.9%	77.1%	100.0%
	Posterior circulation	1	18	19
		5.3%	94.7%	100.0%
	Total	17	72	89
		19.1%	80.9%	100.0%

TABLE 2. Descriptives of patients with headache with regard to gender and circle of Willis

Gender		Headache	
		No headache	Headache
Female	Circle of Willis Anterior circulation	29	26
		52.7%	47.3%
	Posterior circulation	7	9
		43.8%	56.3%
	Total	36	35
		50.7%	49.3%
Male	Circle of Willis Anterior circulation	10	5
		66.7%	33.3%
	Posterior circulation	2	1
		66.7%	33.3%
	Total	12	6
		66.7%	33.3%
Total	Circle of Willis Anterior circulation	39	31
		55.7%	44.3%
	Posterior circulation	9	10
		47.4%	52.6%
	Total	48	41
		53.9%	46.1%

TABLE 3. Descriptives of patients with visual symptoms with regard to gender and circle of Willis

Gender		Visual symptoms	
		No visual symptoms	Visual symptoms
Female	Circle of Willis Anterior circulation	42	13
		76.4%	23.6%
	Posterior circulation	10	6
		62.5%	37.5%
	Total	52	19
		73.2%	26.8%
Male	Circle of Willis Anterior circulation	13	2
		86.7%	13.3%
	Posterior circulation	3	0
		100.0%	0.0%
	Total	16	2
		88.9%	11.1%
Total	Circle of Willis Anterior circulation	55	15
		78.6%	21.4%
	Posterior circulation	13	6
		68.4%	31.6%
	Total	68	21
		76.4%	23.6%

the remaining 18 had an aneurysm located in the posterior circulation (94.7% of the posterior circulation group) (Figure 2). However, no significant correlation between the circle of Willis (anterior circulation or posterior circulation) and vertigo ($p=0.084$), headache ($p=0.517$) or visual symptoms ($p=0.355$) was noted (Tables 1, 2, 3). Additionally, no significant correlation was found between gender and vertigo ($p=0.334$), headache ($p=0.225$) or visual symptoms ($p=0.163$).

A statistically significant correlation was observed between age and vertigo ($p=0.031$). Thus, the mean age was $55.5 (\pm 12.618)$ years old in patients with vertigo and $62.82 (\pm 12.621)$ years old in those with no vertigo. There was no statistically significant correlation between age and headache ($p=0.273$) or visual symptoms ($p=0.650$).

DISCUSSION

Headache and cranial nerve deficits or even ischemic cerebrovascular disease and epileptic seizures are amongst the most frequently reported clinical presentations of UIAs (1, 16). Over the last decades, several articles have mentioned vertigo as either an initial symptom or a coexisting symptom of UIAs (6-9).

Vertigo is described as a sensation of false movement concerning either the environment or the patient. Benign paroxysmal peripheral vertigo, vestibular neuritis, vestibular migraine, Meniere's disease, migrainous vertigo and acute labyrinthitis are common causes of vertigo (17, 18). Interestingly, in our study, vertigo was identified as a significant symptom in the recent medical history of 72 patients (80.9%). Nevertheless, other studies have not presented similarly high percentages, maybe because vertigo was not considered a characteristic symptom of UIAs and it might have therefore been either underestimated or attributed to another cause or mistaken with dizziness, nausea, faint, syncope, etc by patients or doctors. As regards the gender, we found that 71 (79.8 %) patients were women, which was in accordance with the literature, where UIAs are reported to be more frequent in women (about 66–70%) (1) (Figure 3).

Regarding UIAs location and vertigo, it is logical to consider that vertigo is mostly found in aneurysms of the vertebrobasilar circulation especially because the blood of the inner ear is supplied by the internal auditory artery origina-

FIGURE 3. Graph representing the percentages of subgroups concerning gender and circle of Willis with regard to vertigo

ting from the anterior inferior cerebellar artery (7, 8, 19, 20). However, several UIAs have been documented in the anterior portion of the circle of Willis (6, 10, 21-24). This supports our findings since it was not shown any significant correlation between aneurysm location (anterior or posterior circulation) and vertigo. Regarding the aneurysm sites, we found that 27% of them were located in the ACoA and 24.7% in the PCoA, contrary to the findings of Lawton *et al*, who suggested the MCA and ACoA were the two most common aneurysm sites (15).

The main theories explaining the pathophysiology of UIAs associated symptoms, which were described in the literature, concern the local, mechanical influence of aneurysm to the adjacent tissue. Thus, the size and location of the aneurysm are said to be the two important parameters for defining the symptomatology. However, in our study, we did not present any significant correlation between the localization of aneurysms and vertigo.

Vertigo has been linked with dissecting aneurysms of the basilar and vertebral artery leading to cerebral ischemia, subarachnoid hemorrhage, and neurological deficits (19, 25, 26). Additionally, it has been stated that some patients present aneurysmal SAH with atypical symptoms, including nausea or vomiting, dizziness or vertigo, and neck or back pain (27, 20). Even carotid artery stenosis has been linked with vertigo (28, 29).

Since several decades, hemodynamics of cerebral aneurysms has been an attracting site of re-

search in an attempt to examine the factors for aneurysm progression and rupture. Several factors have been found to be associated with an increased risk of rupture including anatomical features (aneurysm size, neck diameter, specific location), demographic characteristics (female gender, age over 60) and medical conditions (hypertension, smoking) (1, 4, 30, 31).

In order to better assess hemodynamics, dimensionless parameters such as Reynolds number have been employed in order to facilitate dynamic similitude (32). It has been proved that turbulence or temporal fluctuations were observed at the aneurysm site in the decelerating systole or near the end of systole, resulting in a high-frequency oscillation with Reynolds numbers of 877 (average) and 1 271 (at the systolic peak), which is in agreement with the critical Reynolds number for the onset of instability estimated at 975 (33). Indeed, Steiger *et al* have demonstrated *in vitro* that saccular aneurysms can locally disturb the laminar flow of the corresponding arterial branch at physiological Reynold's numbers (34). Lieber *et al* used an experimental flow apparatus to demonstrate that an aneurysmal vortex was formed inside the aneurysmal pouch during systole and projected into the parent vessel at diastole, perpendicularly to the vessel axis, leading to disturbances in the streamwise parent flow (32) (Figure 4). Castro *et al* used patient-specific models of IAs from 3D angiography images and found that the more laminar flow pattern within the parent vessel resulted in a less complex intra-aneurysmal flow pattern with fewer vortices (35). Furthermore, Stonebridge *et al* have recently established that the predominant arterial flow pattern was actually spiral laminar flow (35). These studies have done an in depth evaluation of intra-aneurysmal hemodynamics; however, they still cannot explain the existence of vertigo in our patients.

FIGURE 4. Illustration depicting the vortex inside the intracranial aneurysm and the retrograde flow, during the systolic deceleration phase

It is already known that patients with orthostatism, heart arrhythmia or hypovolemia can present vertigo. Given that their mean arterial blood pressure (MAP) is insufficient to maintain a normal cerebral blood flow (CBF), it may affect the cerebral circulation when it decreases below critical levels and patients start complaining about vertigo. Such symptoms are more characteristic of presyncope. However, we believe that a similar mechanism could also apply in the case of UIAs as these aneurysms could lead to insufficient blood flow that cannot be controlled by cerebral autoregulation.

Cerebral autoregulation is a homeostatic process that regulates and maintains CBF constant across a range of blood pressures. In healthy adults, the limits are between 50 and 150 mm Hg for cerebral perfusion pressure (CPP) or 60 and 160 mm Hg for MAP, where $CPP = MAP - \text{intracranial pressure (ICP)}$. This homeostatic mechanism ensures that, as MAP or CPP increases, resistance increases (vasoconstriction), on one hand, and when MAP or CPP decreases, we observe decreasing cerebrovascular resistance or vasodilation leading to increased cerebral blood volume (CBV), on the other hand. In case of impaired cerebral autoregulation, decreases in CPP lead to decreases in CBF (37).

Above mentioned studies have shown that during the diastole, near the end of the systole (at the decelerating phase), the vortex created inside the aneurysmal pouch projected perpendicularly towards the corresponding arterial branch and locally disturbed the spiral laminar flow of the parent artery by creating temporal fluctuations or even turbulence (36, 32-34).

Formation of irregular blood flow

Initially, the blood travels from the parent artery to a spiral laminar flow pattern. This means that blood is transported in parallel layers with no interruption amongst them and by presenting a homogenous flow. When the blood volume from the parent artery at the end of the systole (decelerating phase) reaches the neck of the aneurysm, it faces the retrograde projection of the vortex (created into the aneurysmal pouch during the accelerating phase of systole), which tries to protrude into the parent artery lumen in a counterclockwise pattern. At this time, the vortex protrusion has the maximum of the possibilities to occur since the blood flow of the parent artery is de-

creased. It should be noted that the blood flow inside the vessels is continuous and the decelerating blood volume from the parent artery and the blood volume from the vortex collide. As a result, the blood flow loses its streamwise pattern and becomes irregular, with a high-frequency oscillation and turbulence.

Decrease of efferent blood flow

This means that the layers of the blood are not parallel amongst them and they present different velocities and different directions creating tiny whirlpool regions. Consequently, the formation of turbulence increases the loss of energy in the form of friction and the blood flow at this specific site and timing (aneurysm-parent artery, end of systole-beginning of diastole) can be significantly decreased. In addition, this phenomenon could be probably aggravated by certain movements and positions of the head as well as by possible increased cerebrovascular resistance/vasoconstriction which may affect the local distribution of blood and its hemodynamics (35). It is important to mention that the aforementioned has been described for lateral saccular aneurysms which communicate with one arterial branch.

Unsuccessful cerebral autoregulation response

In reality, most aneurysms are found in bifurcations of the arteries. This makes us consider that the previously described decreased and unstable blood flow cannot sufficiently provide the efferent arteries of a bifurcation, found after the aneurysm. At least one of the two branches would probably be affected since the blood cannot follow both arterial routes in a homogenous flow. We can imagine that in cases where there are arterial branches near the neck of the aneurysm or even in the example of the basilar bifurcation aneurysm where the afferent blood flow from the parent artery collide face-to-face with the aneurysmal vortex. After these events, the blood flow in the affected artery (arteries) will be significantly decreased. If the diminution of the local blood flow is below a critical threshold, cerebral autoregulation does not have the necessary time or ability to respond. The flow returns to its original laminar state near the end of diastole.

Altered central processing

As regards the perception of stability, it is known that it is accomplished primarily by the balance

between the afferent signals received from the two vestibular organs. Hypothetically, the incapability of the above mentioned cerebral autoregulation process could temporarily create a mismatch in the CBV between the right and left part of the circle of Willis leading to unequal supply and, under certain circumstances, possible focal and temporal problems in the microcirculation in different parts of the brain. Indeed, studies describing the effects of ischemia and hypoxia on the brain underline the sensibility of the neuronal tissue in relation to these factors and suggest that changes in the polarization of the neuronal membrane are affected by blood flow and oxygen content (39). Thus, this abnormal hyperpolarization or depolarization could be translated to altered responsiveness of neuronal pathways, having as a result an imbalance among the circuits of vision, proprioception and vestibular systems. This imbalance could lead to altered central processing and false perception of movement (vertigo).

In our study, the no-vertigo group had a mean age of 62.82 (± 12.621) years old as compared to that of the vertigo group, which was 55.5 (± 12.618). Our results are close to the age limit for elderly people who tend to present cardiovascular changes, increased vascular resistance and higher MAP (40). This supports our theory. Thus, if the MAP is under the critical border, it cannot generate sufficient CPP and the CBF of the parent artery is more prone to be disturbed by the aneurysmal vortex. Castro *et al* have similarly stated that more laminar flow pattern within the parent vessel resulted in less complex intra-aneurysmal flow pattern with fewer vortices (35). Also, findings collected from animal and human experiments show that the vestibular system regulates blood distribution and arterial pressure during movement and changes in posture via vestibulo-sympathetic responses (38), which could act as a pathway of balance and possibly explain the fact that the symptom of vertigo is intermittent and not continuous.

Berge *et al* have mentioned that, before rupture, a cascade of hemodynamic events is responsible for stresses on the aneurysm wall, which triggers an inflammatory reaction and vascular remodelling (5). We observe that vertigo can be presented before, and immediately after, rupture as well as in acute conditions such as dissecting aneurysms or carotid artery stenosis, where new hemodynamic changes are observed. Thus, we could assume that vertigo could possibly be linked

to changing hemodynamic status and progression or remodeling of the aneurysm.

Limitations of the study

The high percentage of vertigo in our research could have been affected by the fact that we performed a retrospective study in which the registration of vertigo was performed by different doctors. In some cases, the symptom of vertigo could have been falsely interpreted by either the patient or doctor and it could have been mistaken for symptoms such as dizziness, faint, presyncope, etc. In order to overcome this, prospective studies using a precise and well-defined protocol should be implemented. Furthermore, we did not use any control group and the follow-up of the operated patients was not complete something that can not estimate a possible diminution of the vertigo frequency after surgical treatment, which could be really interesting to see in future studies. In any case, our study proves the implication of vertigo in UIA, because the percentage of vertigo in our sample is much higher than its incidence and prevalence in the general population. $\square$

CONCLUSIONS

Our data strongly indicate that UIAs in both males and females, and independently of their location, have vertigo as a dominant symptom. Whenever patients are investigated for vertigo with no other clinical explanation, they should be examined with at least a non-invasive angiography (CTA, MRA) in order to exclude an UIA. Further prospective studies with visualization of in vivo aneurysmal flow and quantification of hemodynamic characteristics in patients with vertigo and UIAs are needed. $\square$

Conflicts of interest: none declared.

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Ethical approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional research committee and the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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