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Aneurysmatic Subdural Haemorrhage: Brief Review

Patricia Bozzetto Ambrosi[1]

[1]Diagnostic and Interventional Neuroradiologist, Neurological Surgeon, Clinical Professor, Researcher and Neuroscientist, France

*Corresponding author: Prof. Dr. Patricia Bozzetto Ambrosi (patty.bozzetto@gmail.com)

Brief Review

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Abstract Acute subdural hemorrhage caused by a ruptured intracranial aneurysm is a well-known association but usually linked to dismal outcome. We briefly reviewed the clinical and imaging features, surgical management and outcome of 45 patients with subdural bleeding related to intracranial aneurysm. Forty-two reports were encountered in the literature between 1981-2015 and three additional patients were identified in the author personal archives. A total of three patients had bilateral acute subdural hemorrhage, and the epidural hematoma was not present in any of the 45 reports. Coma was identified in 32/45 (71%) patients at admission. Only seven of 45 cases reported were managed by endovascular approach. A good clinical outcome was observed in 23/45 (51%) and early mortality occurred in 10/45 (22%) of the patients analysed. Neurosurgical management has evolved tremendously in recent years leading to more comprehensive understanding about this association. Factors leading to natural history, mechanisms, imaging aspects and improvement of clinical results of aneurysmatic subdural bleeding merit further studies.

Keywords: Subdural Hemorrhage, Intracranial Aneurysm, Epidural Hematoma, Subarachnoid Haemorrhage, Intracranial Haemorrhage

1. Introduction

Aneurysmatic subdural hemorrhage is a not uncommon event (1.8%), but it has been associated with a dismal prognosis.[1-7] Aneurysm-related subdural hemorrhage was first reported by German physician and professor of special pathology Sir Karl Ewald Hasse in 1855 in his publication *Krankheiten des Nervensystems* (Diseases of the Nervous System).[8] Advancing age, an episode of sentinel headache before an index subarachnoid hemorrhage (SAH), an aneurysm originating from the posterior communicating artery and further intracranial hemorrhages on the initial CT scan are often associated with aneurysmal subdural hemorrhage. [1,2] although the natural history and mechanisms leading with aneurysm bleed has been not completely elucidated. Aneurysmatic subdural hemorrhage may be found in different intracranial locations, such as convexity, inter-hemispherical, tentorial, and intradural space, or even in the spinal subdural space, depending on the location of the aneurysm rupture.[9-14] However, in a few cases, the hematoma was reported to have occurred far from the point of aneurysmatic rupture.[12,15]

2. Methods

Five electronic databases: EMBASE, MEDLINE via PubMed, Web of Science, AMED Allied Medicine, and Europe PMC were searched. The following set of specific words were used to devise the search strategy: "subdural hemorrhage", " acute subdural hemorrhage", "subdural hematoma", "subdural haematoma", "subarachnoid hemorrhage", "intracranial

aneurysms", "aneurysmal subdural hematoma". Single and combined words were matched. Thesaurus MeSH and systematization of bibliographic searches were used to improve the search.

At Table 1 were arranged the patients'demographic data, clinical features (symptoms/signs), location and laterality of the aneurysm, characteristics of the aneurysmal subdural hemorrhage on CT scan, treatment and outcome were analyzed from each article separately and further results encountered.

Table 1. Clinical and Management Characteristics of Analysed Reported Cases with Aneurysmatic Subdural Hematoma

n	Author	Age (years)	Sex	Clinical	Aneurym	Subdural Hematoma Convexity Other	Treatment HE Clip/Coil	Outcome
1	Rengachary et al (1981) [6]	49	M	Confusion, Dysphasia	MCA	+ -	+ +/-	Good
2	Williams et al (1983) [28]	18	F	Coma	IC-PC	+ -	+ +/-	Disabled
3	O' Leary et al (1986) [5]	28	F	Coma	MCA	+ -	- -/-	Dead
4	Kondziolka et al (1988) [18]	43	M	Coma	IC-PC	+ Tentorium	+ +/-	Good
5	Kondziolka et al (1988) [18]	38	F	Coma	IC-PC	+ Tentorium	+ +/-	Disabled
6	Shinmura et al (1989) [29]	53	F	Coma	MCA	+ -	+ +/-	Disabled
7	Onda et al (1989) [14]	44	F	Coma	IC-PC	+ -	+ +/-	Disabled
8	Watanabe et al (1991) [7]	51	M	Coma	Distal ACA	+ Interhemispheric	+ +/-	Dead
9	Ragland et al (1993) [32]	27	M	Coma	AcomA	+ -	+ -/-	Dead
10	Eggers et al (1992) [33]	34	F	Headache	IC-PC	+ -	+ -/-	Good
11	Hatayama et al (1994) [34]	55	M	Coma	Distal ACA	+ Interhemispheric	+ +/-	Good
12	Hatayama et al (1994) [34]	66	F	Coma	Distal ACA	+ Tentorium/ Interhemispheric	+ +/-	Disabled
13	Ishibashi et al (1997) [35]	54	F	Headache	IC-PC	+ Tentorium	+ +/-	Good
14	Satoh et al (1999) [36]	58	F	Coma	IC-PC	+ -	+ +/-	Good

15	Satoh et al (1999) [65]	25	F	Headache	IC-PC	+ -	+ +/-	Good
16	Nonaka et al (2000) [37]	52	F	Coma	IC-PC	+ Tentorium	+ +/-	Good
17	Inamasu et al (2002) [38]	28	F	Coma	IC-PC	+ -	+ -/-	Dead
18	Araki et al (2002) [39]	55	F	Headache, ptosis, Coma	IC-PC	+ -	+ +/-	Good
19	Agakhani et al(2002) [40]	56	M	Headache	AcomA	+ -	+ -/-	Dead
20	Agakhani et al(2002) [40]	28	M	Headache	IC-PC	+ -	+ +/-	Good
21	Agakhani et al(2002) [40]	39	F	Headache	IC-PC	+ -	- -/+	Good
22	Agakhani et al(2002) [40]	46	M	Coma	MCA	+ -	+ +/-	Dead
23	Blake et al (2003) [24]	35	F	Coma	IC-PC	+ -	- -/-	Dead
24	Katsuno et al (2003) [41]	63	F	Headache, nausea, dizziness	Distal ACA	+ Interhemisp heric	+ +/-	Good
25	Koerbel(2005) [42]	62	F	Headache, Coma	Bifurcation of ICA	+ -	+ +/-	Good
26	Boujemaa(2006) [26]	44	F	Coma	Left IC-PC	+ Bilateral	Not mention	-
27	Triantafyllopou(2006) [21]	65	F	Coma	Left cavernous ICA	+ -	+ -/-	-
28	Ishikawa (2009) [43]	45	M	Coma	ICA dorsal wall carotid left	+ Bilateral Left frontal	+ -/-	Dead
29	Sorensen (2009) [17]	53	F	Neck Pain and Headache	Pericallosal artery	+ -	+ +/-	Good
30	De Blasi(2010) [44]	47	F	Coma Left VI NC	Left IC-PC	+ Bilateral	- -/+	Good
31	Weil et al(2010) [45]	51	F	Coma	Distal ACA	+ -	+ +/-	Disabled
32	De Blasi(2010) [44]	60	F	Headache Right VI NC	Right MCA	Bilateral	- +/-	Good

33	Field(2010) [47]	33	M	Headache	MCA	+ -	No mention	-
34	Marbarcher (2010) [4]	27	F	Coma	Pericallosal right	+ Interhemispheric	+ -/-	Dead
35	Marbacher(2010) [4]	44	F	Coma	Pericallosal right	+ -	+ +/-	Good
36	Marbacher(2010) [4]	50	F	Left-side hemiparesis	Right MCA	+ -	+ +/-	Disabled
37	Marbacher(2010) [4]	39	M	Coma	Right PC-IC	+ -	+ +/-	Disabled
38	Marbacher(2010) [4]	58	F	Coma	Right MCA	+ -	+ +/-	Good
39	Marbacher(2010) [4]	45	F	Coma	Right PC-PC	+ -	+ +/-	Disabled
40	Marbacher(2010) [4]	68	F	III paresis	Right Distal PC-IC	+ -	+ +/-	Good
41	Case 1 (2010) [46]	43	F	Epileptic status	Carotid siphon	+ -	+ -/+	Dead
42	Case 2 (2011)	58	F	Coma	MCA	+ -	+ -/+	Good
43	Case 3 (2011)	56	M	Coma	MCA	+ -	+ -/+	Good
44	Mrfka (2013) [51]	47	F	Left-sided headache	Left PC-IC	+ -	+ -/+	Good
45	Gong(2014) [48]	43	M	Headache	Left MCA	+ -	+ +/-	Good

M-male, F-female, ACA- anterior cerebral artery, MCA- medial cerebral artery, IC-PC- internal carotid-posterior communication junction, HE- haematoma evacuation

3. Results

There were four hundred eight articles founded. Due to relative rarity of the pathology and consequently lack of meta-analyses or randomized controlled trials, authors considered reported cases and cases series written in French, English, Portuguese, German, Danish, Japanese and Korean (total of 42 reported) and three cases from their personal experience of the author.

The mean age of patients reported was ± 46 years (range, 18-68 years) and 32/45 (71 %) were women. Coma was found in 71% (32/45) of cases. Eleven out of 45 patients had a headache at admission, 4/45 (8%) had mental confusion, (one case) oculomotor paresis, (one case) epileptic status were also reported. Other accompanying symptoms were dizziness, nausea, and vomiting.

The patients harbored intracranial aneurysms in several locations. There was only one patient with multiple aneurysms (one of our cases). The aneurysms were predominantly located at the internal carotid artery and posterior communicating artery junction (IC-PC) in 21/45 patients (46.6%), followed by 9/45 (20%) in MCA, 8/45 (17.7%) in anterior cerebral artery(ACA) one in distal ACA and two cases located in the pericallosal artery, 2/45 (4.4%) in carotid siphon (1/45 in the dorsal wall of the carotid artery and the other in the cavernous segment). We reported the first case located in the ophthalmic segment of the carotid siphon. (Figure 1A-D). In none of the reported cases has an aneurysm been located in the posterior circulation. Three cases of bilateral aneurysmal subdural hemorrhage were reported. None of the 45 patients showed epidural hematomas. Most of the cases of aneurysmatic subdural hemorrhage were located in the brain convexity, and in four cases there was an extension to the tentorial site or to the interhemispheric fissure.

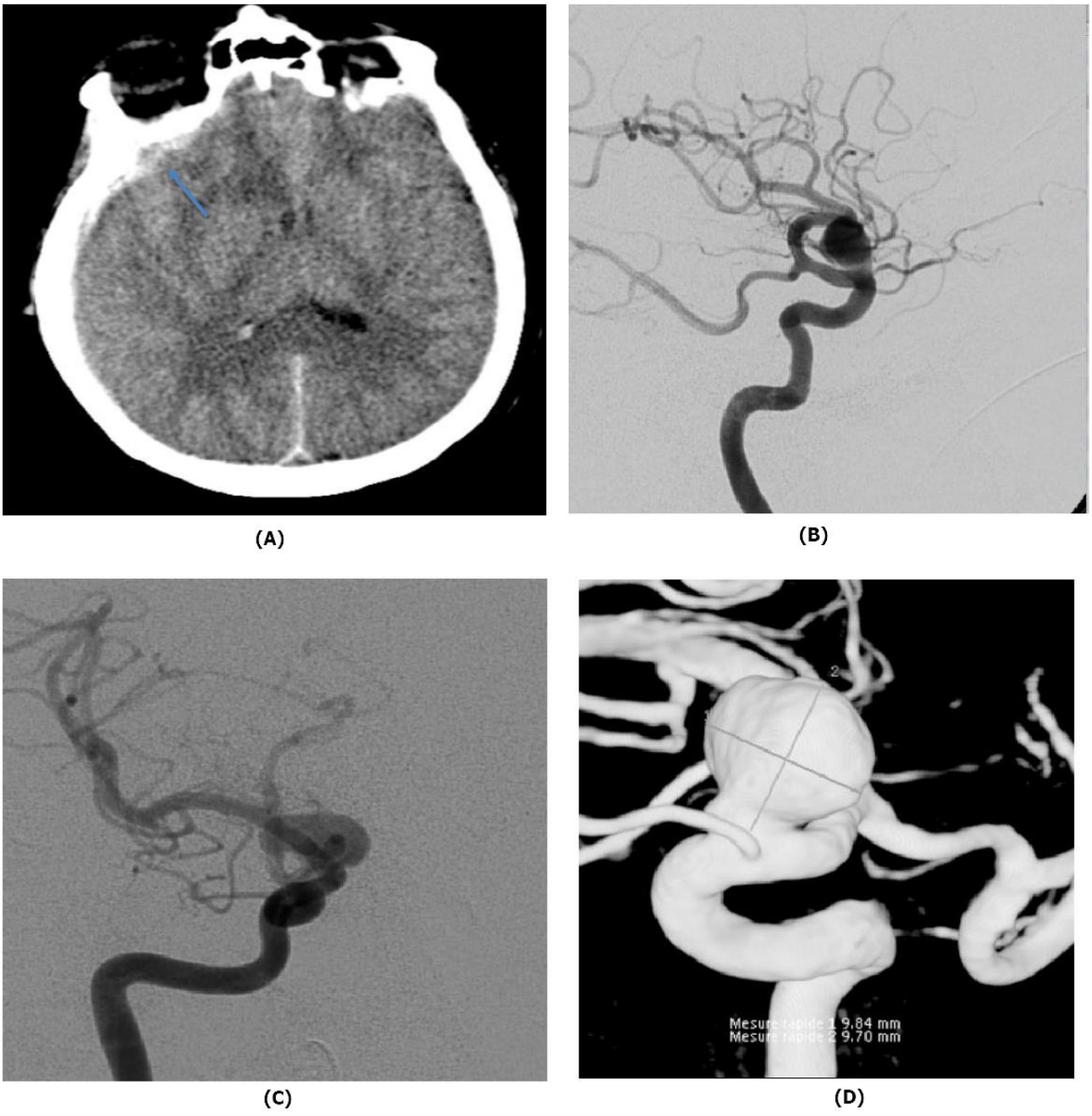

Figure 1. 1a (Axial CT image showing subdural hematoma that extends to frontal basal and temporal cortical surfaces), 1b, 1c, 1d (Right internal carotid angiograms lateral and anteroposterior projections and tridimensional reconstruction demonstrating the aneurysm arising from the ophthalmic segment of ICA

Conventional surgical treatment in almost all cases of the aneurysm was performed by hematoma evacuation and clipping with good results (1981-2005). Mortality occurred in three cases with no mention of the treatment used. Endovascular

coiling started to be performed alone if the aneurysmal subdural hemorrhage was clinically well tolerated or without significant midline shift after surgical evacuation of the aneurysmal subdural hemorrhage and has been described as the treatment of choice since 2002. Seven of 45 cases received endovascular treatment for their ruptured intracranial aneurysm. Data regarding outcome were available in 43/45 (95%) patients. Half the patients (23/45) had a good clinical outcome, ten patients remained disabled and nine patients died.

4. Discussions

The frequency of aneurysm-related subdural hematoma is estimated to be 0.5-8% of cases of intracranial bleeding based on case reports, small clinical series, and autopsy series.[1,2,18,19,20,21] The majority of case reports describe aneurysmal rupture resulting from the posterior communicating artery or IC-PC aneurysms.[1,2,20]

However, Clarke & Walton who divided intracranial vascular anomaly with a complicating subdural hemorrhage into three groups (Group I, II and III), depending upon the amount of clot present and clinical course as illustrated in Table 2. As group I - All posterior circulation aneurysms were included, in other words, large and resulting in death within 1-72 hours of onset.[22] Vertebrobasilar or posterior circulation is less cited probably because subdural collection in the posterior fossa is overwhelming and rapidly fatal as frequently demonstrated by autopsy cases.[22,23]

Table 2. Clarke & Walton, 1953 – Clinical and Outcome Classification of Subdural Hematoma caused by vascular anomaly

Group	Hemorrhage	Clinical	Outcome
I	Massive	-	Rapidly Fatal
II	Insignificant	-	-
III	-	Significant	Not Fatal

One of the likely causes of subdural hemorrhage encountered in association with ruptured intracranial aneurysm is thought to be due to head trauma, which may cause disruption of superficial cerebral or cortical bridging veins. Arteriovenous malformations, cocaine abuse, dural metastasis, coagulopathy, falx meningioma and rupture of a cortical artery located near the Sylvian region are other likely causes.[24,25] Several risk factors have been described, such as sex, age, smoking, hypertension, history of SAH, sentinel headache, a location of the rupture aneurysm, intracerebral hemorrhage and IC-PC aneurysm. Finally, among these associated factors age, the location of the aneurysm, sentinel headache and presence of intraventricular hemorrhage seems to be determinant and have been well described as high risk of an aneurysmatic subdural hematoma by Biesbroek. [1,2]. The same author also suggested that aneurysm anatomy, perianeurysmal environment and extension of subdural hemorrhage probably are probably also important factors that might well need further studies.[1,2]

Two main circumstances among other mechanisms underlying aneurysm-related hemorrhage are hypothesized (i) on bleeding directly into the subdural space, in which case an aneurysm might be projected from the surface of the brain through the leptomeninges and rupture in the subdural space; or (ii) bleeding into the subdural space via the subarachnoid space.

Other possibilities are less cited: (iii) sentinel hemorrhages might cause the adhesion of an aneurysm to the adjacent arachnoid membrane, and the final rupture occurs into the subdural spaces; (iv) the stream of blood may rupture through the arachnoid membrane at some distant weak point; (v) secondary to decompression of an intracerebral hematoma into the subdural space following disruption of the arachnoid covering the cerebral cortex; (vi) when an aneurysm may penetrate the arachnoid mater transfixing and entering the subdural space, before its rupture and where the vessel passes through the subdural space (small cortical artery located at the Sylvian fissure) on its way to the circle of Willis it may cause direct blood flow into the subdural space.[31] At Figure 2 and Figure 3, we demonstrated two giant MCA aneurysms (our experience) which adhered to the dura mater of an anterior clinoid process and the proximal side of plica petroclinoid anterior.

Several locations have been mentioned as potential sites where aneurysmal subdural hemorrhage associated without SAH, may be found. All such cases exhibited blood over the convexity and in other locations: seven cases showed blood tracking along the tentorium, blood was seen within the interhemispheric fissure in five cases, one case had aneurysmatic subdural hemorrhage along the dorsal aspect of the clivus, as well as the tentorium and convexity, and along the diaphragma sellae, migrating down into the spinal canal. Bilateral locations of subdural aneurysmatic hemorrhage are more rare.[26, 27]

Simple assessments based on neuroradiological findings may differentiate aneurysmatic subdural hemorrhage associated with intracranial aneurysm from those in which it is secondary to head trauma. For example, continuity between a convexity subdural hematoma and an interhemispheric hemorrhage could be related to a ruptured aneurysm of AcomA, and continuity between a tentorial hematoma and interhemispheric acute subdural hematoma may indicate a ruptured aneurysm of the internal carotid and IC-PC.[27,28] All patients who present acute subdural hematoma at admission could also be evaluated by neuroimaging examination such as CT angiography (CTA), Magnetic resonance angiography (MRA) or conventional angiography to exclude not only aneurysm, but other conditions such as arteriovenous malformations (MAV) or arteriovenous (AV) fistulas.[4]

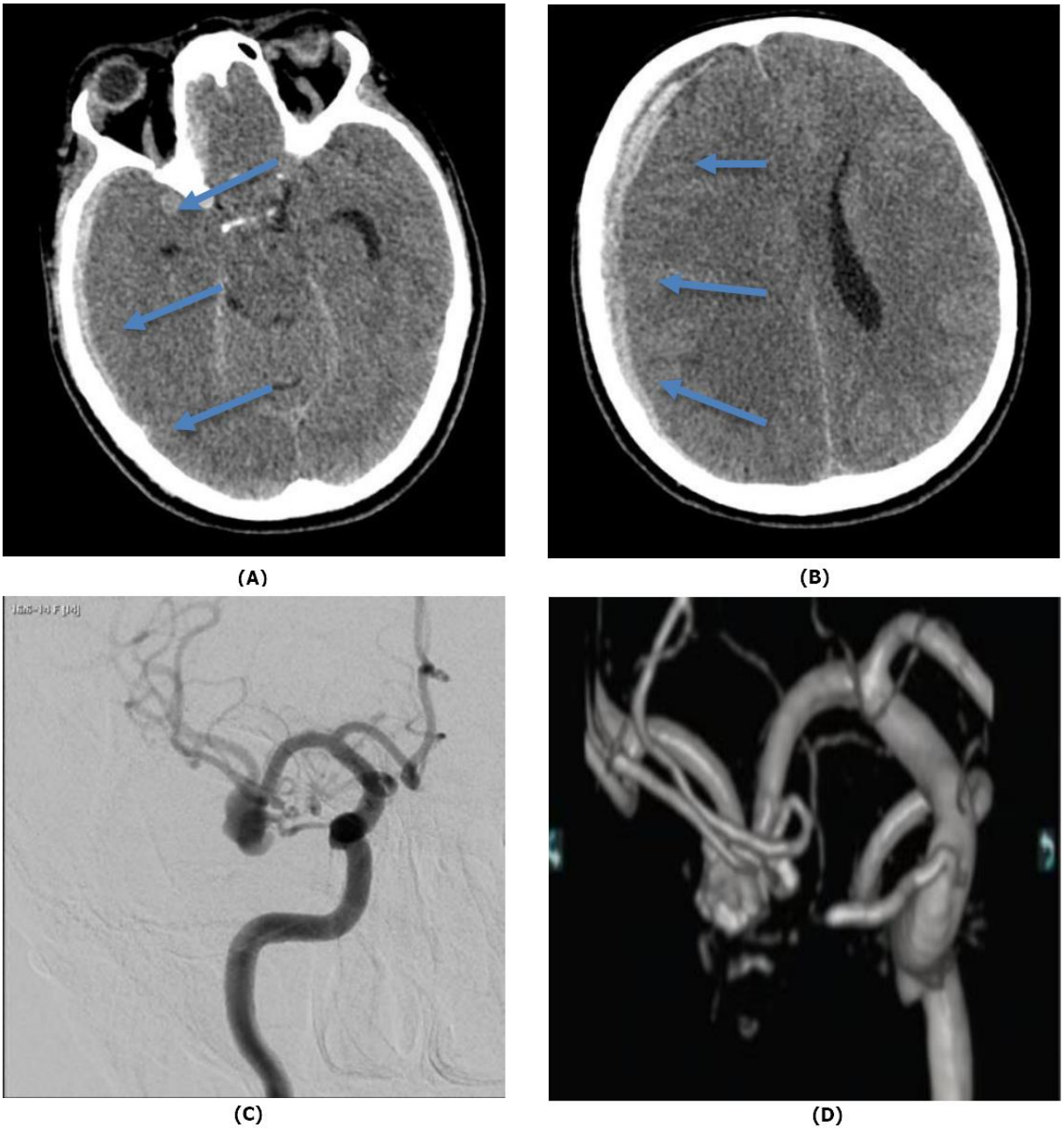

Figure 2. 2a, 2b (Axial CT images showing subdural hematoma that extends to temporal cortical surface), 2c, 2d (Right internal carotid angiograms anteroposterior projection and tridimensional reconstruction demonstrating the aneurysm arising from the MCA segment of ICA)

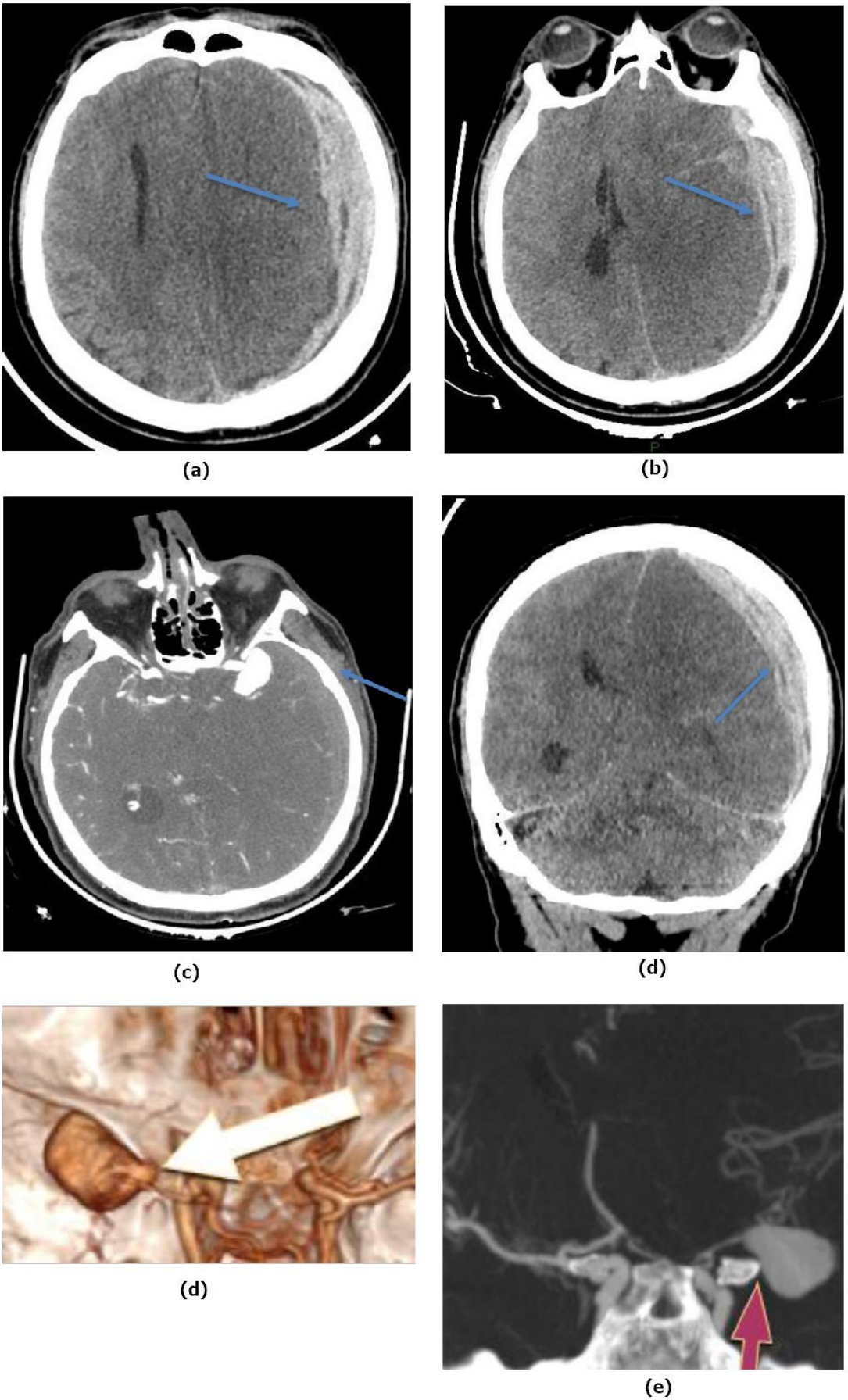

Figure 3. 3a, 3b (Axial CT images showing subdural hematoma that extends to fronto-temporo-parietal cortical surface), 3c (tridimensional CT reconstruction demonstrating the aneurysm arising from the segment of ICA, arrow pointing the giant MCA aneurysm), 3d (Coronal CT images showing subdural hematoma), 3e (3D images show aneurysm extending to anterior clinoid surface), 3f (3D with MCA aneurysm)

Additional tools such as 3-D CTA and VasoCT® might provide a high resolution assessment of the relative composition of the aneurysm and vessels (presence of thrombus or vessel dissection) and delineation of some anatomical aspects, perianeurysmal environment and extent of the hemorrhage.[6,7]

Based on our reviewed data (Table 1), surgical management in almost all cases was hematoma evacuation and clipping, with good results (1981-2005), and only three cases with death or no treatment. Endovascular management with or without hematoma evacuation through craniotomy has been done in seven patients. In the particular patients, coiling treatment, as well as hematoma evacuation, had produced favorable outcome.

The initial management depends on the clinical presentation of the acute subdural hematoma, on the volume of hematoma and on the mass effect that it may cause. Besides, the timing of surgical evacuation of hematoma is still a controversial issue. If a rapid clinical deterioration occurs, an emergency evacuation of hematoma should be performed before further investigations. Also, in the clinical worsening scenario, only subdural hematoma evacuation without an aneurysm-directed treatment (surgical clipping and / or endovascular coil embolization) will cause rapid decrease in intracranial pressure and intramural pressure in the aneurysm wall, and thus can increase risk of aneurysmal subarachnoid hemorrhage and further severe cerebral vasospasm. This situation ultimately complicates surgical clipping and reduces the success of surgical treatment. Within the indication, it seems more likely that early surgical clipping in the same session will increase the success of the treatment in these cases, concurrently with the early subdural hematoma evacuation as much as possible. Based on the review cases, endovascular coiling of ruptured intracranial aneurysm was preferred if subdural hemorrhage was clinically well tolerated or after surgical evacuation of acute subdural hematoma and has been described as the treatment of choice for ruptured aneurysm with subdural hemorrhage since 2005 according to reviewed cases.

New tips on the treatment strategy for aneurysmal subdural hematoma in the endovascular era are methodologically difficult to analyze, since open surgical management involves irrigation and removal of blood products has their unique indications depending on the clinical presentation and the strategy of the treatment aneurysm. New endovascular treatment strategies that aim to interrupt this cycle and tip this balance back in favor of resorption of hemorrhage as endovascular therapies aim to devascularize these membranes are already possible and need to be further investigated. [55] As well as other medical therapies, including the reduction of the micro-hemorrhage rate of the dural membranes, changes in the osmotic environment or changes in angiogenesis, also need to be further studied.

The treatment strategy for ruptured intracranial aneurysms has been decided by the character, shape, size and location of an aneurysm like any other kind of ruptured aneurysm depending by each department protocol.

In conclusion, this study suggests that aneurysmatic subdural hemorrhage due to ruptured aneurysms is quite uncommon and are more frequently seen related to anterior circulation aneurysms. A high degree of clinical suspicion is still important in the initial management of acute hematoma associated with intracranial aneurysm, which may permit an early diagnosis and appropriate treatment. Half of the cases had a good clinical outcome. Futures studies might target underlying mechanisms related with aneurysm-related hemorrhage and factors which influence the clinical and surgical outcome.

Conflicts of Interest/Disclosures

The authors declare no relevant financial or other conflict of interest or disclosures in relation to this paper.

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Abbreviations

M-male, F-female, ACA- anterior cerebral artery, MCA- medial cerebral artery, IC-PC- internal carotid-posterior communication junction, HE- haematoma evacuation, SAH- subarachnoid haemorrhage

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