Original Article

Access this article online

Quick Response Code:

Website: http://www.braincirculation.org

DOI: 10.4103/bc.bc_25_23

Reversible intracranial contrast medium accumulation after embolization of unruptured cerebral aneurysms and its association with transient neurological deficits: A single center experience

Jenna Schellin*, Alexander Neumann*, Peter Schramm, Björn-Hergen Laabs[1], Jan Küchler[2], Hannes Schacht

Department of Neuroradiology, University Medical Center Schleswig-Holstein, Lübeck, Germany, [1]Institute of Medical Biometry and Statistics (I. R. K.), University of Lübeck, Lübeck, Germany, [2]Department of Neurosurgery, University Medical Center Schleswig-Holstein, Lübeck, Germany

Address for correspondence:
Dr. Hannes Schacht, Department of Neuroradiology, University Medical Center Schleswig-Holstein, Ratzeburger Allee 160, 23538 Lübeck, Germany. E-mail: hannes.schacht@uksh.de

*Jenna Schellin and Alexander Neumann contributed equally to this work and shared first authorship.

Submission: 08-04-2023
Revised: 10-06-2023
Accepted: 18-06-2023
Published: 27-09-2023

Abstract:

BACKGROUND: Use of iodine-containing contrast medium (CM) is obligatory for endovascular treatment (EVT) of cerebral aneurysms. After EVT, intracranial density increases (DIs) can be detected in cranial computed tomography (CT). Those DI can correspond to subarachnoid hemorrhage (SAH), infarction or reversible CM accumulation (RCMA). The latter can be mistaken for hemorrhage, especially if they are accompanied by neurological deficits.

OBJECTIVE: To analyze postinterventional DI after EVT of unruptured cerebral aneurysms and associated clinical symptoms and to identify risk factors for the occurrence of RCMA.

METHODS: For differentiation of DI, we compared CT scans following EVT and additionally 24 h ± 5 h later. Diagnosis of RCMA was based on marked regression of DI on follow-up scans. We analyzed continuous variables (age, duration of intervention and anesthesia, aneurysm diameter, amount of CM and renal function) and categorial variables (gender, aneurysm location, devices for EVT, antiplatelet therapy [APT] and associated neurological deficits) to identify risk factors for the occurrence of RCMA.

RESULTS: We studied 58 patients (44 female, mean age 59.5 [range 39–81]) who underwent EVT for a total of 68 cerebral aneurysms in 62 therapy sessions over a 3-year period without periprocedural complications. Postinterventional DI occurred after 17 therapy sessions. All 17 DI turned out to be RCMA in the follow-up imaging. Two patients who had no DI on initial postinterventional CT showed new SAH on follow-up CT. Infarctions were not observed. Transient neurological deficits occurred in eight patients (12.9%) and were associated with RCMA ($P = 0.010$). Postinterventional RCMA was associated with the duration of EVT ($P = 0.038$) and with APT (acetylsalicylic acid [ASA] + clopidogrel: $P = 0.040$; ASA alone: $P = 0.011$).

CONCLUSIONS: RCMA is common after EVT of unruptured cerebral aneurysms and often accompanied by transient neurological deficits. Long procedure duration and APT appear to predispose to the occurrence of RCMA.

Keywords:

Aneurysm embolization, cerebral aneurysms, computed tomography, contrast medium accumulation, subarachnoid hemorrhage

How to cite this article: Schellin J, Neumann A, Schramm P, Laabs BH, Küchler J, Schacht H. Reversible intracranial contrast medium accumulation after embolization of unruptured cerebral aneurysms and its association with transient neurological deficits: A single center experience. Brain Circ 2023;9:178-84.

Introduction

The evolution of endovascular treatment (EVT) of cerebral aneurysms in recent years is accompanied by the increasing use of anticoagulant and antiplatelet drugs with a potential increased risk of periinterventional hemorrhagic complications, which emphasizes the importance of postinterventional imaging. Postinterventional intracranial density increases (DIs), seen on computed tomography (CT) scans were reported following EVT and other endovascular procedures with administration of iodine-containing contrast medium (CM). These turned out to be reversible CM accumulations (RCMA).[1-3] Several other terms have been used to address this phenomenon, such as "contrast enhancement hyperdensity," "focal increased cortical density," "subarachnoid contrast enhancement," "blood-brain barrier (BBB) disruption," "reversible gyral contrast enhancement" and "hyperattenuated intracerebral lesions."[1-8] In the context of associated neurological deficits, the terms "contrast-induced encephalopathy" and "contrast neurotoxicity" are found in the literature.[9-11] Postinterventional RCMA can be mistaken for subarachnoid hemorrhage (SAH), especially if it is accompanied by neurological symptoms, which may lead the treating physician to consider stopping antiplatelet therapy (APT). We aimed to investigate postinterventional DI after uneventful EVT of unruptured cerebral aneurysms, for the first time with the inclusion of new embolization devices and with focus on clinical symptoms associated with such imaging findings, and to identify risk factors for the occurrence of RCMA.

Methods

Patients

Approval of our institutional ethics committee was obtained (registry number: 20-211). We retrospectively studied 58 consecutive patients who underwent EVT of unruptured cerebral aneurysms at our institution from January 2017 to January 2020 without periprocedural complications and who received a flat-panel or multi-slice CT scan immediately after the procedure as well as follow-up CT scan 24 ± 5 h later. Patient age, gender, APT, anticoagulation, glomerular filtration rate (GFR), aneurysm characteristics, duration of procedure and anesthesia, amount of given CM, used embolization materials and clinical findings after treatment were recorded. Following current international guidelines, a GFR of <60 ml/min/1.73 m² was considered to represent impaired renal function.[12] For aneurysm diameter analysis, the sum of the maximal diameters of the individual aneurysms was used when multiple aneurysms were treated simultaneously.

Postinterventional computed tomography: Density increases and definition criteria for subarachnoid hemorrhage/reversible contrast medium accumulation

All CT scans were retrospectively assessed by consensus by two experienced neurointerventionalists with board certification. DIs were anatomically assigned to the brain region and classified into "focal" and "diffuse" according to their distribution pattern. In addition, DI were classified according to their location as superficial along the brain surface and parenchymal. We also documented whether DI occurred downstream of the treated vessel segment. If DI occurred at sites other than the treated vascular territory, they were classified as "distant." Follow-up CT was used to differentiate whether DI represented SAH or RCMA. Complete or marked regression of DI in follow-up CT was used to define RCMA. Constant or minimal regression of DI in follow-up CT was used to define SAH.

Statistics

Descriptive statistics were calculated for the variables of interest. Categorical data are presented as counts and percentages and continuous data are presented as means and ranges with standard deviations (SDs). Risk factors associated with RCMA were examined with multiple logistic regression analysis. These results are given as descriptive *P* values and odds ratios (OR) with 95% confidence intervals. All analyses were carried out using SPSS 24.0 (IBM; Armonk, NY (USA)). $P < 0.05$ were considered as significant.

Results

Patients and aneurysm characteristics

Of the 58 consecutive patients 44 were female (75.9%) and 14 male (24.1%). Their age ranged from 39 to 81 years (mean 59.5, SD 9.9). A total of 68 aneurysms were treated in 62 therapy sessions, all with no periprocedural complications. In 5 cases, two aneurysms were treated in one therapy session. Twenty-four aneurysms (35.3%) were located at the middle cerebral artery (MCA), 21 (30.9%) at the internal carotid artery or posterior communicating artery, another 14 (20.6%) at the anterior cerebral artery and 9 (13.2%) vertebrobasilar. Aneurysm diameters ranged from 2 to 17 mm (mean 7.2 mm, SD 3.0). With regard to gender, no significant association was found with the occurrence of RCMA.

Imaging findings

After 17 of 62 therapy sessions (27.4%), DI were observed in postinterventional CT. Following EVT, a flat panel CT was performed in most cases overall (48/62) respectively in the group of patients with DI (15/17). In the other cases, multislice CT was performed. Postinterventional

DI occurred in all cases superficially along the brain surface, mostly downstream of the treated vessel segment (94.1%) and with a diffuse distribution pattern (76.5%). All of these 17 DI regressed markedly or completely, so they were scored as RCMA. In one case an additional parenchymal DI was observed downstream in the left centrum semiovale with resolution in follow-up imaging. In two patients in whom no DI was detectable on initial postinterventional CT, new superficial hyperdensities appeared on follow-up imaging. Based on the above-mentioned definition criteria, these corresponded to SAH. A detailed description of the cases with postinterventional DI can be found in Table 1. See examples of the CT findings in Figure 1.

Neurological deficits after aneurysm embolization

Subsequent to the 62 EVT, eight patients (12.9%) showed new neurological deficits, which in all cases completely regressed during their hospital stay. Four patients had multiple symptoms (one patient had 3 symptoms simultaneously, three patients had 2 symptoms simultaneously). In particular, neurological deficits occurred in six patients (35.3%) in connection with RCMA (one patient had a seizure, aphasia and hemiparesis simultaneously). Neurological deficits were significantly more frequent in patients with RCMA (P = 0.010). In almost all patients with RCMA and localizable neurological deficits (1 × aphasia, 2 × hemiparesis, 2 × neglect, 1 × paresthesia), RCMA were ipsilateral to the clinically affected hemisphere. In one case with temporary visus reduction on the right side after coiling of a large aneurysm of the right A1 bifurcation and aplasia of the left A1 segment, RCMA occurred in the left frontal and right parieto-occipital region. In this case, additional RCMA of the optic chiasm or the right optic nerve could neither be proven nor excluded because of coil-related artifacts. In addition to neurological deficits, headache (n = 18; five patients with RCMA, 13 without RCMA) and nausea/vomiting (n = 6; three patients with RCMA, 3 without RCMA) were observed. Table 2 summarizes the neurological deficits.

Used devices

The most frequently used method for EVT was stent-assisted coiling in 27 cases (39.7%), with the Neuroform Atlas stent (Stryker Neurovascular, Fremont, California, USA) used in the majority of cases (n = 24), followed by the LEO Baby stent (Balt, Montmorency, France) in two cases and a combination of both stents in one case. Fifteen EVT (22.1%) were performed solely with coiling. Fifteen procedures (22.1%) involved the use of an intrasaccular flow disruptor (IFD; WEB-Device [Terumo, California, USA]; [n = 14], or Contour-Device [Cerus Endovascular, Fremont, California, USA]; [n = 1],). Six EVT (8.8%) were performed with a flow diverter stent (FDS; p64 [Phenox, Bochum, Germany]; [n = 3], Pipeline Flex [Medtronic, Dublin, Ireland]; [n = 2], Surpass Evolve [Stryker Neurovascular, Fremont, California, USA]; [n = 1]). In 5 cases (7.4%) different devices were combined for embolization [Table 3]. No significant association was found between a specific embolization material and the occurrence of RCMA.

Duration of intervention and anesthesia

The mean procedure duration of EVT was 76.3 min (range 16–200 min; SD 38.3). In this context, a

Table 1: Detailed description of all cases with postinterventional density increase, which all turned out to be reversible contrast medium accumulation

Treated CA	Device used for EVT	Location of DI	Additional parenchymal DI	Diffuse/ focal DI	Downstream/distant of treated aneurysm
MCA right	SAC	Parietal right		Focal	Downstream
ACA (A1) right, giant aneurysm	Coils	Frontal and parietal left		Focal	Distant
Distal ICA left	Coils	Frontotemporoparietal left		Diffuse	Downstream
MCA left	SAC	Frontoparietal left		Diffuse	Downstream
Distal ICA right	FDS (p64)	Frontoparietal right		Focal	Downstream
ACOM	IFD (WEB)	Frontoparietal left		Diffuse	Downstream
MCA right	IFD (WEB)	Frontoparietal right		Diffuse	Downstream
MCA right	SAC	Frontoparietal right		Diffuse	Downstream
ACOM	IFD (WEB)	Bifrontal		Diffuse	Downstream
2× MCA right	2× IFD (WEB)	Frontotemporoparietal right		Diffuse	Downstream
MCA left	IFD (contour)	Frontotemporoparietal left		Diffuse	Downstream
Distal ICA left	IFD (WEB)	Frontoparietal left		Diffuse	Downstream
Distal ICA right	IFD (WEB)	Frontal right		Focal	Downstream
Pericallosal artery left	SAC	Frontotemporoparietal left		Diffuse	Downstream
2× distal ICA left	FDS (surpass evolve)	Frontoparietal left		Diffuse	Downstream
Distal ICA right, giant aneurysm	SAC	Frontoparietal right		Diffuse	Downstream
MCA left	WEB + stenting	Frontoparietal left	+	Diffuse	Downstream

ACA: Anterior cerebral artery, ACOM: Anterior communicating artery, CA: Cerebral aneurysm, DI: Density increase, EVT: Endovascular aneurysm treatment, FDS: Flow-diverter stent, IFD: Intrasaccular flow disruptor, ICA: Internal carotid artery, MCA: Middle cerebral artery, SAC: Stent-assisted coiling, WEB: Woven EndoBridge

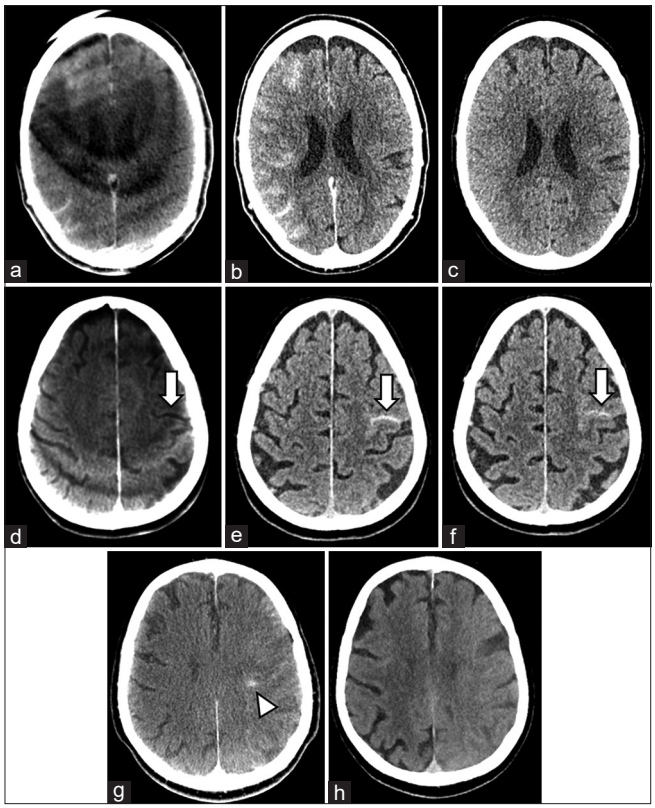

Figure 1: Example cases. Case 1: Flat panel computed tomography (CT) of a 74-year-old patient (a) immediately after embolization of two right-sided MCA aneurysms with WEB-Devices shows superficial densities along the right cerebral hemisphere. After extubation, the patient showed left neglect, left hemiparesis, and aphasia. Therefore, 40 min later a multislice CT scan (b) was performed, showing no further findings. Follow-up CT 18 h later (c) shows marked regression of the densities, consistent with reversible contrast medium accumulation (RCMA). The neurological deficits disappeared. Case 2: Postinterventional flat panel CT of a 65-year-old patient (d) after stent-assisted coiling of a left-sided paraophthalmic internal carotid artery aneurysm shows no abnormalities. CT the following day (e) showed a new sulcal density frontally on the left side (arrows) with only mild regression after 4 days (f), consistent with subarachnoid hemorrhage. The patient did not show any neurological deficits at any time. Case 3: Flat panel CT of a 45-year-old patient (g) after treatment of a left-sided MCA aneurysm with a WEB Device and stenting shows superficial densities on the left side and in addition a focal parenchymal density in the left centrum semiovale (arrowhead). Both findings have regressed markedly in the CT the next day (h), consistent with RCMA. The patient had transient motor aphasia, mild right arm paresis and a generalized seizure

significant difference was found between patients with RCMA (mean 93.7 min; range 35–200 min; SD 54.5) and without RCMA (mean 69.8 min; range 16–147 min; SD 28.2) (P = 0.038). The mean duration of anesthesia for EVT was 148.3 min (range 63–320 min; SD 50.6). Anesthesia durations did not differ significantly between patients with RCMA (mean 165.4 min, range 85–320 min; SD 71.9) and without RCMA (mean 141.8 min; range 63–227 min; SD 38.9).

Amount of given contrast medium

Iodine-containing CM (Imeron, Bracco Imaging Deutschland GmbH) was administered intra-arterially for EVT with iodine concentrations of 300 mg/ml, 350 mg/ml or 400 mg/ml. For standardization, the pure iodine quantity was calculated in mg for statistical analysis. Overall, a mean of 49,141 mg iodine (range 28,000–132,500 mg; SD 18,999) was applied. In the patient group with RCMA, the amount of CM was higher (mean 54,971 mg; range 28,000–132,500 mg; SD 26,756) than in the group without RCMA (mean 46,939 mg; range 30,000–80,000 mg; SD 14,899), but without significant difference.

Table 2: List of neurological deficits after aneurysm embolization

Neurological deficit after EVT	Overall	With RCMA	Without RCMA
Generalized seizure	2	1	1
Aphasia	3	1	2
Visus reduction	1	1	0
Hemiparesis	3	2	1
Neglect	2	2	0
Paresthesia	1	1	0
No adequate waking response	1	1	0

CM: Contrast medium, RCMA: Reversible CM accumulation, EVT: Endovascular aneurysm treatment

Table 3: Devices used for aneurysm embolization

Devices	*n* (%)
SAC	27 (39.7)
Coiling	15 (22.1)
Intrasaccular flow disruptor	15 (22.1)
Flow diverter stent	6 (8.8)
Flow diverter stent + coiling	2 (2.9)
Intrasaccular flow disruptor + stenting	1 (1.5)
Intrasaccular flow disruptor + coiling	1 (1.5)
Intrasaccular flow disruptor + coiling + stenting	1 (1.5)

SAC: Stent-assisted coiling

Renal insufficiency

Preinterventional GFR values were available for all 58 patients. A total of 12 patients showed impaired renal function (20.7%). Renal impairment was present in two patients with RCMA (11.8%) and in 10 patients without RCMA (22.2%), with no significant difference between the two groups.

Antiplatelet therapy and anticoagulation

Related to 62 interventions, in 40 cases (64.5%) ASA + clopidogrel was given and in 15 cases (24.2%) ASA alone. ASA was given at a dosage of 100 mg/d after loading with 300 mg. Clopidogrel was given at a dosage of 75 mg/d after initial loading with 450 mg. In 5 sessions (8.1%) other combinations were used: 1 × ASA + tirofiban; 1 × ASA + clopidogrel + tirofiban; 1 × tirofiban; 2 × clopidogrel + phenprocoumon. Tirofiban dosage was adjusted for body weight and renal function. In 2 cases (3.2%), neither APT nor anticoagulation was used. Logistic regression analysis showed a significant association between APT and RCMA (P = 0.040) with

a higher risk for RCMA with the use of ASA alone, compared to dual APT (OR: 5.4).

A detailed summary of the results of the logistic regression analysis for the continuous and categorical variables is given in Tables 4 and 5.

Discussion

In this Study, we addressed the occurrence of DI after EVT of unruptured cerebral aneurysms. All cases of DI represented RCMA. This phenomenon occurred relatively frequently but did not represent hemorrhage or infarction in any case. Following EVT, some patients showed transient neurological deficits that were associated significantly with RCMA. Duration of EVT and use of APT were associated with the occurrence of RCMA.

In the context of endovascular procedures, postinterventional DI on CT scans appeared in the literature about 40 years ago, with early discussion of the problem of distinguishing SAH from contrast accumulation.[6] In 1998, Eckel *et al*. reported subarachnoid contrast accumulation after spinal catheter angiography and were able to distinguish it from hemorrhage by examination of the cerebrospinal fluid (CSF).[1] In 1999 Stone *et al*. described a case with extensive subarachnoid DI after coronary angiography that regressed by more than 50% in 24 h, which was considered as evidence for the presence of contrast accumulation and allowed differentiation from SAH.[2] The occurrence of transient neurological symptoms has also been described in association with various endovascular procedures involving iodine-containing contrast administration. Transient cortical blindness was reported after coronary angiography and spinal catheter angiography.[13,14] In connection with aneurysm treatment, a transient cortical DI after embolization of a ruptured aneurysm was first reported in 2004.[3] In the context of cerebral angiography, Li *et al*. found a correlation of high doses of CM and transient cortical blindness.[15] Vangosa *et al*. reported asymptomatic and reversible subarachnoid contrast enhancement after carotid stenting.[7] Many more data are available on contrast enhancement and contrast-induced encephalopathy after mechanical thrombectomy and after i.v. lysis therapy for acute ischemic stroke.[8,9,16] However, these patient collectives are only limitedly comparable to that of the present work, as contrast enhancement occurring in the context of stroke can be explained by ischemic breakdown of the BBB.

To our knowledge, the present study is the largest to date that focuses on the occurrence of DI on CT after EVT of unruptured cerebral aneurysms. If new intracranial DI are found after intervention, the distinction between RCMA and SAH is important as it affects the postprocedural management of patients, especially as it relates to dual APT after intracranial stent or flow diverter implantation. There are no uniform criteria that allow a definition of RCMA. Currently, four main characteristics can be found in the literature that can be used for differentiation. These include density measurement in Hounsfield units, density change over time and detection of blood in CSF obtained by lumbar puncture. In addition, dual energy CT provides the ability to differentiate between intracranial hemorrhage and contrast enhancement with high accuracy.[17] In our study, the diagnostic criterion for the presence of RCMA was the marked regression of intracranial DI on short-term control CT, similar to the works by Ozturk *et al*. and Brisman *et al*., who were the first to systematically study this phenomenon.[4,5] In the study by Ozturk *et al*. with a slightly smaller number ($n = 56$) of patients with unruptured aneurysms, and besides that of 37 patients with ruptured aneurysms, an association with the use of remodeling balloons, which were not used in our patient collective, was

Table 4: Associations of continuous variables with reversible contrast medium accumulation

Continuous variable	OR	95% CI	*P*
Age	0.968	0.914–1.026	0.277
Duration of intervention	1.016	1.001–1.031	0.038
Duration of anesthesia	1.009	0.998–1.020	0.110
Aneurysm diameter	1.198	0.995–1.444	0.057
Amount of given CM	1	1.000–1.000	0.154

CI: Confidence interval, CM: Contrast medium, OR: Odds ratio

Table 5: Associations of categorial variables with reversible contrast medium accumulation

Categorial variable	OR	95% CI	*P*
Aneurysm location			
ICA (including PCOM)-reference category			0.999
MCA	0.933	0.247–3.524	0.919
ACA	0.800	0.175–3.651	0.773
Vertebrobasilar	0.000	0.000	0.999
Devices			
Coiling – reference category			0.179
Coiling + stenting	1.136	0.187–6.889	0.889
IFD	5.833	0.900–37.818	0.064
FDS	3.333	0.319–34.830	0.315
Other combinations of devices	1.250	0.087–17.975	0.870
APT			
ASA + clopidogrel – reference category			0.040
ASA alone	5.388	1.466–19.801	0.011
Other combinations	1.886	0.302–11.772	0.497
Gender	0.589	0.144–2.417	0.463
Renal insufficiency	1.013	0.985–1.041	0.362

ACA: Anterior cerebral artery, APT: Antiplatelet therapy, ASA: Acetylsalicylic acid, CI: Confidence interval, FDS: Flow-diverter stent, ICA: Internal carotid artery, IFD: Intrasaccular flow disruptor, MCA: Middle cerebral artery, OR: Odds ratio, PCOM: Posterior communicating artery

discussed. Interestingly, Ozturk *et al*. refer to RCMA as a "clinically insignificant finding" because neurological deficits were not observed in their patient population. Brisman *et al*. studied a group of 30 patients, but only 14 with unruptured aneurysms, and found contrast enhancement in 23%, similar to our results, but without the appearance of neurological symptoms. We observed neurological symptoms in a good third of the patients with RCMA. These resolved completely in all cases.

To our knowledge, we also systematically investigated for the first time the occurrence of RCMA in connection with the use of newer devices such as FDSs and intrasaccular flow disruptors. Here, we could not find a significant difference in the occurrence of RCMA compared to the longer established methods of coiling or stent-assisted coiling. Regarding the association between iodine-containing CM and transient neurological symptoms, a potential neurotoxic effect of CM in the context of BBB breakdown has been repeatedly discussed, although the exact pathophysiological mechanisms have not yet been clarified.[1,10] Zevallos *et al*. observed CM-associated neurotoxicity in only 0.4% of 2510 "neuroendovascular procedures," although a comparison with our work seems limited because the exact type of procedures was reported by the authors only for the 0.4% of cases with neurotoxicity.[18] A recent study with 579 patients showed contrast-induced encephalopathy after embolization of unruptured cerebral aneurysms in 2.4%, but without addressing postinterventional CM accumulation on imaging, APT, or devices used for EVT.[19] It has been described that the structure of the BBB changes with age.[20] Moreover, there are studies that found age-and gender-dependent changes in the BBB, so one might also suspect a relationship between RCMA and age as well as gender.[21-23] However, in our work, no significant association was found for these two parameters with the occurrence of RCMA. As another potential factor influencing the occurrence of RCMA, we investigated the presence of renal insufficiency, because it could be assumed that impaired renal function with longer intravascular stay of the CM might increase the likelihood of passage through the BBB. Fittingly, subarachnoid contrast enhancement has been reported after coronary intervention in a patient with renal insufficiency, mimicking SAH.[11] However, we found no significant association between the presence of renal insufficiency and the occurrence of RCMA, while the number of patients with renal insufficiency in our study was small. In line with the work of Brisman *et al*.,[5] our results showed a significantly increased incidence of RCMA in patients with APT, both single and dual APT. Clopidogrel inhibits platelet function by irreversible binding to the P2Y12 receptor. Lou *et al*. demonstrated that clopidogrel also impairs juxtavascular microglial processes in resealing the injured BBB via P2Y12 receptor inhibition.[24] This could also play a role in the association of APT with the occurrence of RCMA that we observed, but in the end the exact underlying mechanisms still remain unclear. Earlier studies found a more frequent occurrence of RCMA at high doses of CM.[4,5,25] As the size of the aneurysms to be treated increases, a higher amount of CM may eventually be required for EVT, depending on the type of treatment, so one could speculate that there might also be a relationship between RCMA and aneurysm size. Consistent with this, Shinohara *et al*. found an association of postinterventional subarachnoid hyperattenuation with both aneurysm diameter and the given amount of CM.[26] In our results, there was only a statistical tendency but no significant association with aneurysm size. A correlation with the amount of CM was not shown in our work, but a more frequent occurrence of RCMA with increasing intervention duration. We found no significant association between aneurysm location and RCMA. Overall we observed nine patients with a vertebrobasilar aneurysm location of which no one had an RCMA. This resulted in an OR of 0 in the logistic regression analysis ($P = 0.999$). Nevertheless, we assume that the true OR is still small but > 0 resulting in a protective effect of this location.

In addition to the retrospective design of our study, a limitation to be mentioned is that although RCMA was found significantly more frequently in patients with APT, the number of patients without APT is relatively small. However, here our results are consistent with those of Brisman *et al*.[5] Even though we studied patients treated with newer devices in the context of RCMA for the first time, only a small case number is available here as well.

Conclusions

In conclusion, our results suggest that RCMA is common after treatment of unruptured aneurysms and significantly associated with neurological symptoms, which were invariably reversible in our cases. In addition to intervention duration, APT also appears to be associated with the occurrence of RCMA. Further research is needed to better understand the underlying processes at the level of the BBB.

Data availability statement

All data generated and/or analyzed during this study are included in this published article.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Eckel TS, Breiter SN, Monsein LH. Subarachnoid contrast enhancement after spinal angiography mimicking diffuse subarachnoid hemorrhage. AJR Am J Roentgenol 1998;170:503-5.
2. Stone JA, Sharp S, Castillo M. Subarachnoid contrast enhancement mimicking subarachnoid hemorrhage after coronary angiography. AJR Am J Roentgenol 1999;172:831-2.
3. Uchiyama Y, Abe T, Hirohata M, Tanaka N, Kojima K, Nishimura H, *et al.* Blood brain-barrier disruption of nonionic iodinated contrast medium following coil embolization of a ruptured intracerebral aneurysm. AJNR Am J Neuroradiol 2004;25:1783-6.
4. Ozturk A, Saatci I, Pamuk AG, Erdogan C, Akmangit I, Geyik S, *et al.* Focal increased cortical density in immediate postembolization CT scans of patients with intracranial aneurysms. AJNR Am J Neuroradiol 2006;27:1866-75.
5. Brisman JL, Jilani M, McKinney JS. Contrast enhancement hyperdensity after endovascular coiling of intracranial aneurysms. AJNR Am J Neuroradiol 2008;29:588-93.
6. Numaguchi Y, Fleming MS, Hasuo K, Puyau FA, Nice CM Jr. Blood-brain barrier disruption due to cerebral arteriography: CT findings. J Comput Assist Tomogr 1984;8:936-9.
7. Vangosa AB, Tortora D, Modestino F, Cotroneo AR, Caulo M. Reversible and asymptomatic gyral and subarachnoid contrast enhancement after carotid stenting. Neuroradiol J 2015;28:184-6.
8. Lummel N, Schulte-Altedorneburg G, Bernau C, Pfefferkorn T, Patzig M, Janssen H, *et al.* Hyperattenuated intracerebral lesions after mechanical recanalization in acute stroke. AJNR Am J Neuroradiol 2014;35:345-51.
9. Chu YT, Lee KP, Chen CH, Sung PS, Lin YH, Lee CW, *et al.* Contrast-induced encephalopathy after endovascular thrombectomy for acute ischemic stroke. Stroke 2020;51:3756-9.
10. Velden J, Milz P, Winkler F, Seelos K, Hamann GF. Nonionic contrast neurotoxicity after coronary angiography mimicking subarachnoid hemorrhage. Eur Neurol 2003;49:249-51.
11. Şimşek EÇ, Ertürk E, Uçar R, Yilmaz AO, Ekmekçi C, Mutlu İ, *et al.* Transient contrast neurotoxicity after percutaneous coronary intervention mimicking subarachnoid hemorrhage in a patient with chronic kidney disease. Clin Med Insights Case Rep 2019;12:1179547619867671.
12. Kidney Disease, Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2012 clinical practice guideline for the evaluation and management of chronic kidney disease. Kidney Int Suppl 2013;3:1-150.
13. Fischer-Williams M, Gottschalk PG, Browell JN. Transient cortical blindness. An unusual complication of coronary angiography. Neurology 1970;20:353-5.
14. Lo LW, Chan HF, Ma KF, Cheng LF, Chan TK. Transient cortical blindness following vertebral angiography: A case report. Neurointervention 2015;10:39-42.
15. Li M, Liang H, Liu C, Liu H, Zheng Y, Shi W, *et al.* Risk factors of transient cortical blindness after cerebral angiography: A multicenter study. Front Neurol 2019;10:1005.
16. Yoon W, Seo JJ, Kim JK, Cho KH, Park JG, Kang HK. Contrast enhancement and contrast extravasation on computed tomography after intra-arterial thrombolysis in patients with acute ischemic stroke. Stroke 2004;35:876-81.
17. Phan CM, Yoo AJ, Hirsch JA, Nogueira RG, Gupta R. Differentiation of hemorrhage from iodinated contrast in different intracranial compartments using dual-energy head CT. AJNR Am J Neuroradiol 2012;33:1088-94.
18. Zevallos CB, Dandapat S, Ansari S, Farooqui M, Quispe-Orozco D, Mendez-Ruiz A, *et al.* Clinical and imaging features of contrast-induced neurotoxicity after neurointerventional surgery. World Neurosurg 2020;142:e316-24.
19. Li M, Liu J, Chen F, Fan C, Yang X, Sun X. Contrast-induced encephalopathy following endovascular treatment for intracranial aneurysms-risk factors analysis and clinical strategy. Neuroradiology 2023;65:629-35.
20. Mooradian AD. Effect of aging on the blood-brain barrier. Neurobiol Aging 1988;9:31-9.
21. Parrado-Fernández C, Blennow K, Hansson M, Leoni V, Cedazo-Minguez A, Björkhem I. Evidence for sex difference in the CSF/plasma albumin ratio in ~ 20 000 patients and 335 healthy volunteers. J Cell Mol Med 2018;22:5151-4.
22. Castellazzi M, Morotti A, Tamborino C, Alessi F, Pilotto S, Baldi E, *et al.* Increased age and male sex are independently associated with higher frequency of blood-cerebrospinal fluid barrier dysfunction using the albumin quotient. Fluids Barriers CNS 2020;17:14.
23. Moon Y, Lim C, Kim Y, Moon WJ. Sex-related differences in regional blood-brain barrier integrity in non-demented elderly subjects. Int J Mol Sci 2021;22:2860.
24. Lou N, Takano T, Pei Y, Xavier AL, Goldman SA, Nedergaard M. Purinergic receptor P2RY12-dependent microglial closure of the injured blood-brain barrier. Proc Natl Acad Sci U S A 2016;113:1074-9.
25. Baik SK, Kim YS, Lee HJ, Park J, Kim GC. Immediate CT findings following embolization of cerebral aneurysms: Suggestion of blood-brain barrier or vascular permeability change. Neuroradiology 2008;50:259-66.
26. Shinohara Y, Sakamoto M, Takeuchi H, Uno T, Watanabe T, Kaminou T, *et al.* Subarachnoid hyperattenuation on flat panel detector-based conebeam CT immediately after uneventful coil embolization of unruptured intracranial aneurysms. AJNR Am J Neuroradiol 2013;34:577-82.