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Considerations about Occlusion of the Intracranial Distal Internal Carotid Artery

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Abstract Occlusion of the intracranial distal internal carotid artery (ICA) is one of the most critical conditions among the cases of acute stroke in the anterior circulation.

The introduction of selective endovascular treatment first using thrombolytic agents replaced later by the mechanical thrombectomy using various devices has improved the prognosis in a certain number of these patients. Among the factors influencing the prognosis of these patients, one is the collateral circulation which in these cases is mainly characterized by leptomeningeal anastomoses. The collateral can, however, be impaired, by distal embolization and by anomalies of the Circle of Willis: the aim of this study is to describe these aspects.

Keywords Distal ICA occlusion · T occlusion of ICA · Collateral circulation

Introduction

Occlusion of the intracranial distal internal carotid artery (ICA) is one of the most critical conditions among the cases of acute stroke in the anterior circulation, commonly characterized by a poor outcome. Some improvements of the prognosis of these patients have been achieved through the endovascular therapy, using first thrombolytic agents [1–5].

The progressively replacement of the thrombolytic agents with the mechanical thrombectomy, using various devices has led to some further improvements [6–17]. It has become progressively clear that the initial clinical condition and the short interval between stroke and reopening of the occluded ICA, with complete or partial revascularization of its distal branches are among the most important factors influencing the final clinical result [12, 16, 17]. Another important factor which has already been described is the presence of the collateral circulation [7, 18, 19]. The presence and extent of it, however, can be influenced by some factors which will be the subject of this study.

Materials and Methods

We reexamined eight patients, three male and five females, with distal ICA occlusion, admitted in our hospital in the past 12 months. The median age of the patients was 68. In all the cases, the interval between stroke and examination was 4–6 h. The National Institutes of Health Stroke Scale NIHSS range was 6–19. Computed tomography (CT) was performed in every case, the angio-CT in five. A complete angiographic study, including both carotid and one vertebral angiogram, was performed in six cases. In two other, only the occluded ICA was examined and immediately treated after the demonstration of the occlusion. Endovascular treatment was performed using a Solitaire stent in five associated with Penumbra clot retrieval in two, in one patient only conservative therapy was carried out. Every patient was examined with CT 24 h after the treatment.

Results

The patient number 1 on the table was an 80-year-old man presented with aphasia, right hemiplegia, and agitation, about 5 h after stroke. No certain ischemia was visible on CT, angio-CT was not performed, angiography showed occlusion of the distal ICA with extension to M1 and A1 (Typical complete “T” occlusion). The right Posterior Cerebral

Artery (PCA) arises from the basilar artery and that of the left was not recognizable, arising probably from the occluded left ICA. There was a poor collateral circulation.

It was possible to reopen partially the left distal ICA and PCA, but not the Anterior Cerebral Artery (ACA) and Middle Cerebral Artery (MCA). The patient developed a large left hemispheric infarct and died 72 h later.

The patient 2 was a 62-years-old man, presenting at admission about 6 h after the stroke with mild clinical symptoms, characterized by a stable light motor deficit. There was no aphasia. No certain infarct was recognized on CT.

On the angiogram, occlusion of the left ICA and M1 was demonstrated. A contralateral ICA and vertebral angiograms showed a rich collateral circulation, through leptomeningeal anastomoses from the left ACA and PCA toward the MCA. The left ACA was injected through the AComA. The PCA arised from the basilar artery. Considering the good clinical conditions and the rich leptomeningeal anastomoses, conservative medical therapy was chosen. The patient recovered well with only a mild motor deficit.

In two other patients (cases 3 and 4 on the table) were two women aged 58 and 72 respectively, presenting both with right hemiplegia and aphasia. The patient 5 was 68-yearsold woman and the 6 was a man aged of 65 both presenting with left hemiplegia and agitation. All four patients were examined and treated between the 4th and 5th hour after stroke. In all these four patients CT was normal, angio-CT was similar in all cases, apart the occlusion of distal ICA on the left in two cases and on the right in the other two. The occlusion extended to M1 segment. The contralateral carotid angiogram showed the filling of the A1 of the occluded ICA.

There was a partial retrograde leptomeningeal injection of the occluded MCA. In all cases, both PCAs arise from the basilar artery. A rapid recanalization was achieved. CT per- no hemorformed 24 h later showed ischemic foci in the basal ganglia (Figs. 1 and 2). All the four patients recovered well.

The patient 7 was a 70-year-old woman presenting with right hemiplegia and aphasia 5 h after stroke. CT was normal. Angio-CT was not performed; the angiographic study showed the distal left ICA occlusion with involvement of M1. There was a poor collateral circulation toward the MCA from the ACA. The endovascular treatment allowed reopening of the distal ICA and of the fetal PCA and AchA and of the proximal M1. CT performed 24 h later showed infarct at the level of the basal ganglia (Fig. 3). The aphasia resolved rapidly and there was a progressively improvement of the motor deficit in the following weeks.

The patient 8 was a 70-year-old cardiopathic woman presenting with left hemiplegia and agitation 5 h after stroke. CT was normal. Angio-CT showed occlusion of the right distal ICA with involvement of M1. There was a filling of the A1 segment and a poor collateral circulation towards the MCA. The right PCA was not recognizable, probably involved in the occlusion (Fig. 4). Angiography showed the ICA occlusion. A rapid recanalization was achieved. On the lateral angiogram, however, the distal branches of the MCA where sparely injected, probably occluded by emboli. A final angiographic study of the contralateral ICA and of the vertebral artery showed the injection of both ACAs and of the left fetal PCA, also injected through a small P1. There was no injection of the right PCA. CT 24 h later showed a large infarct involving MCA and PCA vascular territories (Fig. 4). The patient survived with a severe hemiplegia and hemianopia. Summarizing, we can observe that in five cases the occlusion involved the ICA and M1. There was a good leptomeningeal collateral circulation arising from ACA and PCA towards MCA. Among these five, in four patients (case 3–6) the endovascular treatment allowed a complete recanalization with a final good clinical result. Also in the case number 2 the clinical result was good, despite that no vascular treatment was performed due to the mild clinical symptoms and the presence of a rich collateral circulation.

In the patients 7 and 8 there was occlusion of ICA and M1. A collateral circulation was present between ACA and MCA. The PCA was not involved in the collateral circulation, since it arised from the occluded ICA. In one patient (case 7) it was possible to reopen the distal ICA as well as the PCA, and partially the proximal M1. The distal M1 remained occluded. There was relatively good leptomeningeal collateral circulation probably also through the reopened PCA, which helped to obtain a good clinical result. In the patient 8 there was a rapid recanalization of M1. However, the distal MCA branches remain occluded as well as the PCA, arising from the occluded ICA. Large hemispheric infarct developed and the patient remained severe disabled.

In the patient 1, there was a complete T occlusion (ICA, A1, and M1) with involvement also of the PCA. Only partial recanalization was possible, involving partially the ACA and PCA. A large infarct developed, and the patient died 72 h later. With exception of a small hemorrhagic component in the ischemic area of the basal ganglia in one patient, no hemor rhagic complications occurred. All dates of the patients are represented in the Table 1.

Discussion

Differently from intracranial ICA occlusion proximal to the origin of the ophthalmic artery, in which there is a collateral circulation through anastomoses between the external carotid artery and the ophthalmic artery, and through the Circle of Willis, provided that this latter does not present unfavorable variations, in the cases of intracranial occlusion distal to the origin of the ophthalmic artery, there is involvement of the supraclinoid segments (communicating—choroidal) and also of the first segment of MCA (M1) and ACA (A1) in various combinations. The most frequent pattern that we observed as also reported in other larger studies [5, 20] is characterized by involvement of ICA and M1, which can be called partial “T” occlusion. It is possible and thinkable that also part of the proximal A1 is involved, but this cannot be identified with certainty. The patency of the distal part can be demonstrated on angio-CT, and cerebral angiography. More rarely, the occlusion involves ICA and both M1 and A1 segments forming the so-called typical “T” occlusion. In all these cases, the only possible collateral circulation are leptomeningeal anastomoses

between distal branches of the cerebral arteries. It can occur that this is already very efficient shortly after the occlusion, explaining in some patients the presence of mild neurological symptoms and a good clinical outcome [7, 20]. In the majority of the other cases, however, the collateral circulation is poor, and this is associated with a bad clinical outcome. In a certain number of these patients, the endovascular treatment can improve their prognosis, leading to a complete or to a partial revascularization favoring, in this latter cases, the collateral circulation. There are, however, a few basically unfavorable conditions which can impair the final results.

The one is the distal embolization, leading to occlusion of the vessels involved in the leptomeningeal collateral circulation. This occurred in one of our cases. Distal embolization can be linked with the endovascular treatment. This complication is a well-known adverse event. In a recent large review about the endovascular treatment performed in the patient with stroke in the anterior circulation, it is reported to occur in 5 % of the cases. Distal embolization can also be due to emboli arising immediately or progressively from the thrombus occluding the ICA. In this latter condition, the impairment of the leptomeningeal collateral circulation, leading to a suffering of the involved brain parenchyma, could explain the negative evolution with infarct without or with an hemorrhagic component, occurring in some patient in spite of the technically obtained complete revascularization [12, 19, 21].

The second unfavorable condition are anatomical variations of the Circle of Willis [22]. Among these, the most frequent are that characterized by the origin of both the ACA from the occluded ICA, commonly associated with an hypoplastic or aplastic contralateral A1, and that in which the PCA (fetal type) arises from the occluded ICA, being directly involved in the occlusion. This latter situation occurred in three of our cases, influencing the clinical evolution, which was very bad in two.

Finally, we should realize that even in the cases of the complete recanalization, ischemia with various extents at the level of the basal ganglia is almost always present [22].

This is due to the involvement of the perforating branches supplying these structures. These arteries are end-arteries, and so their occlusion, even for a short time, leads to ischemic lesion in their vascular territories.

Conclusions

The distal ICA occlusion, in spite of the improvement of the endovascular therapy remains a very serious condition. Certainly, the initial clinical condition and the short interval between the onset of stroke and the achieved revascularization are important aspects, influencing the possibility of the positive result. Also important is the presence of an efficient leptomeningeal collateral circulation.

In spite of the fact that the study presents some limitations, due to the relatively small number of patients, some interesting aspects can be recognized. In the two types of distal ICA occlusion (complete and partial “T”) the leptomeningeal collateral circulation can be the same. It is its impairment due to distal embolization and/or anomalies of the Circle of Willis, which can influence negatively the final result.

The complete examination of the cerebral vessels which can be today obtained rapidly with angio-CT followed by a complete angiographic study, including both carotid and at least one vertebral angiograms is essential in the investigations of these patients preceding the treatment.

Table 1 Table indicating the clinical aspects, the type of the occlusion and final results

Patient	Age/Sex	NiH	Interval to treatment (hours)	Technique	Revascularization	Distal embolization	Willis anomalies	Results
1	80 M	19	5	Solitaire/Penumbra	Partial	–	Yes	Death
2	62 M	6	6	Conservative	–	No	No	ID
3	58 F	12	4	Solitaire	Complete	No	No	CR
4	72 F	12	5	Solitaire	Complete	No	No	ID
5	68 F	15	5	Solitaire	Complete	No	No	CR
6	65 M	15	4	Solitaire	Complete	No	No	ID
7	70 F	15	5	Solitaire/Penumbra	Partial	No	Yes	ID
8	70 F	16	4	Solitaire	Partial	Yes	Yes	SV

CR complete recovery, ID independent with minimal deficit, SV severe disabled

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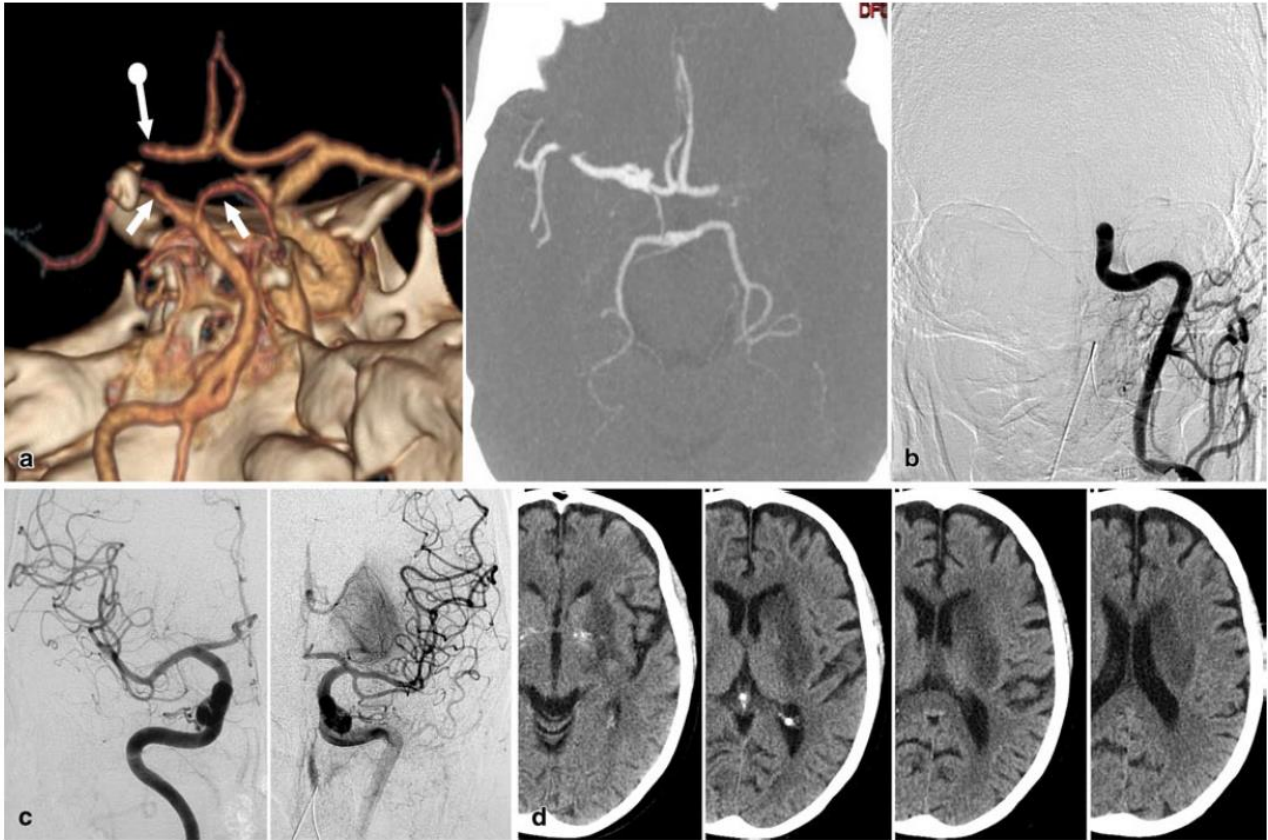
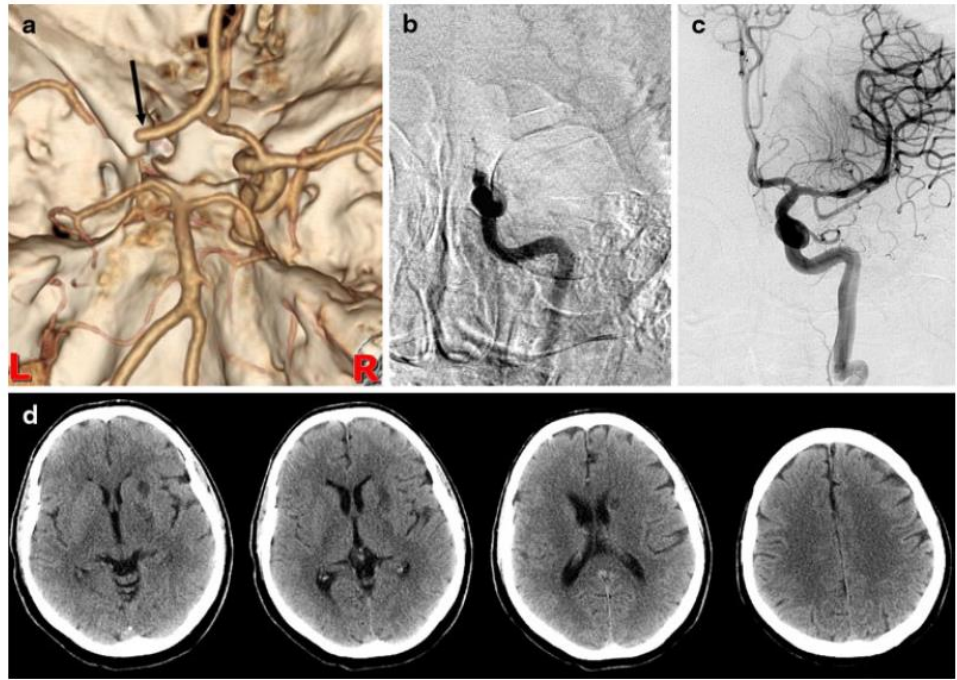


Fig. 1 (Patient 5) **a** Angio-computed tomography (CT) represented by volume rendering (*left*) and Multi Planar Reconstruction technique (*right*). Occlusion of the left distal internal carotid artery (ICA). The efficient AcomA allows the filling of the left A1 (*arrow with dot*). Both PCAs (*arrows*) arise from the basilar artery. **b** Left carotid angiogram

showing the occlusion of the distal ICA. **c** Posttreatment angiograms. Revascularization of the left ICA. Note the blush in the vascular territory of the perforators with early filling of the draining veins. Right carotid angiogram. **d** Posttreatment CT. Ischemia involving pallidum, putamen, and the superior part of the head of the nucleus caudatus

Fig. 2 (Patient 3) **a** Volume rendering technique angio-computed tomography (CT). Occlusion of the left distal internal carotid artery (ICA). Through the AcomA filling of the well-developed left ACA and the left A1 (*arrow*). Both PCAs arise from the basilar artery. **b** Left carotid angiogram showing the occlusion of the distal ICA. **c** Posttreatment angiograms. Complete revascularization. Blush in the vascular territory of the perforators. **d** Posttreatment CT. Small Ischemia at the level of the basal ganglia



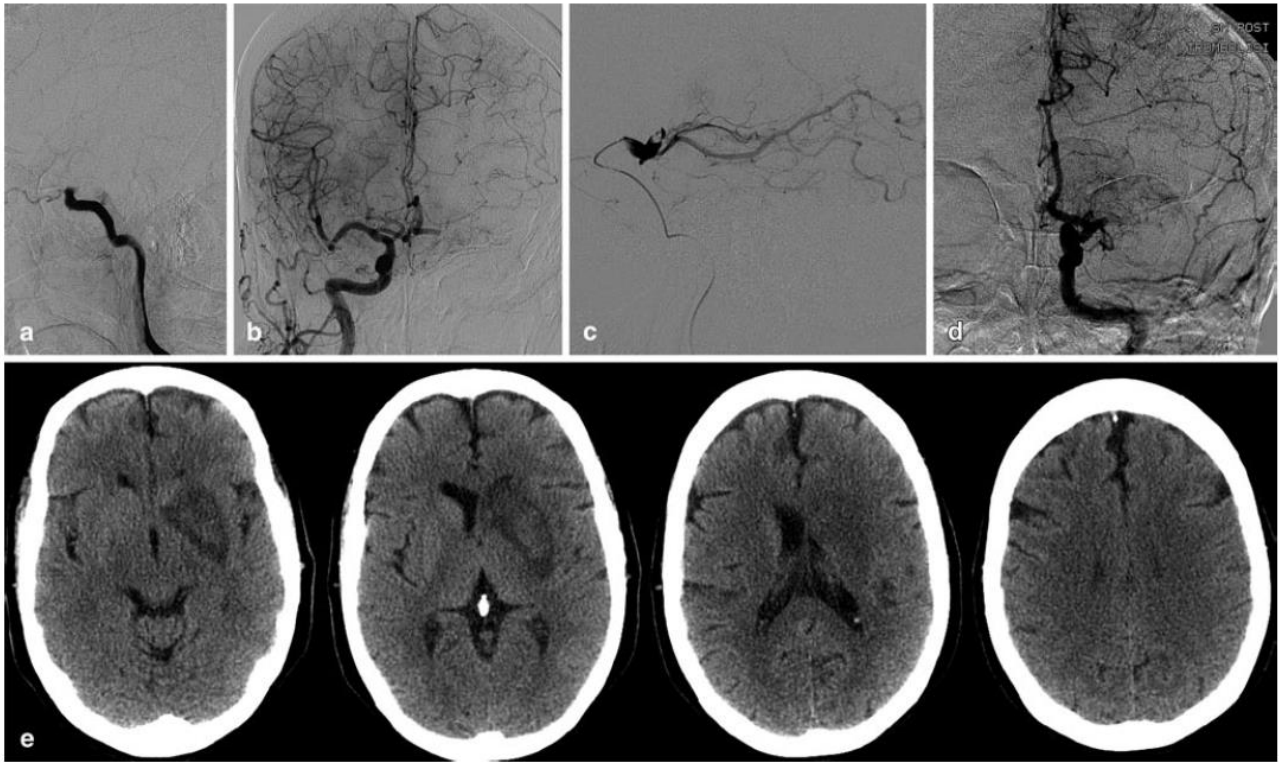


Fig. 3 (Patient 7) **a** Left carotid angiogram showing occlusion of the distal internal carotid artery (ICA). **b** Right carotid angiogram showing a good collateral circulation towards the MCA. **c** Endovascular treatment with revascularization of the distal ICA as well as the AchA

and PCA. This latter arising from the occluded ICA. **d** Posttreatment angiogram showing the persistence of the occlusion of the distal M1. **e** Posttreatment CT showing ischemia in the basal ganglia sparing the inferior head of the nucleus caudatus

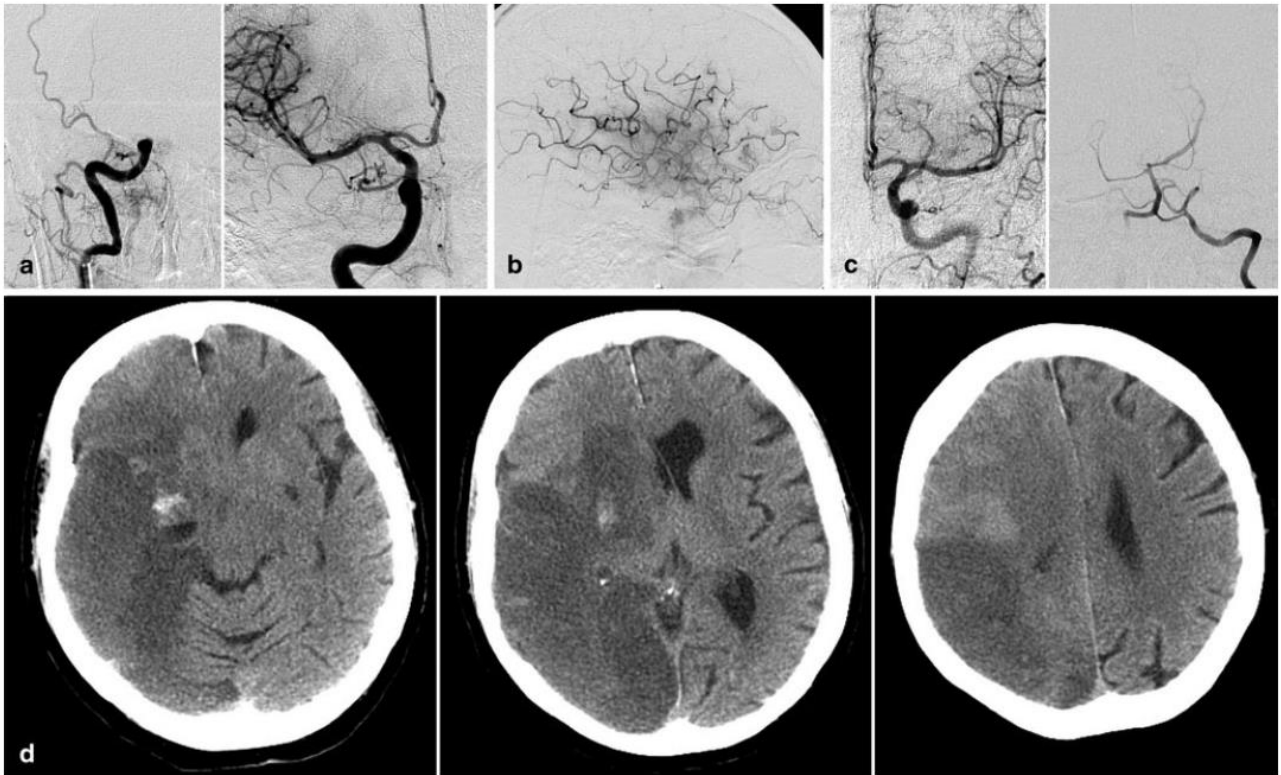


Fig. 4 (Patient 8) **a** On the left, right carotid angiogram showing occlusion of the distal internal carotid artery (ICA). On the right, same angiogram posttreatment showing the revascularization. **b** On the post-treatment lateral carotid angiogram, there are however occlusion of several distal branches of the MCA. **c** Posttreatment angiographic con-

trol of the left carotid and vertebral arteries. The left PCA arise from the ICA. Minimal filling also through a small P1. There is no filling of the right PCA. **d** Posttreatment computed tomography showing a large ischemia involving the vascular territories of MCA and PCA. Minimal hemorrhagic component in the basal ganglia