Clinical study

Arterial inflow and venous outflow in idiopathic intracranial hypertension associated with venous outflow stenoses

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Summary

A reduction in the proportion of the arterial inflow drained by the sagittal sinus in idiopathic intracranial hypertension (IIH) patients without stenoses has been noted and this has suggested elevated collateral flow. This study defines the interaction between arterial inflow and venous outflow in patients with outflow stenoses and IIH. Forty patients with clinical IIH underwent standard MRI, MR venography and MR flow quantification studies of the cerebral arteries and veins. There were 21/40 patients with

venous stenoses. The arterial inflow was 21% higher than normal ($p = 0.01$); however, the superior sagittal sinus outflow was normal, giving a reduced percentage of venous outflow compared to inflow. Seven patients were followed up after remission of their symptoms and the arterial inflows and percentage outflow returned to normal. There is a spectrum of findings in IIH; patients with stenoses have lower inflows than those with patent sinuses but still have evidence of collateral flow.

Key words Idiopathic intracranial hypertension; cerebral blood flow; MRV; stenosis

1. Introduction

The clinical syndrome of idiopathic intracranial hypertension (IIH), also known as benign intracranial hypertension or pseudotumor cerebri, occurs in patients who present with high-pressure type headaches and/or papilledema and visual obscuration.[1] At lumbar puncture, cerebrospinal fluid (CSF) pressures are elevated but by definition the composition of the CSF is found to be normal.[1] Routine CT scan and MRI show no brain parenchymal abnormality.[2] Kalahalios et al. have hypothesized that an elevated venous sinus pressure is the underlying cause of all cases of IIH[3] and King et al. have confirmed this by directly measuring sinus pressures in a cohort of IIH patients.[4] An elevation in venous sinus pressure reverses the pressure gradient between the subarachnoid space and the venous sinuses through the arachnoid granulations, thus elevating CSF pressure.[5] In many patients the elevated venous sinus pressure is due to venous sinus thrombosis or stenosis; however, in a proportion of patients these stenoses are reversible[4] and in some patients the sinuses are patent with no evidence of outflow obstruction.[6] In a previous report an association was noted between patients with patent or mildly stenosed sinuses

and cerebral inflow hyperemia but with normal outflow volumes giving a reduced percentage of venous return compared to the inflow.[7] This suggests elevated collateral flow on the venous side of the vascular tree. The interaction between blood flow and patients with higher grade stenoses has not been described. With this in mind, 21 patients with intracranial hypertension and venous outflow stenoses are described; the clinical presentation, MR venogram (MRV) and cerebral blood flow findings are reviewed. The purpose of this study is to define the interaction between arterial inflow and the varying degrees of venous outflow obstruction found in these patients.

2. Methods

2.1 Subjects

Forty patients who presented with a suspected clinical diagnosis of idiopathic intracranial hypertension and confirmed elevated intracranial pressure were prospectively entered into the study. The patients were referred for MRI by neurologists in a tertiary referral hospital setting between June 1999 and February 2006. The patients presented with typical clinical features of headache ± papilloedema ±visual obscuration. Raised CSF pressure (above 25 cm of water) and no abnormalities of CSF composition were confirmed in all patients. This group was then subdivided on the basis of the MRV findings into three groups of patients, that is, those with thromboses, those with patent sinuses and those with stenoses. The patients with thrombosed and patent sinuses have been previously reported.[7] The third group of 21 patients consisted of 20 females and one male of mean age 29±14 (SD) years. These patients had evidence of stenoses in the venous outflow on MRV. Seven of the patients with evidence of stenoses on MRV (all female, mean age 24±14 years) were re-imaged following remission of their symptoms

and these patients were analyzed separately. The control group was selected from patients referred to the MR unit for diagnostic studies for conditions not related to raised intracranial pressure, chronic headaches or ischemia and consisted of 12 females and two males, mean age 31±13 (SD) years. Informed consent was obtained from all patients and the hospital ethics committee reviewed the study protocol.

3. MRI and analysis

All patients were imaged on a 1.5 T superconducting magnet. Standard T_1 sagittal and T_2 axial images were acquired to rule out an intracranial mass lesion. A time-of-flight 3D MRV study was performed. Following MRV three groups of patients could be defined, that is, those with normal sinus morphology (see Fig. 1), those with an intrinsic filling defect (thrombosis, see Fig. 2) and those with a narrowed, stenosed dominant sinus (Fig. 3) or with areas of flow void in the dominant transverse sinus (Fig. 4). Filling defects were differentiated from flow voids by positive identification of clot on T_1 or T_2 imaging or by reconstruction of a post-contrast 3D T_1 study along the course of the sinus outflow to show either a clot or tapering stenosis (Fig. 5). The patent and thrombosis patients have been previously reported.[7] A retrospectively cardiac-gated phase contrast flow quantification sequence was used with a time-to-repeat of 29 ms, echo time 7 ms, flip angle 30°, slice thickness of 6 mm, matrix 192 × 512, field-of-view 200 and a single nex. The velocity encoding (venc) values of 40 cm/sec and 75 cm/sec were used. The lower venc value was selected to maximize the measurement of the venous structures with the higher one used to maximize the arterial measurements. The plane of section was selected to intersect the superior sagittal sinus (SSS) approximately 2 cm above the confluence of sinuses and to continue through the straight sinus and pass through the

basilar artery and the cavernous portion of the internal carotid arteries as per the literature.[6] Regions of interest were placed around the SSS and the carotid arteries and basilar artery in each patient. Care was taken to exclude aliasing by retrospectively manipulating the base lines of each resultant graph, giving an effective venous upper flow limit of 80 cm/sec and arterial flow limit of 150 cm/sec.

The addition of the flow in both the carotid and the basilar arteries gave the total arterial inflow for each patient. The SSS outflow was calculated from the respective region of interest. Mean and standard deviations were obtained for each group of patients. Differences between the groups were tested using a nonpaired *t*-test.

4. Results

The blood flow data for the stenosis patients are summarized in Table 1.

In 21/40 or 53% of the patients the MRVs showed narrow outflow sinuses or flow gaps and review of the T_1 pre- and/or post-contrast studies showed no filling defects. The stenosed regions were in both dominant and non-dominant transverse sinuses in 19 patients and in the dominant transverse sinus alone in two. A visual rating of the degree of stenosis in the dominant transverse sinus showed five patients with stenoses between 40–70%, six with stenoses between 70–80% and 10 with flow gaps secondary to spin dephasing suggesting a > 80% stenosis. Fifteen patients presented with headache, 16 presented with papilledema and six with visual obscuration with some overlap between patients. The mean weight was 90 ± 25 kg and the mean CSF pressure 31 ± 4 cm H_20 with no biochemical or cytological abnormality on CSF examination.

The mean arterial inflow in the stenosis patients was 1070 mL/min, the 95% confidence limits being 1172 to 968 mL/min verses 880 mL/min in the controls, the 95% confidence limits being from 960 to 800 mL/min, i.e. 21% above normal ($p = 0.01$). The

sagittal sinus outflow was within the normal range. The elevated inflow but normal outflow volume meant that the mean outflow as a percentage of the total inflow was reduced to 35% with 95% confidence limits of 37% to 33% verses the control value of 48% with 95% confidence limits of 51% to 45% ($p < 0.0001$). At follow-up, seven patients showed a reduction in arterial inflow back toward normal but no change in outflow volumes, indicating a reduction in collateral flow.

5. Discussion

Traditionally, patients who present with the clinical syndrome of IIH are split into two groups, that is, those with a major venous thrombosis (secondary intracranial hypertension) and those with seemingly "normal" venous outflow or IIH. Most of these latter patients have been shown to have significantly elevated pressure gradients in their transverse sinuses at catheter manometry.[4] A closer review of the MRV studies in IIH patients has shown many to have flow gaps and narrow segments in the dominant outflow transverse sinuses,[8] with these abnormalities corresponding to the site of the pressure gradients. These dominant sinus flow gaps are not seen in normal patients.[8] Despite the findings of stenoses and thromboses in many of these patients, there still remains a group with apparently normal venous outflow morphology.[7,8]

A previous study showed the outflow volumes in the sagittal sinuses of patients with thrombosed sinuses to be less than the controls with the percentage of the total inflow drained by the sagittal sinuses of these patients being 27±10% as opposed to 48±6% in the control group ($p < 0.0001$). This indicated evidence of collateral flow bypassing the obstruction.[7] Patients without evidence of an outflow obstruction have a significantly elevated arterial inflow approximating 50–60% above normal,[6,7] but a

normal outflow volume similarly giving a reduced percentage of the outflow compared to inflow and indicating collateral flow.[7] The aim of this paper is to use the same methodology to define the interaction between inflow and outflow in IIH patients with evidence of outflow stenoses.

5.1 Blood flow changes in patients with stenoses

The majority of patients studied with clinical IIH have stenoses evident on MRV in the dominant transverse sinus region with three-quarters of these being greater than 70% by morphology. Similar to the patients with patent sinuses, the stenosis patients had an elevation in cerebral inflow.[6,7] However, this elevation was not as large as that noted in the patients with patent sinuses. The elevated inflow but normal outflow gave a reduced outflow as a percentage of the inflow. As discussed above, this is evidence of collateral flow. Figure 6 graphically shows these flow changes. The patient in Figure 6a presented with headache and papilledema and had an elevated pressure at lumbar puncture. One month later, following medical treatment only, she represented while in remission of her symptoms and signs (Fig. 6b). Finally the patient represented 5 months later, again symptomatic (Fig. 6c). While symptomatic, there was evidence of a venous outflow stenosis (large arrow) and also increased collateral flow leaving the intracranial cavity via the scalp, face and skull base (smaller arrows); while asymptomatic both the stenosis and collateral flow resolved. The sagittal sinus normally drains close to 50% of the inflow. Irrespective of the cause, an elevated venous sinus pressure opens collateral channels and thus the percentage of flow represented by the sagittal sinus drops in all forms of IIH.

5.2 Venous stenoses: fixed or dynamic?

Seven patients were followed up while in remission of their symptoms to see whether changes in the stenoses or the elevated arterial inflow would correlate with clinical improvement. A spontaneous improvement in the stenosis was seen in three of the seven follow-up patients (Fig 6. is an example). A reversal or reduction in a stenosis while in remission or following treatment has previously been described in the IIH literature.[9,10] In the remaining four patients who went into remission there was no apparent change in vein diameter (Fig. 7). Non-reversibility despite treatment or remission also has been described in the literature.[11] These latter four cases could represent fixed stenoses. This finding is illustrated by Figure 7b where CSF removal reduced collateral flow but did not change the appearance of the outflow stenosis. In all seven patients who improved clinically there was a mean reduction in inflow volume back to normal and an increase in outflow drainage percentage from a mean value of 32% back to normal at 48% ($p = 0.008$). This finding suggests that both the stenoses (whether dynamic or fixed) and also the elevation in inflow volumes are involved in the pathophysiology of this group. It is not difficult to understand how an elevation in inflow volume could stress the efficiency of the outflow drainage in a patient with a fixed outflow stenosis and make these patients more likely to be symptomatic, but what of the dynamic stenoses?

5.3 Sinus wall compliance, dynamic resistors and the feedback loop

The findings of King et al. suggest that many stenoses such as those noted in these 21 patients may be non-fixed or dynamic stenoses.[4] They found a significant increase in sinus pressure in IIH patients and an abrupt reduction in venous pressure once the subarachnoid pressure was returned to normal by removing CSF. This suggested to the authors that the increased sinus wall compliance and reversible stenoses in the eight

patients studied were not the cause of the underlying condition, but were secondary to it.[4] However, the findings of this study suggest that reversible stenoses are associated with increased collateral flow and therefore are haemodynamically significant and may be important in causation.

The main abnormality in IIH is an increase in intracranial pressure (ICP). Intracranial pressure is dependant on a balance between CSF production and reabsorption. Davson et al. modeled the relationship between ICP and the formation and reabsorption of CSF showing that

$$ICP = R_{out} \times FR_{CSF} + P_{SS}$$

where R_{out} is the resistance of CSF outflow, FR_{CSF} is the formation rate of CSF and P_{SS} is the sagittal sinus pressure.[12] This equation holds in conditions of steady state where the formation rate is equal to the reabsorption rate but not, for example, during the pressure rise before a plateau is reached.[12] Thus at steady state, the equation suggests that the elevation in CSF pressure noted in IIH must come about either because of an elevation in the CSF production rate and/or an elevation in the resistance to CSF reabsorption and/or an elevation in sagittal sinus pressure. Janny et al. simultaneously measured the CSF and superior sagittal sinus pressure in 16 patients with "benign intracranial hypertension". Excluding the four patients they studied with recent head injury and the one patient with symptoms secondary to a drug reaction (these clearly did not have IIH) they found a mean CSF-SSS gradient of 0.5 mmHg.[13] In the 21 IIH patients studied by King et al. a mean CSF pressure of 27 mmHg and sagittal sinus pressure of 22 mmHg, gave a CSF-SSS gradient of 5 mmHg, which is in the normal range (2–6 mmHg).[4,14] Owler et al. similarly found a normal CSF-SSS gradient in the patients they studied using a similar technique.[15] Rearranging Davson's equation we find that the CSF-SSS pressure gradient

is equal to the product of the CSF production rate and the resistance to flow across the arachnoid granulations, that is, $ICP–P_{SS} = R_{out} \times FR_{CSF}$. Two groups have directly measured FR_{CSF} (as opposed to indirectly calculating it) in IIH; Gideon et al. used phase contrast MR measurement of aqueduct flow[16] and Malm et al. used a constant flow technique,[17] with both showing the CSF formation rate to be normal in this condition. If direct measurement by three groups has shown the gradient to be normal and direct measurement by two groups has shown the formation rate to be normal, then the R_{out} must also be normal during the steady state pressure elevations found in IIH. This finding is in conflict with the literature, which suggests that there is an elevated R_{out} in IIH.[17–19] This discrepancy can be explained if the assumptions inherent in calculating the R_{out} using CSF infusion tests are understood. R_{out} is calculated to be the slope of the graph obtained when mock CSF is infused into the spinal canal at varying rates or pressures and the resultant CSF pressure is plotted against the infusion rate. Ekstedt states that the slope of this regression line is the R_{out} but also notes that the slope could theoretically be explained by an elevation in the sagittal sinus pressure occurring during the course of the test.[20] The requirement that the sagittal sinus pressure not change during the course of the test is routinely ignored, the conclusion being: "whether or not the assumption of a rectilinear extrapolation is true can only be proved when the sagittal sinus pressure is measured directly during a CSF hydrodynamic experiment".[20] As King et al. have shown, the sagittal sinus pressure does change when CSF pressures are changed in IIH and it is this fact that invalidates infusion testing in this condition. Direct measurements indicate that the sagittal sinus pressure elevation is the sole variable bringing about the elevation in CSF pressure in IIH despite itself being secondary to the CSF pressure elevation. This

apparent contradiction can be explained if one accepts that a positive feedback loop exists in the venous outflow pathways of patients with IIH and dynamic stenoses.

In patients where there is increased CSF pressure and increased venous wall compliance, there is the risk of an unstable feedback loop developing. The initiation of this unstable feedback loop is due to the development of dynamic resistance.[7] Dynamic resistance literally means that the resistance to flow along the vein changes depending on the circumstances the vein finds itself in, that is, the pressure gradient across its wall. The resistance is elevated in a linear fashion as the pressure gradient across the vein wall increases. It is the hallmark of dynamic resistors that the flow through the tube remains constant no matter what the pressure gradient from inflow to outflow.[21] A well-known example of a dynamic resistor is that of the bronchial tree where there is an upper limit to airflow rate no matter how hard a patient tries to exhale. The increasing intrathoracic pressure above a certain point compresses the bronchioles and increases the resistance linearly with effort thus setting a fixed expiratory flow.[21] The fact that the sagittal sinus outflow volumes in IIH patients have been shown to be normal (i.e. a fixed rate) by both this study and others[6,7,16,22] despite the range of stenosis severities (from fully patent to almost occluded) indicates that the stenoses in these patients are behaving like dynamic resistors. Figure 8 shows the pressures actually found in the 21 IIH patients studied by King et al.[4] Most normal patients have incompliant sinuses[23] but in IIH patients they are compliant which allows the CSF pressure to compress them, this has the effect of increasing the venous pressure and reducing the CSF-to-sinus pressure gradient. Thus, a dynamic resistor develops in the transverse sinuses of patients with compliant sinuses. This means that the CSF flow through the granulations can no longer act as a pressure relief valve but now acts as a positive feedback mechanism.[7] It can be seen that the

pressure gradient across the transverse sinus wall (CSF-to-venous pressure gradient) in King et al.'s patients is at the upper limit of the normal range for the opening pressure of the granulations (normally 2–6 mm Hg).[14] Thus, whatever the elevated CSF pressure, if the venous pressure is set to 6 mmHg less than the CSF pressure by the dynamic resistor then there is no drive for CSF flow through the granulations. Note, the very same CSF-to-sinus pressure gradient that is reduced by the dynamic resistor is the same one needed to drive CSF resorption. Removing the pressure relief valve in this way does not necessarily lead to an increased CSF pressure *per se* but any increase in CSF pressure that occurs, from whatever cause, would be maintained at a new higher set-point because the relief valve is inoperative. Patients with totally fixed stenoses in the venous outflow, such as occur secondary to thrombosis, have elevated venous and CSF pressures in the absence of a raised cerebral blood flow[6,7] but a feedback loop such as that described above requires an initiator. What initiates and maintains the pressure rise? It is well known that an increase in cerebral blood inflow can increase CSF pressure.[24] In patients with an underlying transverse sinus stenosis it appears from the data that the increase in CSF pressure is being brought about by a modest increase in cerebral blood inflow and remission by a reduction in this inflow. It is the inability of the venous outflow (exacerbated by the stenoses) to cope with this modest extra inflow, which promotes the venous pressure and therefore the CSF pressure.[7]

6. Conclusion

There appears to be a spectrum of outflow obstruction and inflow hyperemia in patients with IIH. Both of these variables may interact in any one patient to produce elevated collateral flow with normal sagittal sinus flow. In patients presenting with IIH, three separate subgroups can be defined: (i) those with filling defects and a venous

thrombotic intracranial hypertension; (ii) those with normal sinuses and a hyperemic intracranial hypertension; and (iii) those with sinus narrowing and a venous stenotic intracranial hypertension. In the stenotic form two subgroups were discernable at follow-up, that is, those with predominantly fixed stenoses and those with dynamic stenoses. Both subgroups are associated with a moderate elevation in inflow volumes.

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TABLES (1)

Table 1 Mean blood flow

	Age (years)	Total inflow (mL/min)	SSS outflow (mL/min)	Outflow as a % of inflow
Control n=14	31±13	880±150	415±75	48±6
Stenosis n=21	29±14	1070±240	370±100	35±5
p-value	0.7	0.01*	0.16	0.0001*
Follow-up patients				
Symptomatic n=7	24±11	1090±180	350±86	32±6
Remission n=7	24±11	850±210	390±81	47±12
p-value	1.0	0.02*	0.27	0.008*

SSS, superior sagittal sinus;*$p < 0.05$.

FIGURE LEGENDS **(1)**

Fig. 1 MR venogram of a patient with idiopathic intracranial hypertension showing no venous outflow stenosis.

Fig. 2 Axial reconstruction of an MR venogram with arrows showing intraluminal filling defects in both transverse sinuses.

Fig. 3 An MR venogram of an intracranial hypertension patient with arrows indicating a stenosis of the dominant transverse sinus.

Fig. 4 An MR vengoram of an intracranial hypertension patient showing a tapering stenosis with flow void (arrow) in the dominant transverse sinus.

Fig. 5 An oblique axial post-contrast reconstruction along the plane of the left transverse sinus showing a tapering stenosis but no filling defect.

Fig. 6 (a) An MR venogram of an intracranial hypertension patient while symptomatic showing a tapered stenosis (large arrow) of the dominant transverse sinus and collateral flow through the face, scalp and skull base (smaller arrows). No clot was visible on reconstructions. The total inflow was 780 mL/min with the superior sagittal sinus (SSS) outflow being 300 mL /min, i.e. 38% return. (b) An MR venogram of the same patient now 1 month later and in remission of her symptoms showing that a normal flow has been restored to the transverse sinus and the collateral flow abolished. The total inflow was now 640 mls/min and the SSS outflow 320 mL/min, i.e. 50% return. (c) An MR venogram of the same patient 5 months later and now symptomatic again. Note the return of the stenosis and the collateral flow.

Fig. 7 Two oblique MR venograms of the same patient before (a) and after (b) remission of symptoms showing no obvious change in the stenoses (arrows) of both transverse sinuses.

Fig. 8 A graphical representation of the cerebrospinal fluid (CSF) and venous outflow in intracranial hypertension. The venous and CSF pressures that have been appended are the mean values obtained by King et al.[4] Note that there is compression of the transverse sinus caused by the CSF pressure (arrow) and the transverse sinus pressure is 6 mmHg less than the CSF pressure. Because the compression is dynamic, variations in the CSF pressure will be transferred to the sinus and its pressure will always stay 6 mmHg below the CSF pressure, i.e. if fluid were infused into the spinal canal then the CSF pressure may be elevated by a few mmHg but the sinus pressure would also be elevated by the same amount. At the arachnoid granulations the pressure gradient is 5 mmHg meaning that no significant CSF absorption occurs whatever the elevation in CSF pressure.

Figure 1

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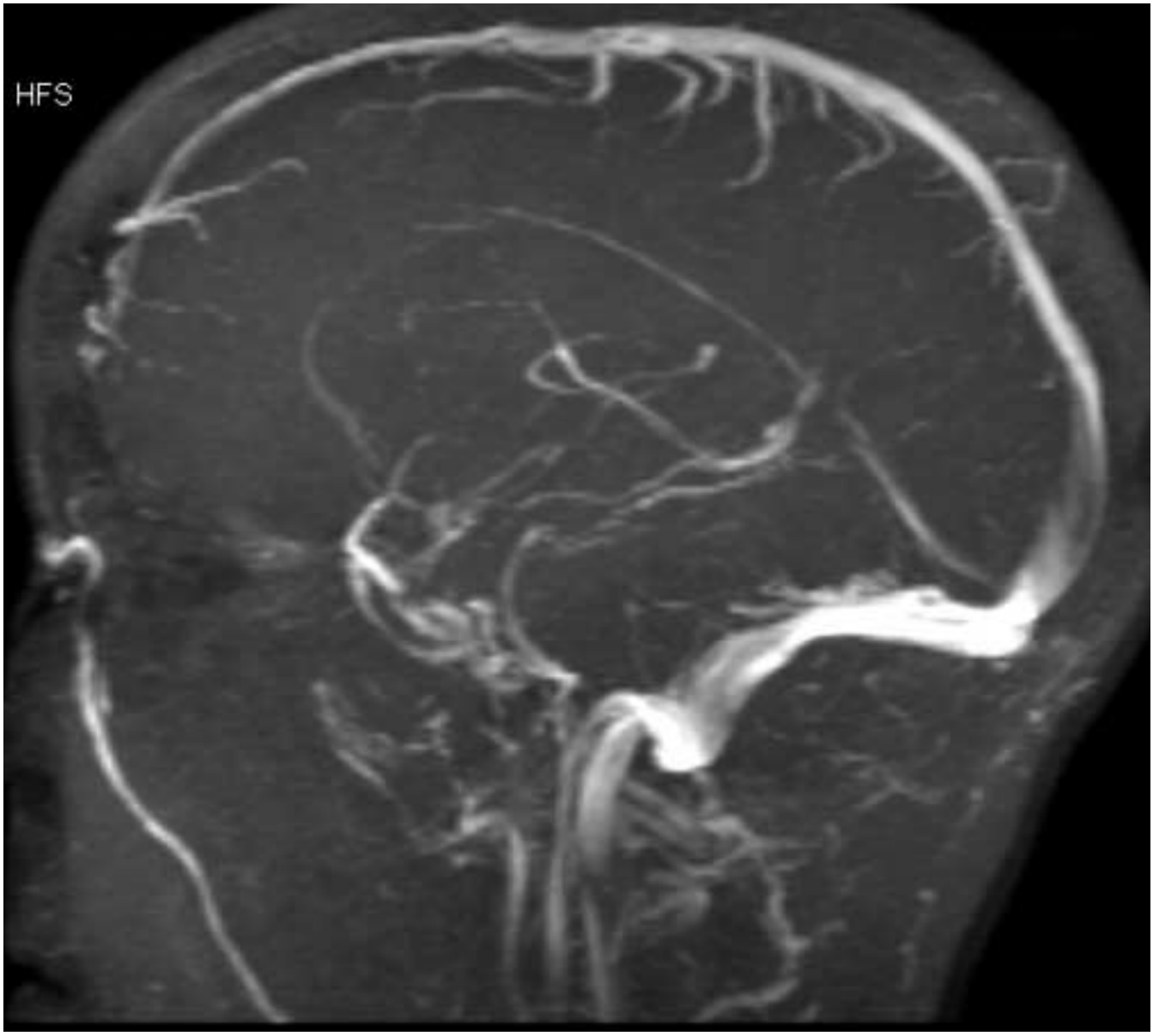

Figure 2

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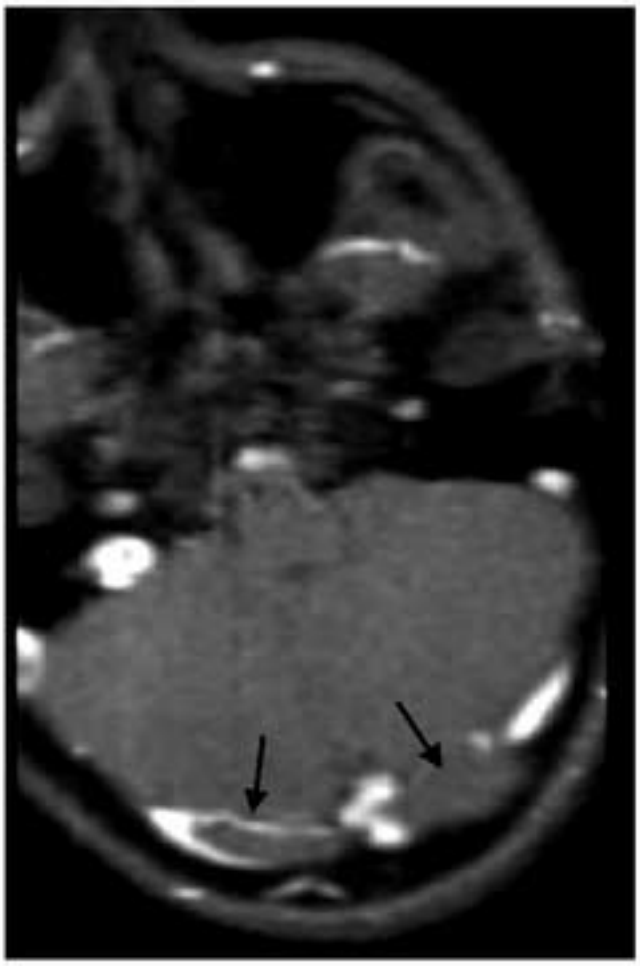

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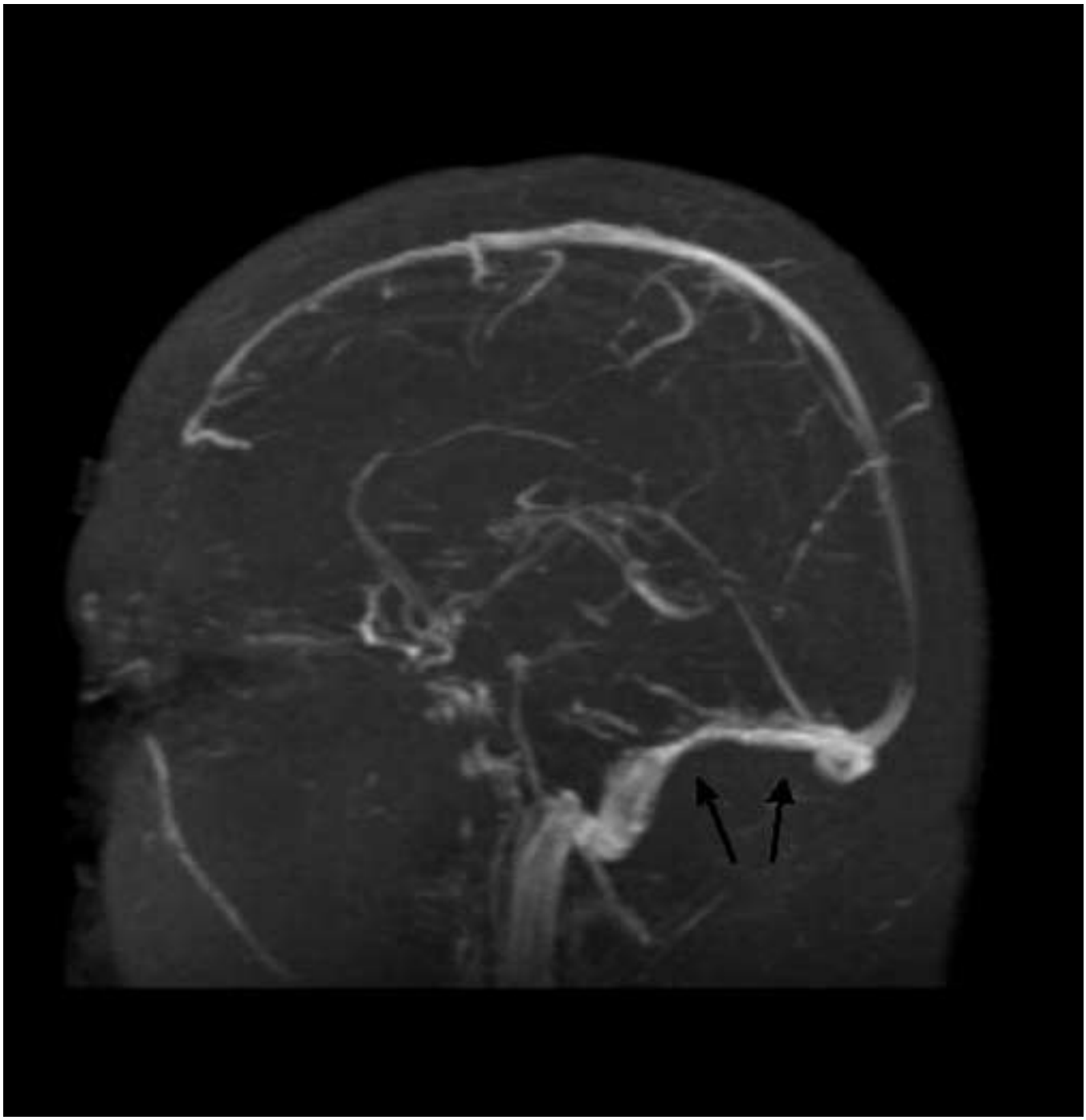

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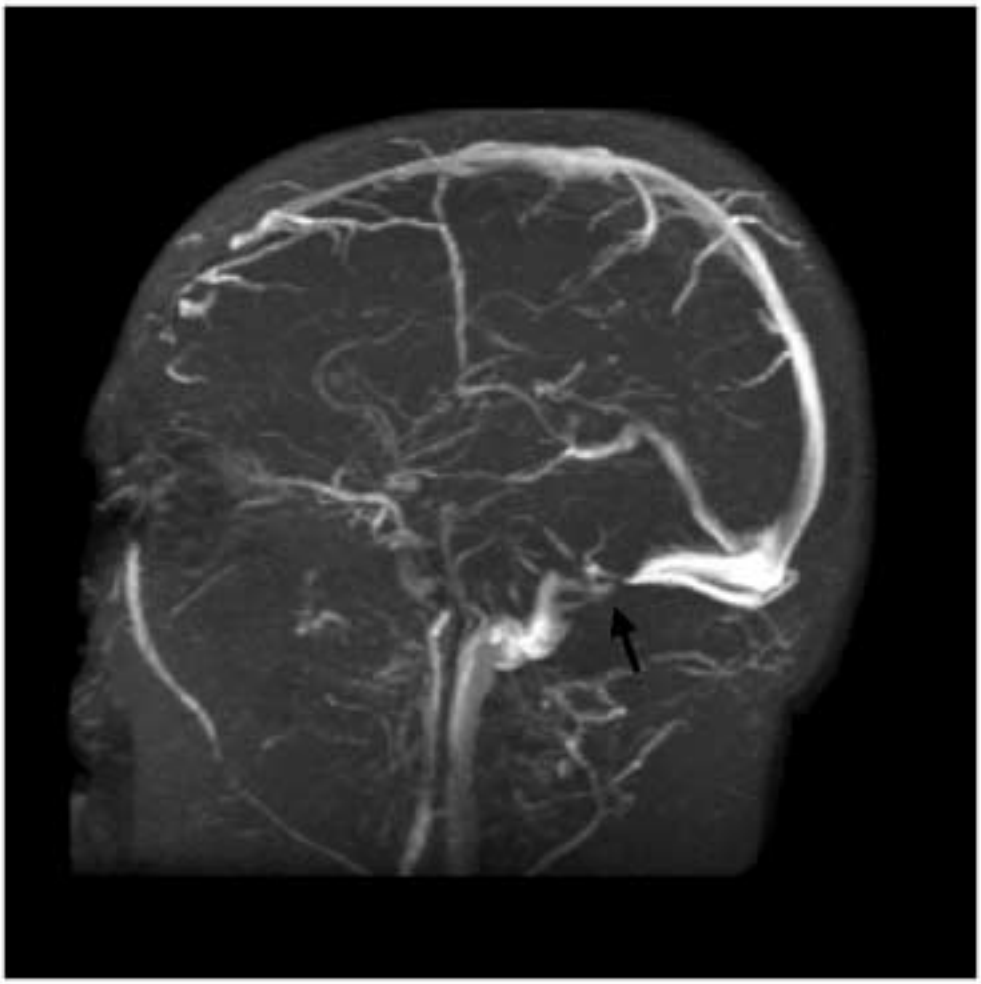

Figure 5

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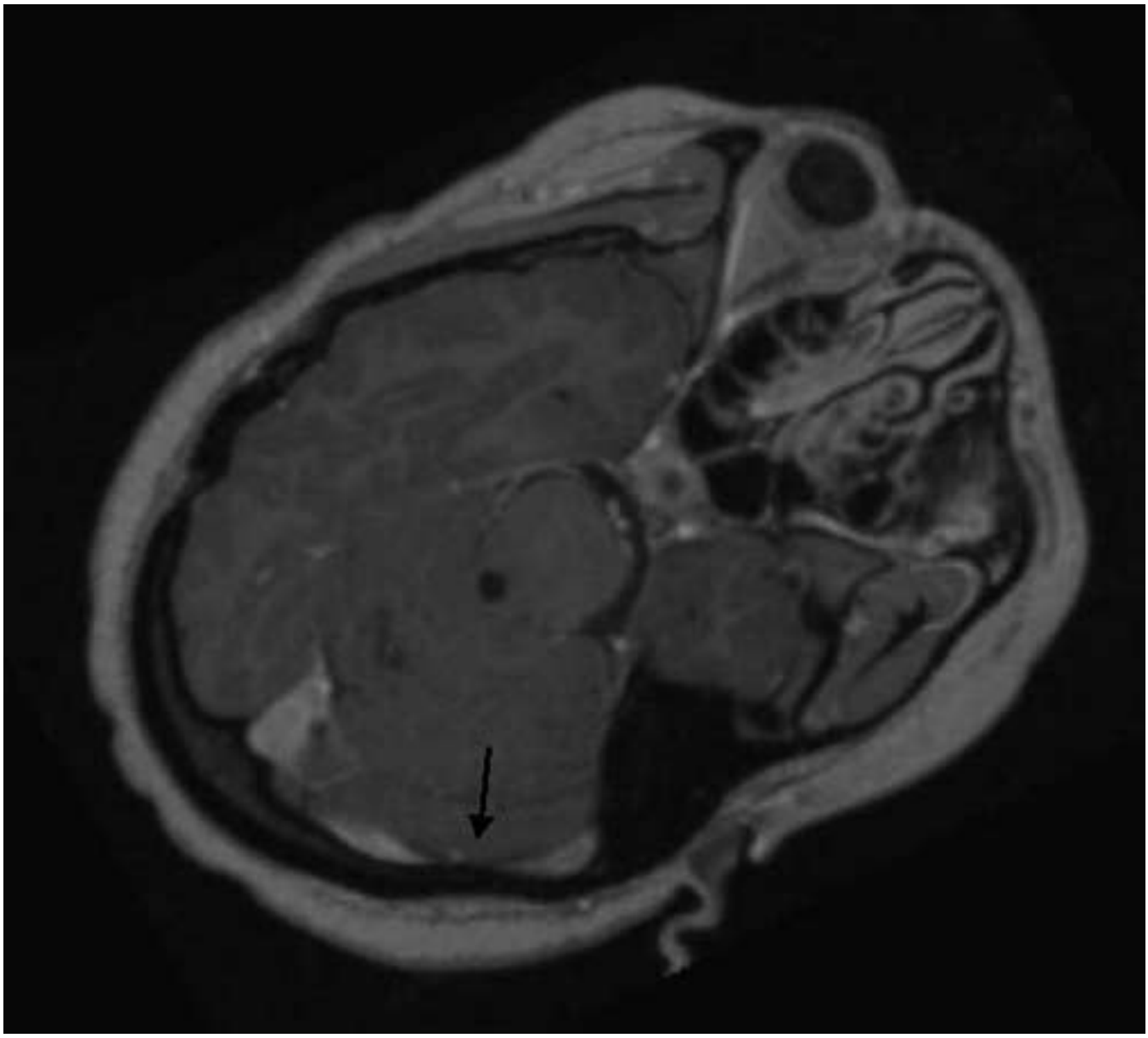

Figure 6a

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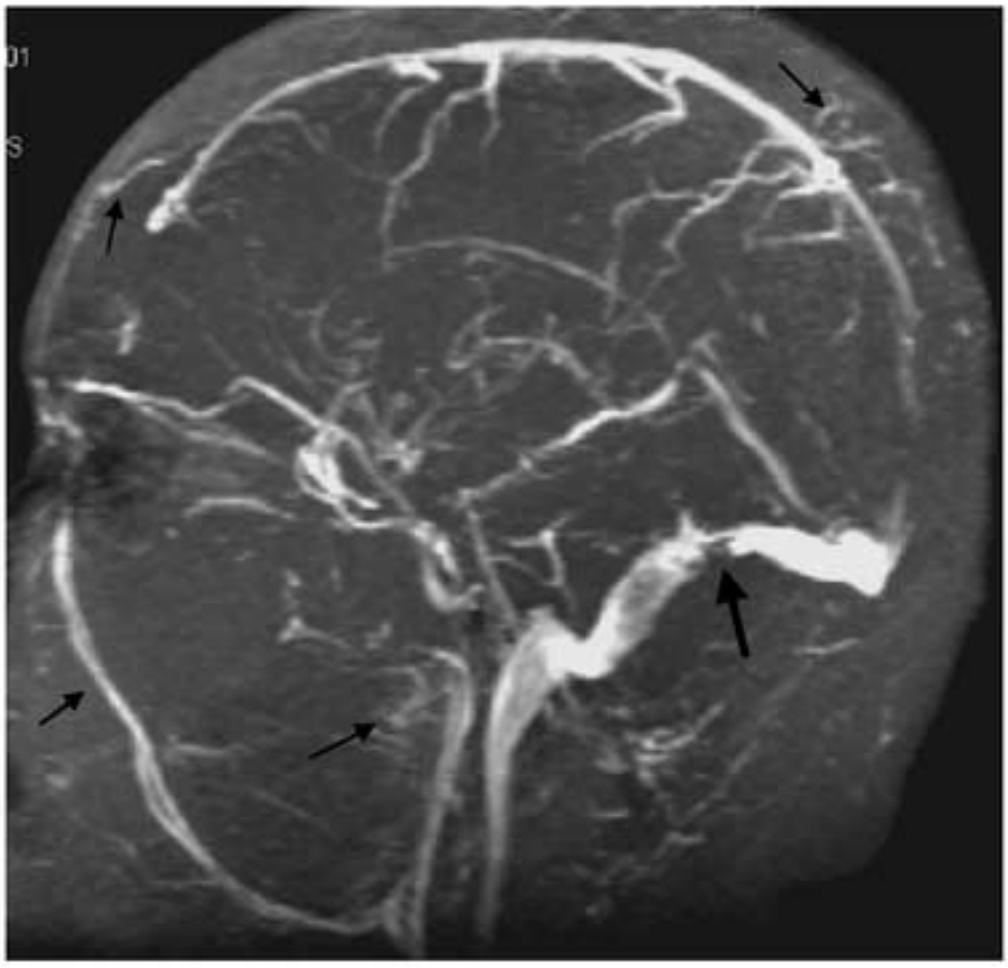

Figure 6b

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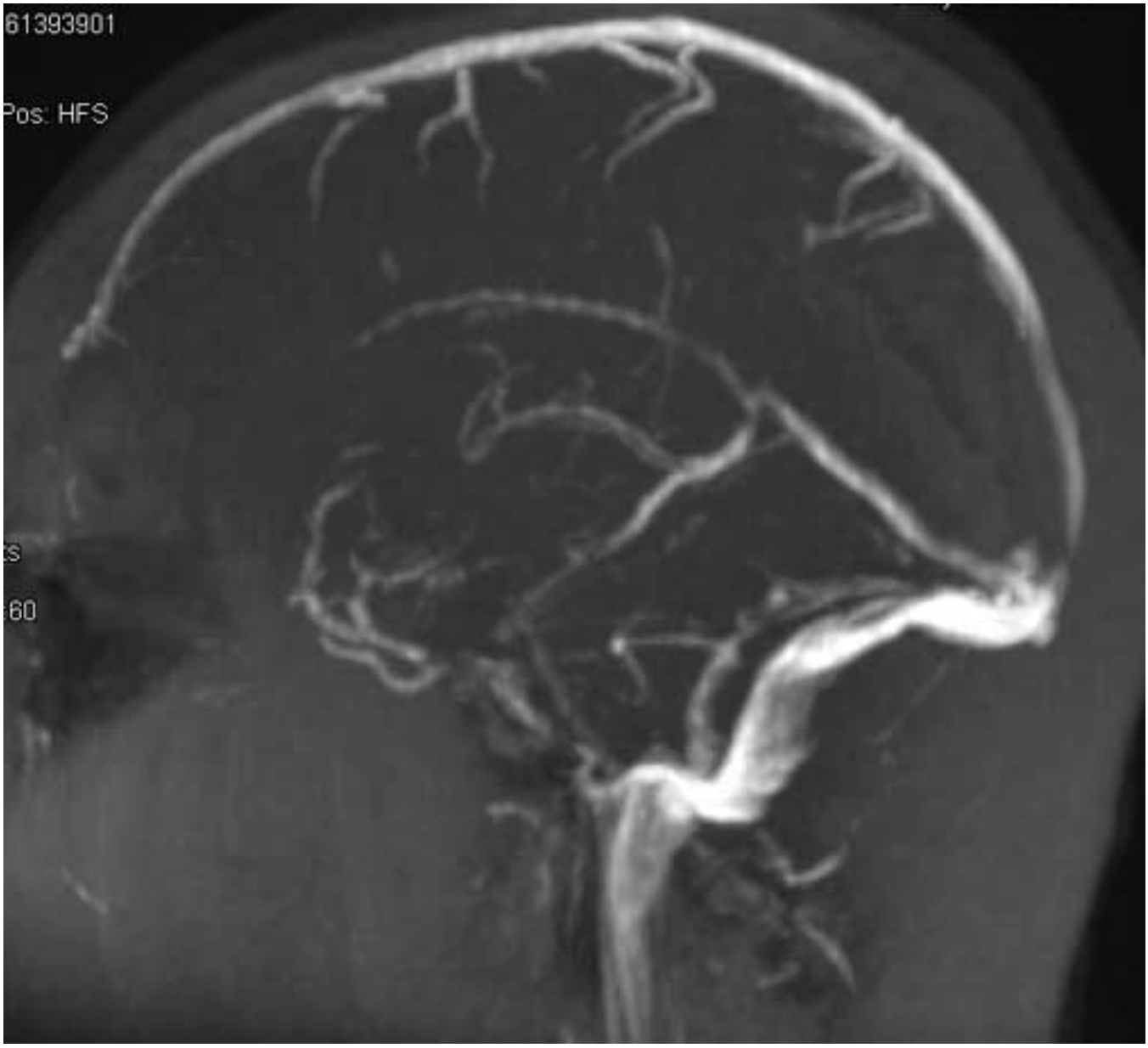

Figure 6c

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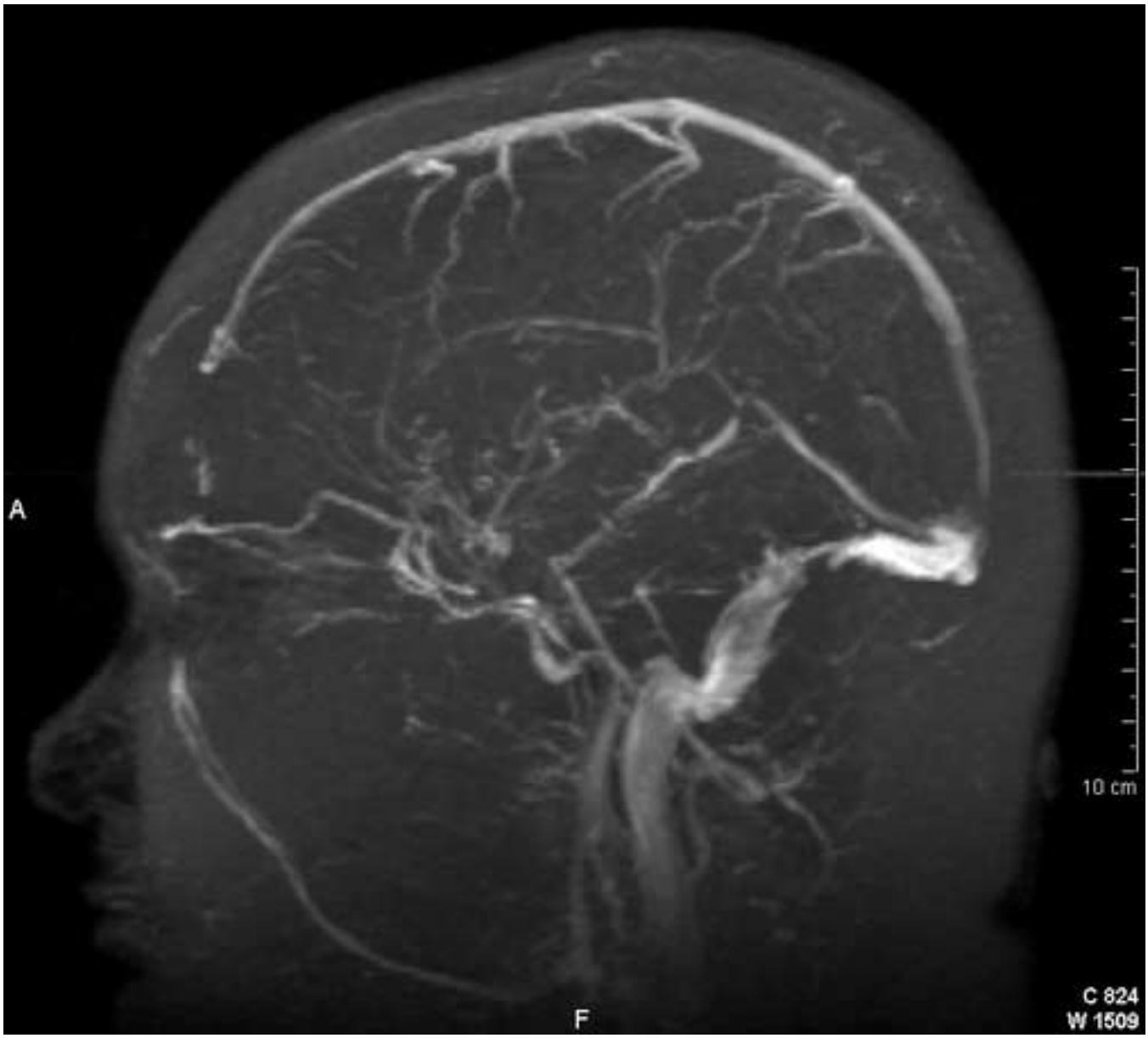

Figure 7a

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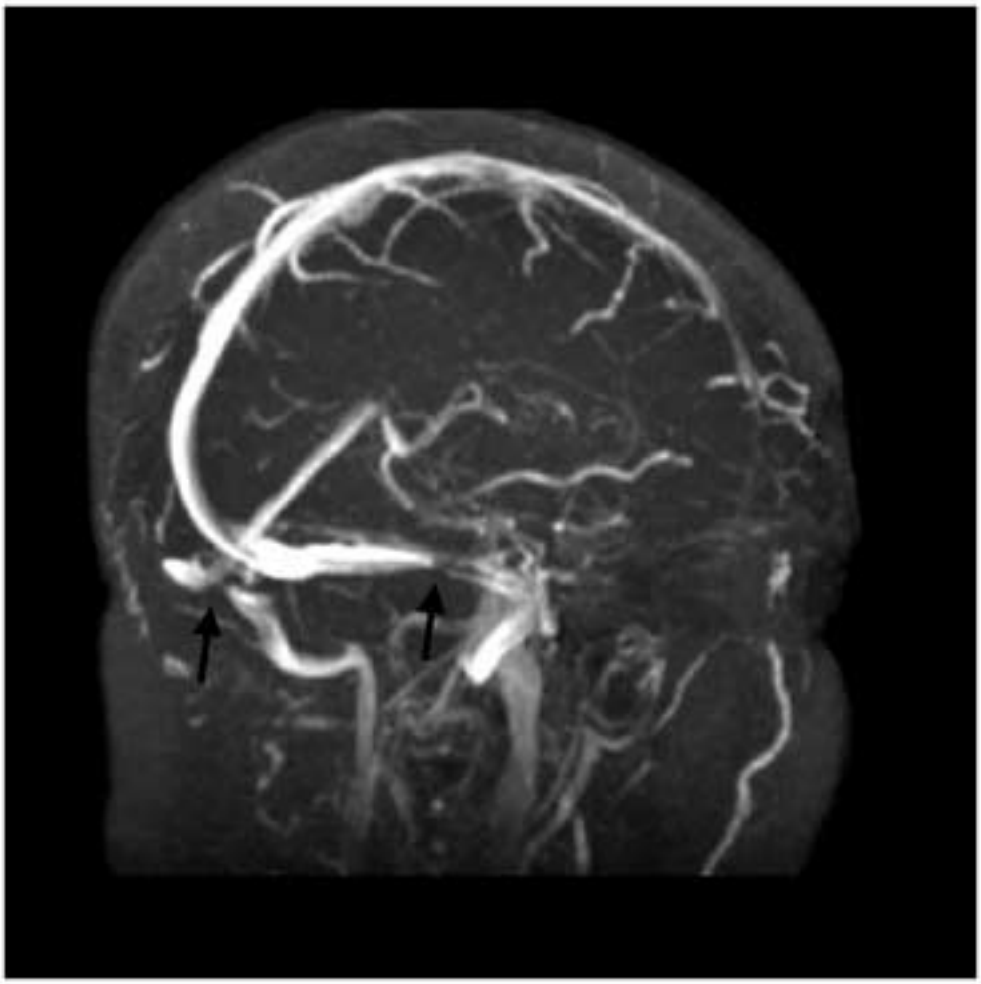

Figure 7b

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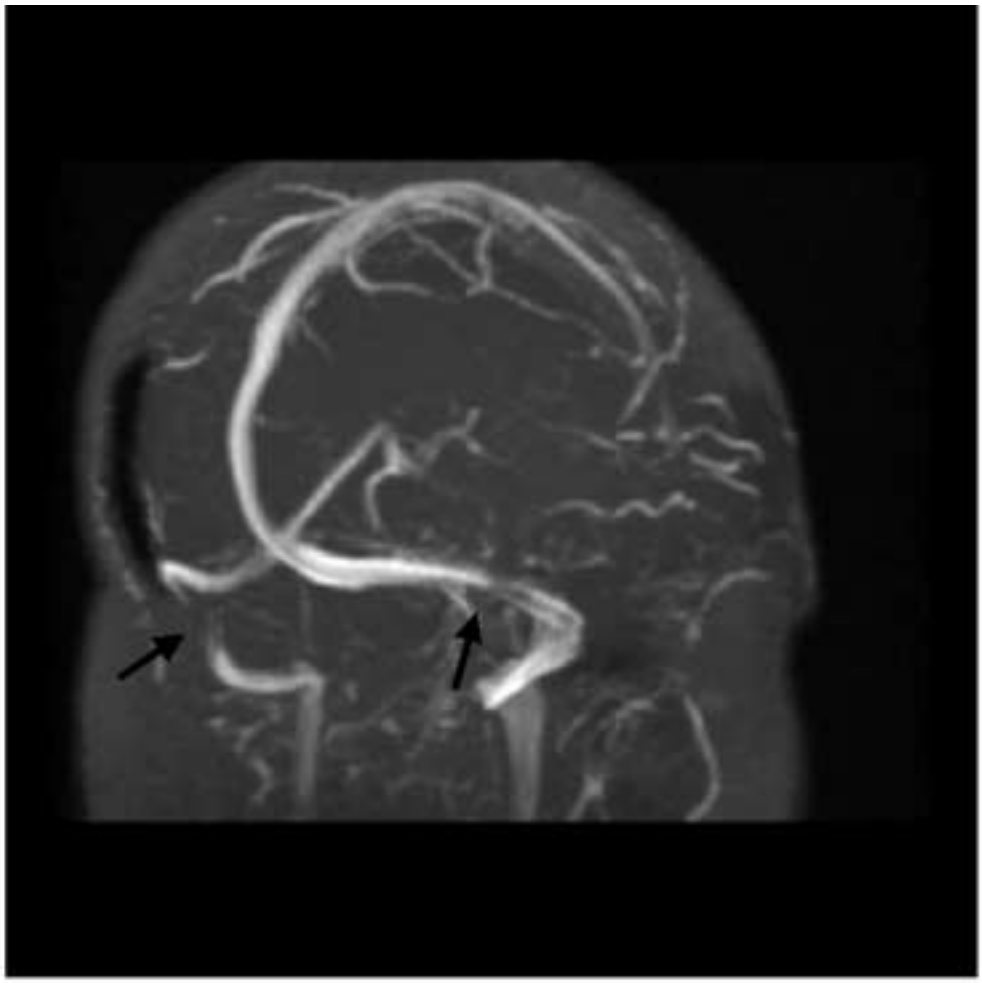

Figure 8

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