Vol 4 | Issue 18 | 2023

Interesting cases NEUROLOGY

Neurology Case Series

Vol 4 | Issue 18 | 2023

Dear Doctor,

Greetings from Micro Labs Limited.

We are happy to bring you "Neurology case series", which contains latest and interesting cases in the field of Neurology.

This issue contains

1. A case report on the misdiagnosis of the most uncommon form of sporadic Creutzfeldt-Jakob disease
2. A case report of vestibular seizures and spontaneous downbeat nystagmus caused by a ganglioglioma
3. A case report of an anterior cerebral artery dissection in a patient with an ipsilateral aplastic or twig-like middle cerebral artery
4. A case report of mechanical thrombectomy for persistent hypoglossal artery mediated acute basilar artery blockage

Should you require any specific article or medical information, please feel free to contact us at medicalservices@microlabs.in

Looking forward to your valuable feedback

Best Regards,

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Vol 4 | Issue 18 | 2023
Interesting cases NEUROLOGY

1. A case report on the misdiagnosis of the most uncommon form of sporadic Creutzfeldt-Jakob disease

Introduction:

Creutzfeldt-Jakob disease (CJD) is a neurological disorder caused by inherited or sporadic mutations in the prion protein-encoding gene. CJD classified as sporadic (sCJD) (most prevalent), familial, variant, or iatrogenic[1]. The prevalence of CJD is 1 to 2 cases per million people annually, with the majority (about 85%) of CJD cases being sCJD. Intellectual impairment, myoclonus, visual or cerebellar abnormalities, pyramidal or extrapyramidal symptoms, and akinetic mutism are all part of the specific clinical picture. The use of follow-up Magnetic resonance imaging (MRI) for CJD diagnosis has not been widely documented[2]. This case report describes a case of the VV1 subtype of sCJD, which was first misdiagnosed as schizophrenia, frontotemporal dementia, and herpes encephalitis. A subsequent MRI supported the diagnosis.

Magnetic resonance imaging (MRI) data interpretation may be challenging in the early stages of Creutzfeldt Jakob Disease, a follow-up MRI is necessary to provide an accurate diagnosis.

Form	Cause	Distinguishing Features
Sporadic/ Classical	Unknown	Affects mainly over 50s. S/S: Ataxia, dementia, spongiform changes, rarely plaques. Short course.
Familial/ Infectious	Inherited mutation in the PrP gene	Younger age of onset than sCJD, symptoms similar to sCJD, longer course.
Iatrogenic	Contamination during brain surgery, corneal transplant, dura mater grafts	Age dependant on the exposure source. Clinical and pathological symptoms indistinguishable from sCJD.
Variant	Exposure to BSE	Younger age of onset, longer duration of symptoms. Psychiatric signs present. Distinctive daisy plaques seen.

Forms of CJD

Case Presentation:

A 41-year-old male patient with rapidly developing dementia, akinetic mutism, and problems with walking and speaking reported to the Neuropsychiatry Department. He was unable to give further information due to his difficulties with speaking and mutism. According to his son, his condition began 7 months ago with amnesia, disorganised behaviour, and disorganised speech, which advanced rapidly and was accompanied by aphasia, apraxia, agnosia, and akinetic mutism in the previous 2 months. Throughout this time, he was hospitalised in several hospitals, both public and private, for various diseases such as herpes encephalitis, schizophrenia, and frontotemporal dementia, and he received multiple medications, but his health worsened rapidly day by day. The patient appeared awake and conscious on a general physical examination. His vital signs were normal, and no abnormalities in the cardiovascular, respiratory, gastrointestinal, or genitourinary systems

Vol 4 | Issue 18 | 2023
Interesting cases NEUROLOGY

were identified. Hypertonia, hyperreflexia, frontal ataxia, bradykinesia, gait apraxia, and aphasia were identified during a neurological examination. Routine lab tests, such as a complete blood count, fasting plasma glucose, serum electrolytes, a liver function test, a kidney function test, a vitamin B12 level, and a thyroid function test are in the normal range. A Cerebrospinal fluid (CSF) examination revealed normal cells, proteins, and glucose. He was diagnosed with herpes simplex encephalitis six months prior, and an MRI performed at that time revealed modest volume loss, flare high signal, and patchy diffusion restriction in bilateral frontotemporal lobes. CJD was considered the most likely diagnosis based on clinical features and the rapid onset of symptoms. On Diffusion Weighted Imaging (DWI), follow-up MRI revealed indications of widespread cortical damage in both cerebral hemispheres, as evidenced by diffusion restriction and involvement of bilateral corpus striatum (Fig. 1A): diffuse cortical injury on Susceptibility Weighted Imaging (SWI) (Fig. 1B); diffuse cortical hyperintensity on Fluid-attenuated inversion recovery (FLAIR) (Fig. 1C); and evidence of cerebral volume loss on T2 WI (Fig. 1D). On the right side, the EEG revealed lateralized periodic theta slow waves. The diagnosis of probable sporadic CJD and the diagnostic criteria for CJD were developed, according to the Centres for Disease Control and Prevention (CDC). The following symptomatic treatment strategy was proposed, along with supportive care: donepezil 5 mg initially, followed by gradually increasing it to 10 mg; memantine 5 mg initially, followed by gradual increases to 20 mg; and quetiapine 25 mg daily. The patient's condition drastically worsened and he became totally bedridden and incontinent after a one-month follow-up. After an estimated 13-month illness, the patient passed away.

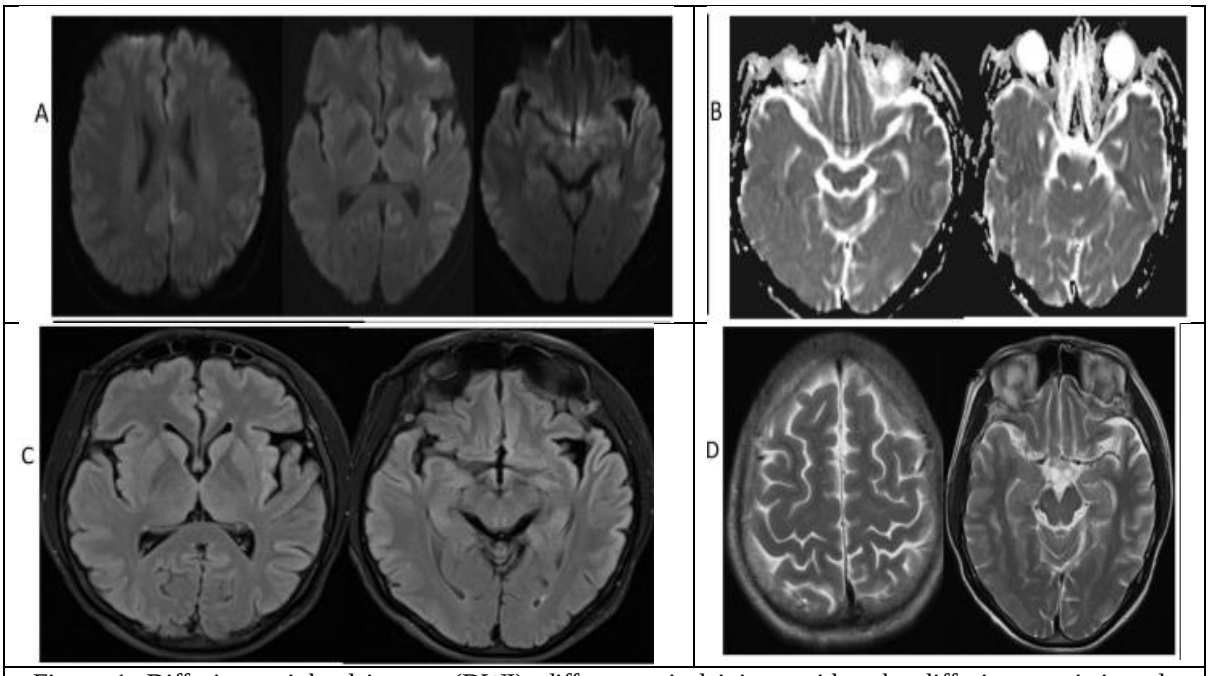

Figure 1: Diffusion-weighted images (DWI), diffuse cortical injury evident by diffusion restriction also affecting the corpus striatum, **Figure 1A:** susceptibility-weighted images (SWI), **Figure1B:** diffuse cortical injury evident by diffusion restriction, FLAIR, **Figure 1C:** diffuse cortical hyperintensity and **Figure 1D:** T2 WI, evidence of mild volume loss in both cerebral hemispheres.

Discussion:

Based on the CDC criteria, this patient was diagnosed with probable sCJD. sCJD is divided into seven subgroups, with the VV1 subtype being the most uncommon, accounting for about 1% of cases [(2)]. A study by Filatov A, et al. showed that, CJD is caused by hereditary or random abnormalities in the gene that codes for the prion protein [(1)]. In a similar study by Appleby BS, et al. demonstrated that, CJD in young people is incredibly rare and typically caused by a genetic mutation or external exposure [(3)].

Conclusion:

This case report highlights the need to treat CJD in people who arrive with rapidly progressing dementia. The proper diagnosis must be made with the use of a follow-up MRI because the interpretation of MRI data may be challenging in the early stages of the disease.

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Vol 4 | Issue 18 | 2023
Interesting cases NEUROLOGY

2. A case report of vestibular seizures and spontaneous downbeat nystagmus caused by a ganglioglioma

Introduction:

Vestibular epilepsy is described as recurrent simple, complex, or partial seizures marked by vertigo. Vertigo attacks are often the only clinical manifestation of epilepsy.[1]. Low-grade neoplasms called gangliogliomas (GGS) often affect people under the age of 30 and manifest as epilepsy and symptoms of mass impact. Gangliogliomas constitute 0.3-1.4% of all CNS tumours [2]. GGS are often identified as benign, low-grade tumours in children's and young adults' temporal lobes. In epilepsy surgical series, GGS are among the most prevalent histological diagnoses, accounting for 10% of the cases [1]. This is the first known case of a low-grade cerebral ganglioglioma that manifested as solitary vertigo and had the distinctive symptom of spontaneous downbeat nystagmus (DBN).

Brain tumours are a rare cause of solitary vertigo. Primary vertical nystagmus indicates possible temporal lobe damage, whereas déjà vu implies possible central vestibular abnormalities.

Case Presentation:

A 26-year-old man was referred to an otolaryngologist after experiencing recurrent rotatory vertigo, nausea, and palpitation. Without any circadian clock, the repeating event took place three to four times each day. The initial attack lasted roughly 10 minutes, with subsequent episodes lasting 10 to 15 seconds. Changes in head posture were not connected to the seizure's onset. These symptoms had been persistent for a half year prior to this appointment, unresponsive to either physical therapy or Lorazepam. By observing an essentially normal curve in the cervical spine, orthopedicians had already ruled out the cervical origin. Although a right temporal lobe lesion measuring 2.0×2.4 cm (Fig. 1) was detected by brain magnetic resonance imaging (MR) scanning, no causal relationship could be established since the electroencephalogram (EEG) was negative. Both the caloric test and the audiometry results were normal. However, the patient disclosed an episode of déjà vu during the follow-up session, which is strongly suggestive of additional neurological testing. In order to capture ictal sharp and slow waves of temporal lobe origin in conjunction with the

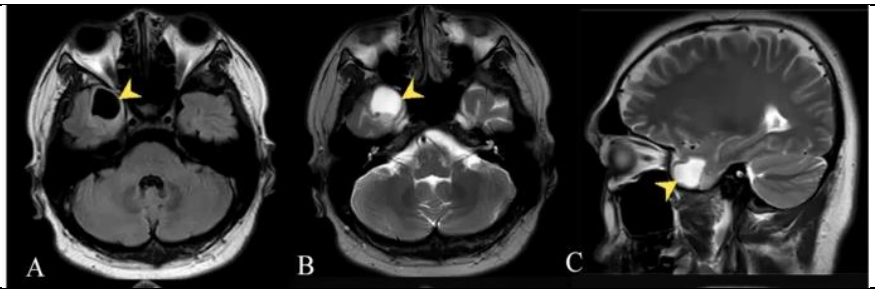

Figure 1 A B C: A lesion in the right temporal pole with hyperintensity on both T1- and T2-weighted images was discovered on pre-operative MRI.

Vol 4 | Issue 18 | 2023
Interesting cases NEUROLOGY

onsets of vertigo and associated palpitations, a 24 hour-EEG monitoring was also carried out. This result was supported by rhythmic shifts lasting 15–20 seconds that were picked up by synchronous EEG. As a result, a diagnosis of vestibular epilepsy was made. Only four days into the treatment of oxcarbazepine (300 mg bid), the frequency of the onset decreased to once daily or fewer, and the length was reduced to 2 seconds. Despite the efficacy of anticonvulsants, the patient insisted on surgical excision of the tumour.

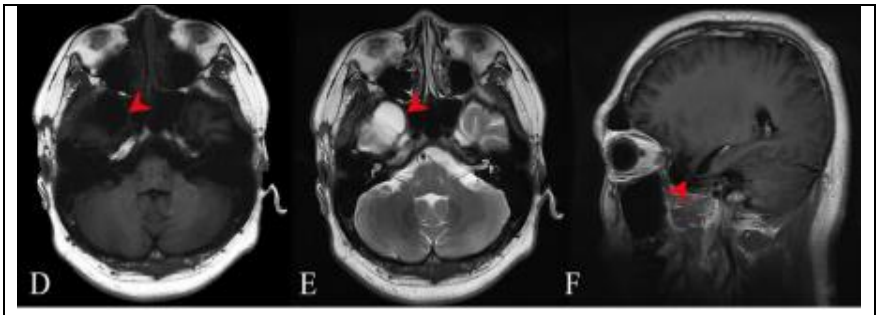

Figure 1 (D,E,F):six months after the surgery, follow-up MR imaging revealed increased T1 and T2 signal intensity but no localised enhancing lesion in the right temporal lobe, demonstrating post-operative modification without residual cancer.

The right temporal lobe lesion was removed. The tumor's histopathologic study revealed typical grade I findings, including restricted neuroglial tissue made up of mature ganglion cells that was surrounded by low-grade astrocyte-like cells with coarse processes. The BRAF-V600E mutation was diagnosed by molecular genetics. Following surgery, the patient had Oxcarbazepine (300 mg bid) for a further six months, during which time there were only three minor episodes. The patient has not had any symptoms for the past 19 months.

Discussion:

The most typical presenting symptoms of GGS are epilepsy, however this tumour has very rarely been linked to vestibular seizures. Two distinct signs suggested that DBN in this patient was caused by intracranial hypertension (ICH) rather than seizures. DBN became faint but remained visible with video-goggles after epileptic attacks were treated by Oxcarbazepine on Day 10. Oxcarbazepine inhibited repeated neuronal fringing and spreading by blocking the voltage-gated sodium channel. The inhibitory regulation of Purkinje cells in the cerebellum over anterior projections was disrupted in this patient due to persistent ICH caused by tumour development. The GABAergic neurons decreased the inhibitory signals from the damaged Purkinje cells to the focculus, resulting in DBN [(1)]. Tran TM, et al. demonstrated that Oxcarbazepine administration increased GABA receptor activation, lowering the excitability of anterior projections and thereby alleviating the DBN [(3)] .

Conclusion:

This study results showed that, brain tumours are an uncommon cause of isolated vertigo. Primary vertical nystagmus refers central vestibular abnormalities, while déjà vu suggests probable temporal lobe injuries. Continuous EEG monitoring for 24 hours offered critical evidence for identifying the causal lesion, which was occasionally ignored by conventional examinations.

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Vol 4 | Issue 18 | 2023
Interesting cases NEUROLOGY

3. A case report of an anterior cerebral artery dissection in a patient with an ipsilateral aplastic or twig-like middle cerebral artery

Introduction:

Anomalies in the middle cerebral artery (MCA) are less prevalent than those in the other primary intracranial arteries. Accessory MCA, duplicated MCA, fenestrated MCA, and aplastic or twig-like MCA (Ap/T-MCA) are some of the MCA aberrations. Collateral plexiform networks are present along with either a stenosis or a blockage of the MCA trunk in an Ap/T-MCA.[1] Aplastic or twig-like middle cerebral artery (Ap/T-MCA) is a very uncommon occlusive lesion with reticular arteries in the unilateral middle cerebral artery (MCA); its prevalence is very low, at about 0.088-1.17%. Collateral circulation occurs in Ap/T-MCA patients, especially from the ipsilateral anterior cerebral artery (ACA)[2]. This case describes a right A2 acute occlusion with restenosis caused by arterial dissection and ipsilateral Ap/T-MCA that was treated with thrombectomy first, followed by Wingspan stenting.

Patients with Aplastic or twig-like middle cerebral artery (Ap/T-MCA) must be aware of changes in the collateral pathway's arteries, particularly the ipsilateral ACA, due to the increasing hemodynamic burden.

Case Presentation:

A 77-year-old woman with untreated hypertension and hyperlipidaemia admitted to the hospital with acute left hemiparesis. The patient had a 6 on the National Institutes of Health Stroke Scale. At the time of admission, the electrocardiogram showed sinus rhythm and no atrial fibrillation. An infarction in the right medial side of the front lobe was detected using brain magnetic resonance imaging (MRI). A right A2 occlusion and a poorly visible right MCA were detected on brain magnetic resonance angiography (Figure 1). A right A2 was nearly occluded by reticulated arteries that had grown from the proximal right MCA, according to emergent digital subtraction angiography. These findings suggested that the left hemiparesis was caused by the right A2's blockage, which resulted in decreased flow of the right MCA.

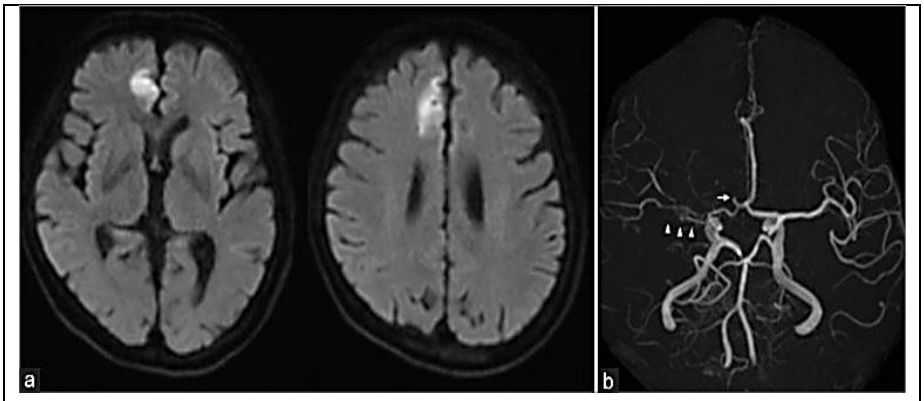

Figure 1: (a) Diffusion-weighted imaging of the right anterior cerebral artery on magnetic resonance imaging revealed a cerebral infarction. (b) Brain magnetic resonance angiography revealed a right A2 blockage (white arrow) and inadequate blood flow in the right middle cerebral artery (white arrowhead).

Vol 4 | Issue 18 | 2023
Interesting cases NEUROLOGY

A right A2 thrombectomy was carried out immediately. The lesion was determined to be a stenotic lesion after the right A2 segment's stent retriever was deployed. A dual-antiplatelet loading treatment (300 mg of clopidogrel and 200 mg of aspirin) was started orally. Mild stenosis was successfully recanalized when the stent was progressively extracted. With the presence of dilatation and the string indication, the lesion was deemed to be a dissection. One week after starting therapy, the patient's symptoms improved without any MRI recoil, and both the MRI and MRA revealed appropriate dilatations of the right A2 segment. Continued the treatment with the 75-mg clopidogrel and 100-mg aspirin dual antiplatelet therapy. However, 2 months following therapy, transitory left hemiparesis commonly occurred. Right A2 segment stenosis deterioration and a rise in right ACA region infarction were also seen on the MRI. As a result, prasugrel (3.75 mg) was used in instead of clopidogrel. Since the cerebral blood flow (CBF) in the right hemisphere significantly decreased according to the perfusion analysis of the CT scan, conducted another endovascular procedure under general anaesthesia 7 days following the second hospitalisation. Right internal carotid angiography revealed that the bilateral A2 segment was identified, and determined that it was possible to continue therapy with a temporary wedge of the left A2 segment. The dissecting right A2 section had a wingspan 2.5× 15 mm deployed. The right A2 segment was sufficiently dilated, and the right distal ACA flow was improved. The left A1 segment's wedge time was 15 minutes. Without any noticeable neurological damage or additional effects, the patient was discharged. Six months after the stenting, there was no restenosis [Figure 2].

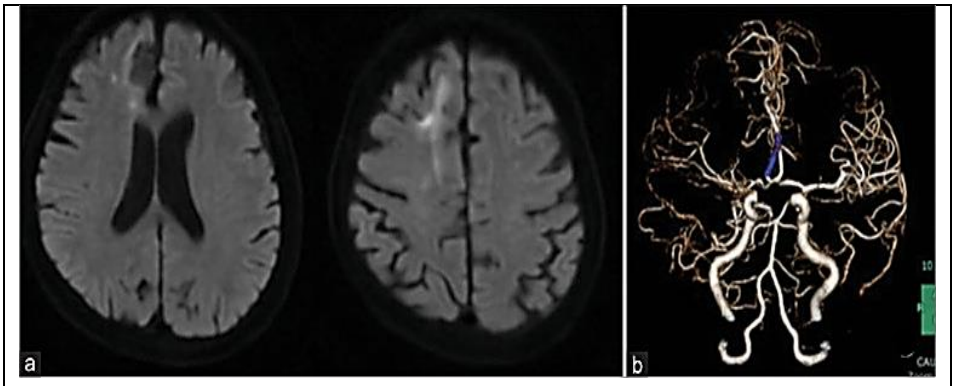

Figure 2: (a) Diffusion-weighted imaging revealed no new infarctions. (b) CT angiography revealed recanalization of the right A2 segment.

Discussion:

A rare vascular abnormality called Ap/T-MCA can occasionally lead to an ischemic infarction. Due to reduced CBF in the Ap/T-MCA region, cerebral ischemia takes place in these situations [(2)]. Furthermore, Cho KC, et al. showed a use of superficial temporal artery-to-MCA bypass to avoid stroke recurrences [(3)]. Because the ipsilateral ACA is the primary collateral channel in Ap/T-MCA patients, ipsilateral ACA stenosis can result in a substantial

CBF drop not only in the ACA region but also in the MCA territory, as observed in the present case. In a similar study by Takeda, et al. revealed a 62 years old male patient with TIA (Transit ischemic attack) and CI (Cerebral infarction) treated with superficial temporal artery - Middle cerebral artery (STA-MCA) anastomosis [(4)].

Conclusion:

This is the first case of a patient with ipsilateral A2 dissection complicating Ap/T-MCA who underwent Wingspan stenting. When treating collateral route dissection with Ap/T-MCA, aggressive action and short-term follow-up should be taken into account because the symptoms might get worse with time. Due to the rising hemodynamic load, patients with Ap/T-MCA must be watchful of alterations in the collateral pathway's arteries, especially the ipsilateral ACA.

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MICRO LABS SYNAPSE
Vol 4 | Issue 18 | 2023
Interesting cases NEUROLOGY

4. A case report of mechanical thrombectomy for persistent hypoglossal artery mediated acute basilar artery blockage

Introduction:

Proximal flow control, aspiration using a balloon-guiding catheter, and the injection of intra-arterial alteplase or tirofiban may enhance the clinical outcomes of ischemic stroke based on intracranial atherosclerotic stenosis (ICAS).

Basilar artery obstruction (BAO) is the most dangerous form of acute ischemic stroke. High mortality and disability rates as well as a poor clinical prognosis might result from slow recanalization [(1)]. Occlusion of the basilar artery, which accounts for around 10% of all ischemic strokes caused by intracranial proximal large-vessel occlusion [(2)]. Many studies have shown the safety and efficacy of mechanical thrombectomy (MT) for the treatment of acute large-vessel blockage in the anterior circulation. Persistent hypoglossal artery (PHA) is an uncommon carotid-vertebrobasilar anastomosis in adults. BAO and PHA have only been mentioned in a few research [(1)]. In this case, MT was provided to a 44-year-old male patient suffering from BAO, and PHA was identified during the procedure.

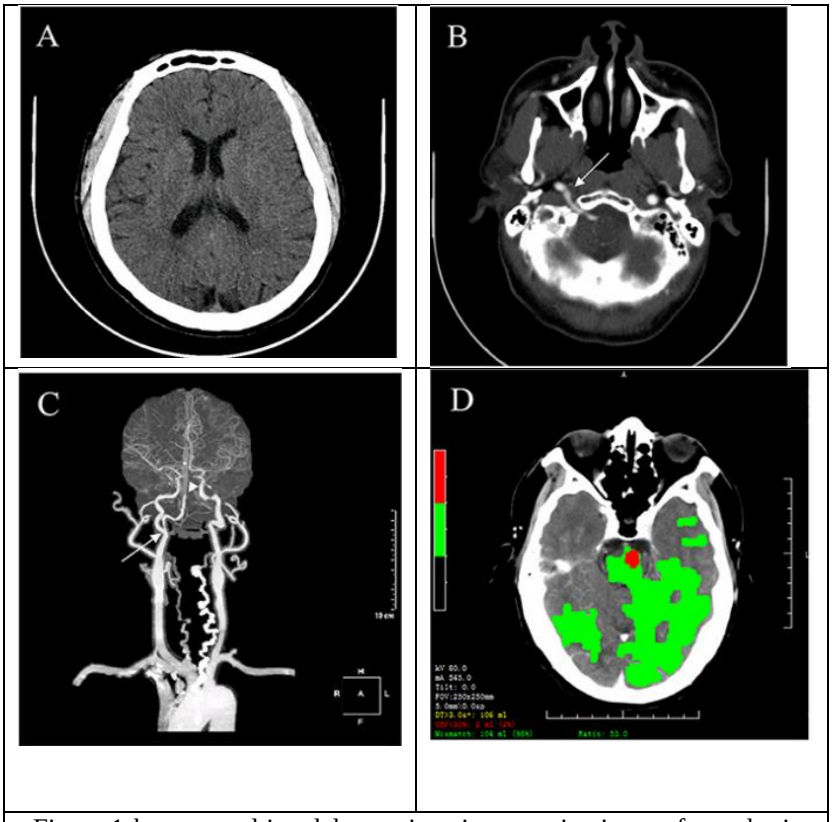

Figure 1shows a multimodal neuroimaging examination performed prior to surgery. (A) Computed tomography (CT) was used to rule out intracranial haemorrhage. (B) A cross-sectional contrast CT examination indicated an improper anastomosis. (C) Anterior-posterior CT angiography revealed basilar artery blockage. (D) The mismatch volume was calculated by automatically evaluating the volumes of the infarct core and penumbra from CT perfusion.

Case Presentation:

A 44-year-old man with dysarthria and an unsteady gait was hospitalised in the stroke unit for two hours. The baseline Modified Rankin Scale (mRS) score was 1, whereas the National Institutes of Health Stroke Scale (NIHSS) score was 4. The National Institutes of Health Stroke Scale (NIHSS) score raised to 33 and the mRS score to 4 after 95 minutes as the clinical symptoms progressively got worse. The patient was a chain-smoker, and nifedipine was used to treat primary hypertension. Thrombolytic treatment was contraindicated since the patient also had a history of an unexplained intracranial haemorrhage. Intracranial haemorrhage was ruled out by multimodal

computed tomography (Figure 1A). The basilar artery (BA) was absent, and the internal carotid artery (ICA) and the intracranial section of the vertebral artery (VA) situated in the hypoglossal canal (Figures 1B, C) had an aberrant anastomosis. CT perfusion was used to measure the extent of the region with cerebral blood flow greater than 30% and a delay period of 3 seconds. MIStar software (Apollo Medical Imaging Technology) was used to determine the mismatch volume, which was 104 mL (Figure 1D).

Right radial access digital subtraction angiography revealed full BAO and a modified Thrombolysis in Cerebral Infarction (mTICI) level of zero. PHA began at the right ICA and anastomosed to the right VA. A right subclavian artery angiography revealed that the hypoplasia was caused by VA. Additionally, a thin left VA and a left middle cerebral artery blockage with collateral vascular development were seen. Because of the challenges found in establishing MT access with appropriate supporting strength via transradial access, transfemoral access was chosen. A 4×20 mm Solitaire AB stent was inserted from the P1 segment of the left posterior cerebral artery (PCA) via the PHA in order to perform thrombectomy. Angiography revealed thrombotic migration to the tip of the BA as well as significant stenosis in the trunk of the BA. After angioplasty with a 3× 9 mm noncompliant balloon (Gateway, Stryker Neurovascular, Kalamazoo, Michigan), thrombectomy with proximal aspiration was done from the right PCA. At a mTICI of level 3, complete recanalization of BA flow was accomplished. There was no evident cerebral haemorrhage on postoperative CT, so a loading dose of tirofiban was given. After the operation, the patient received a half-loading dose of intravenous antiplatelet treatment (Tirofiban) and his systolic blood pressure was carefully maintained between 130 and 140 mmHg. Following surgery, MRI revealed many tiny, freshly created infarcts in the cerebellum and medulla oblongata. An examination of the BA trunk using CT angiography (CTA) three days after the procedure revealed satisfactory patency. With an NIHSS score of 25 and an mRS score of 5, the patient was discharged after 10 days for additional rehabilitation. The NIHSS score dropped to 18 and the mRS score to 4 during the 10-month follow-up.

Discussion:

The carotid artery and vertebrobasilar system have a number of anastomotic channels that occur in the early stages of human embryonic development. These channels are crucial for irrigating the posterior circulatory bed before it completely develops. This is the first report on the use of a stent retriever with aspiration in the treatment of BAO through PHA. Rescue angioplasty after MT is required for preserve forward blood flow, much like the pathophysiology of ICAS. More significantly, even though the BA had been totally recanalized and had a mTICI level of 3, the migration of embolic debris and the lack of collateral circulation may have left the patient with a very unfavourable prognosis [(1)]. In a previous study by Ospel JM et al, demonstrated that A balloon-guiding catheter may also be more useful for flow management in the proximal ICA, and this option should be carefully examined in future clinical practise [(3)].

Conclusion:

BAO with PHA is relatively rare, and preoperative multimodal CT scans can detect such vascular variations and help choose an interventional strategy for early recanalization.

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