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Neurology Case Series

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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 Wernicke Encephalopathy in a Non-Alcoholic Obese Adolescent**
2. **A case report on the early manifestation of benign paroxysmal positional vertigo**
3. **A Rare Case report on Cardioembolic Spinal Stroke in a Young Female**
4. **A case report on the entrapment of an embolic protection device inside a stent during carotid artery stenting**

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,

Medical Team,

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1. Case Report on Wernicke Encephalopathy in a Non-Alcoholic Obese Adolescent

Introduction:

Wernicke's encephalopathy (WE) is a serious and life-threatening condition caused by thiamine deficiency. WE's prevalence in the general population has been estimated to be between 0.4 and 2.8%.[1] WE is typically seen in adults who use alcohol excessively. Severe malnutrition, high-calorie malnutrition, and prolonged fasting can all lead to this condition in individuals of any age. If left untreated, WE can progress to Korsakoff syndrome, an irreversible neuropsychiatric state characterized by anterograde and retrograde amnesia as well as confabulation. Thiamine is essential for both the Kreb's cycle and the pentose phosphate pathway, making thiamine shortage a significant systemic consequences.[2] This case report described a rare case of Wernicke encephalopathy in a non-alcoholic, obese adolescent.

Knowledge of risk factors, clinical symptoms, and brain MRI data is necessary for an accurate diagnosis and course of therapy for Wernicke encephalopathy in pediatric patients.

Case Presentation:

A 17-year-old non-alcoholic obese male with a 4-month history of significant weight reduction (34 kg) and strict diets was hospitalized for persistent vomiting and stomach pain for a week. A recent diet consisted of a three-week water fast followed by three weeks of modest portioned meals. Upon admission, the patient's test results showed significant AST (95 u/L) and ALT (169 u/L) levels, but low alkaline phosphatase (40 u/L). His total blood count, coagulation studies, and thyroid function were normal. A CT scan of the abdomen revealed a liver with fatty infiltration. Antiemetic and supportive therapy were initiated. On the second day of hospitalization, the patient continued to have nausea and non-bilious, non-bloody emesis with a flat affect. Abdomen ultrasound revealed no evidence of superior

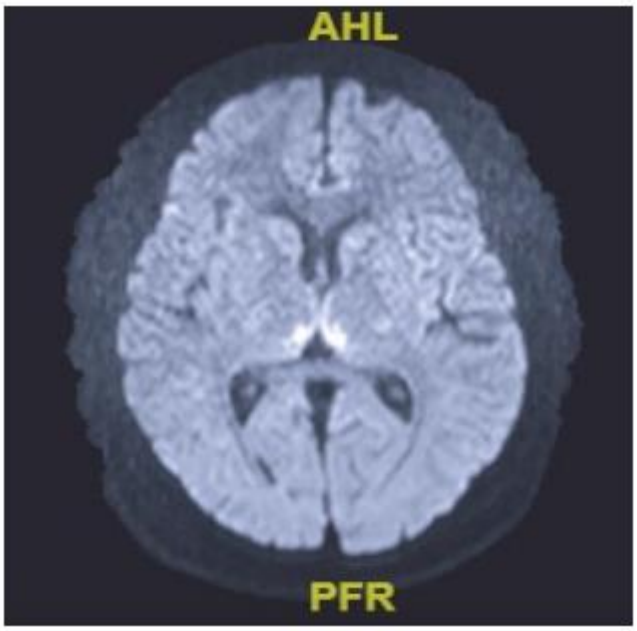

Figure 1: Brain MRI on the eighth day of hospitalization. Wernicke encephalopathy is indicated by an aberrant increase in T2 signal in periaqueductal gray matter and medial thalami.

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mesenteric artery (SMA) condition. He underwent esophagogastroduodenoscopy, which revealed minor chronic gastritis and lactase enzyme deficiency from a duodenal biopsy. On day 3 of hospitalization, he was tired and unable to walk independently. He continued to experience emesis after feedings, which improved with Phenergan. On day 6 of hospitalization, the patient reported no emesis and tolerated a clear liquid diet. Extensive lab workup for Epstein-Barr virus (EBV) and cytomegalovirus (CMV) was conducted, including EBV viral capsid antigen (VCA) IgG positive, EBV Nuclear antigen was positive, CMV IgG Ab was reactive, while EBV VCA IgM and EBV early antigen (EA) were negative. On the eighth day of hospitalization, the patient had hallucinations, dysconjugate gaze, and limb weakness. The patient got a brain MRI to rule out occupying lesion or pseudotumor cerebri, which revealed Wernicke encephalopathy (Figure 1). The patient was given 500 mg of thiamine intravenously three times daily. Prior to thiamine delivery, vitamins A, B1, B6, and B12 levels were found to be normal. Vitamin C, Vitamin D, and Vitamin E (alpha tocopherol) levels were low. On day 11 of hospitalization, the patient was conscious, had better cognitive, and could tolerate oral intake. The patient's brain MRI confirmed Wernicke encephalopathy (Figure 2), whereas the spine MRI revealed degenerative disc desiccation at L4-5 and L5-S1. The patient made slow and steady improvement with walking and was finally discharged to an inpatient rehabilitation center.

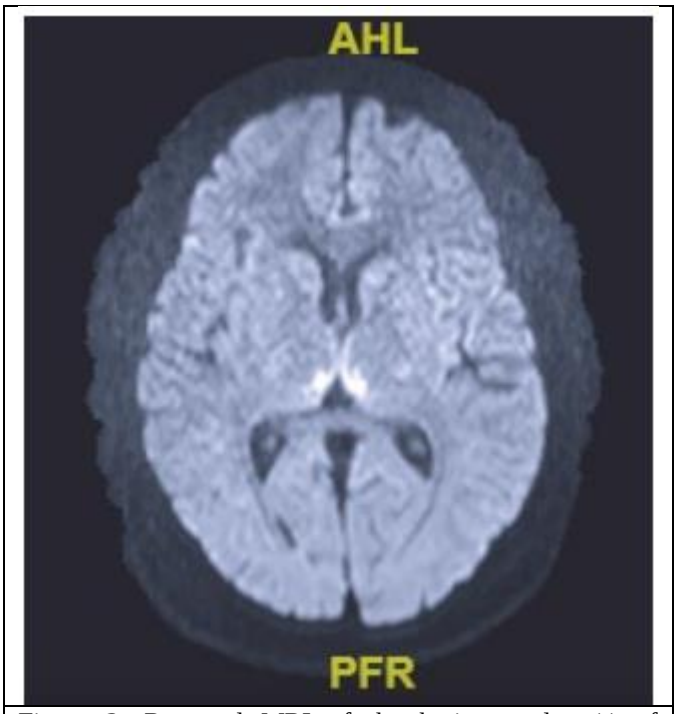

Figure 2: Repeated MRI of the brain on day 11 of hospitalization. There was no difference in the appearance of the brain compared to prior studies. The data imply Wernicke encephalopathy.

Discussion:

Patients with imbalanced diet, frequent vomiting, and diarrhea should be more likely to develop Wernicke encephalopathy. Thiamine administration is crucial due to the unclear nature of the illness, as delayed treatment might result in neurological deterioration and possibly death.[2] M. Jimenez Cabanas et al. showed that the patient's clinical state improved rapidly when the diagnosis was confirmed and thiamine was administered.[3]

Conclusion:

Accurate diagnosis and treatment of Wernicke encephalopathy in children requires understanding risk factors, clinical manifestations, and brain MRI results.

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2. Case report on the early manifestation of benign paroxysmal positional vertigo

Introduction:

Benign paroxysmal positional vertigo (BPPV) is an inner ear condition caused by semicircular canalolithiasis.[1] BPPV is the most prevalent kind of vestibular vertigo, affecting around 17-42% of people with this disorder. Patients with BPPV experience transitory periods of vertigo caused by head position shifts. The lifetime prevalence of BPPV is estimated to be 2.4%, with a much greater 1-year prevalence among the elderly than in other age groups.[2] The prevalence of vertigo in elderly people is estimated to be around 30%, whereas in paediatric patients it is approximately 10%. This problem can be treated with repositioning techniques. Currently, no patients under 5 years old have been described in the literature.[1] This case report describes a 4.5-month-old female infant presented to the emergency room with many periods of nystagmus.

The diagnosis of right lateral semicircular canalolithiasis is supported by the clinical symptoms and repositioning procedures. Comprehensive diagnostic evaluations are essential because benign paroxysmal positional vertigo condition is uncommon at young age.

Case Presentation:

A 4.5-month-old female infant was taken to the emergency room after having many episodes of nystagmus. During a single episode of horizontal quick eye movements lasting 30 seconds, parents reported that the infant was unresponsive and vomiting was severe. The differential diagnosis for paroxysmal aberrant eye movement in babies has been established. The most prevalent etiology is occulogenic, however it is less possible due to the paroxysmal character of the symptoms. Despite the negative EEG results during the assumed interictal stage, epileptic nystagmus might be a potential cause. Intoxication with alcohol, pharmaceuticals, or narcotics can cause nystagmus. This can be caused by direct cerebellar poisoning or, like alcohol, by changing the specific gravity inside the semicircular canals. Neuroinfections can induce nystagmus and may require a spinal tap for treatment. BPPV, vestibular neuronitis, labyrinthitis, perilymphatic fistula, and superior semicircular canal

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dehiscence are among the possible causes of peripheral vestibular dysfunction. Whiplash-related diseases can cause abnormal eye movements, including nystagmus, due to compromised cervico-oculomotor functions. A comprehensive history of past injuries is necessary. Functional nystagmus is considered but unlikely in this situation due to the patient's age. The physical examination and acute blood tests (ions, CRP, liver and kidney function) were normal in the presenting patient. Transcranial ultrasonography revealed expansion of the subarachnoid space. Due to the wide range of diagnoses for nystagmus in infants, the baby was brought to the hospital for additional assessment and monitoring. At 33 weeks and 3 days' gestation, the baby was born via C-section after a natural lengthy labor. ATB treatment was administered for 48 hours to treat suspected sepsis, which was later confirmed as fetal inflammatory response syndrome. Blood tests indicated minor thrombocytosis and neutropenia, along with a modest rise in the Fahraeus-Westergren test (34/57) indicating a probable parainfectious result.

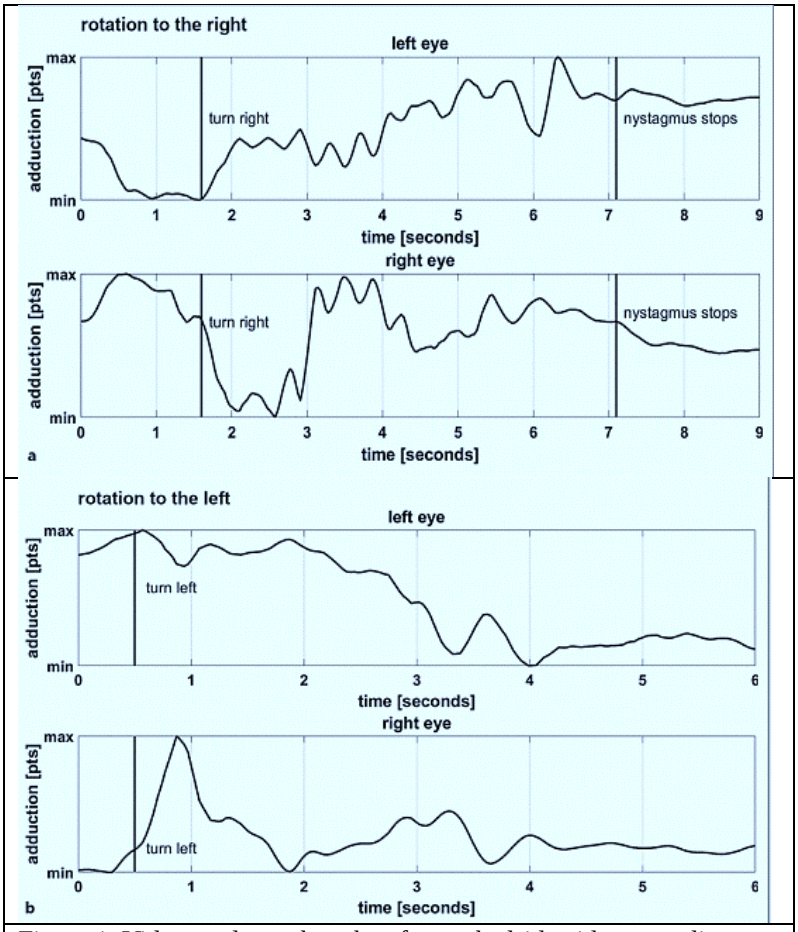

Figure 1: Video-oculography taken from a bedside video recording.

The neurological and otorhinolaryngological exams were normal. BAEP revealed bilaterally slightly longer wave III and V latencies. The brain's magnetic resonance revealed bilateral expansion of SA spaces (BESS, or benign enlargement of the subarachnoid space). During the hospital stay, the mother noticed a correlation between the baby's position and nystagmus occurrences. The diagnostic roll test revealed horizontal nystagmus lasting less than 30 seconds. A diagnosis of transitory positional geotropic nystagmus, most likely caused by right lateral semicircular canal canalolithiasis, was established. To address the lack of electrophysiological documentation during the fit, this study created a semi-automatic pathway for acquiring video-oculography from existing recordings. The Kanade-Lucas-Tomasi feature-tracking technique was used in MATLAB R2021b to monitor motion at chosen places of interest. This instance included the patient's pupil and medial palpebral commissures. The pupil's Euclidean distance from the neighboring one was determined. To account for zooming, the medial commissures' Euclidean distance was used to normalize the image. In online supplementary video 1,

passive rotation to the right was followed by horizontal bidirectional nystagmus at 2.5 Hz, which declined to 1.5 Hz after 4 seconds and stopped completely after 3 seconds. The amplitude remain steady during the fit. Nystagmus disappeared after turning to the left. Figure 1 illustrated the dynamics of eye movements. The vestibulo-ocular reflex caused a rapid shift in feature proportions after a head rotation, making it difficult to accurately measure movement amplitude.

Discussion:

In this case a 4.5-month-old child was diagnosed with positional nystagmus on right side. Classic clinical symptoms and rapid remission with repositioning procedures confirmed the diagnosis of right lateral semicircular canal lithiasis. In the present case, the Lempert roll maneuver ("barbecue roll") was used to get rapid results. Parents were told to repeat the procedure at home if they had remission, which was successful in resolving the issue.[1] In contrast, Yue Lou et al. concluded that the BPPV diagnosis and treatment system was more successful than classical manual repositioning for treating the condition.[3]

Conclusion:

This case demonstrated a rare occurrence of BPPV in a newborn. The clinical symptoms and repositioning techniques support the diagnosis of right lateral semicircular canal lithiasis. Given the rarity of this illness at such a young age range, comprehensive diagnostic assessments were crucial. This paper presented an automated video analysis pipeline to detect aberrant eye movements in a home setting.

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3. An uncommon case report on cardioembolic spinal stroke in a young female

Introduction:

This case emphasized the significance, especially in the absence of typical symptoms on neurological examination, of a comprehensive and rapid investigation to rule out Spinal cord infarction (SCI) in patients presenting with acute paraparesis.

Spinal cord infarction is a rare but severe condition caused by a variety of pathologic situations, and is less common than ischemic brain damage. It accounted for just 1–2% of all neurological vascular crises and was similar to other acute myelopathic syndromes in many clinical characteristics.[1] It results in ischemia, infarction, and spinal cord dysfunction due to an immediate interruption of the blood supply to the spinal cord. The clinical presentation was determined by the arterial region implicated, with the most frequent being anterior spinal artery syndrome. Compared to high cervical spinal strokes, lower thoracolumbar spinal strokes were more frequent. Iatrogenic causes account for the majority of spinal stroke cases.[2] This was a case report of a 48-year-old woman who was hospitalized with acute paraparesis and a spinal cord infarct on magnetic resonance imaging.

Case Presentation:

A 48-year-old patient with a history of depression and no known vascular risk factors reported experiencing sudden lower limb weakness and discomfort that was related to lower back pain. Upon neurological assessment, she exhibited complete consciousness and alertness, without any neck stiffness, cranial nerve impairments, or upper limb deficits. There was weakness in the proximal 3–4/5 limbs, but more distal 1-2/5 weakness in the lower limbs. On the left patellar tendon, deep tendon reflexes were fast, but on both Achilles, they were hypoactive. Plantar reactions were flexor. Upon sensory assessment, both feet had reduced pinprick feeling, normal temperature and position sensitivity, and no sensory level. She had reduced rectal tone and was experiencing urine incontinence while at the emergency ward for assessment. Urinary retention led to the insertion of a catheter. About 20 hours following the beginning of symptoms, a spine MRI scan was conducted, and the results showed an aberrant T2 signal in the central spinal cord at the T12 and L1 vertebrae levels. The lesion was symmetric, bilateral, and mostly affected gray matter in the shape of "owl eyes." At the lesion level, there was evidence of diffusion limitation inside the spinal cord (Figure 1). The results were consistent with acute ischemia in the Adamkiewicz artery distribution. The absence of extra lesions on the brain and cervical spine MRI supports an infectious or inflammatory etiology. Aortic, brain, and neck artery vascular abnormalities were not found using CT angiography. A transthoracic echocardiography revealed an aberrant left ventricle regional wall motion and significant left ventricular failure with an ejection fraction of 38%. Cardiac CT did not reveal any signs of coronary disease or thrombus. Non-obstructive hypertrophic cardiomyopathy was shown by cardiac MRI. Initially, the patient was given antiplatelet medication. After identifying a cardiac condition and excluding

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atherosclerotic disease and vascular risk factors, a multidisciplinary team determined that a cardioembolic etiology was the most likely cause. Empirical anticoagulation treatment was initiated until further investigation. Therapy for heart failure was started, along with a full-dose low-molecular-weight heparin regimen. In order to look for cardiac arrhythmias, an implanted loop recorder was placed. The patient remained taking Apixaban after a month of follow-up, during which a brief, asymptomatic atrial flutter was seen. After intensive multidisciplinary physical therapy, the patient's functional condition improved and she regained independence.

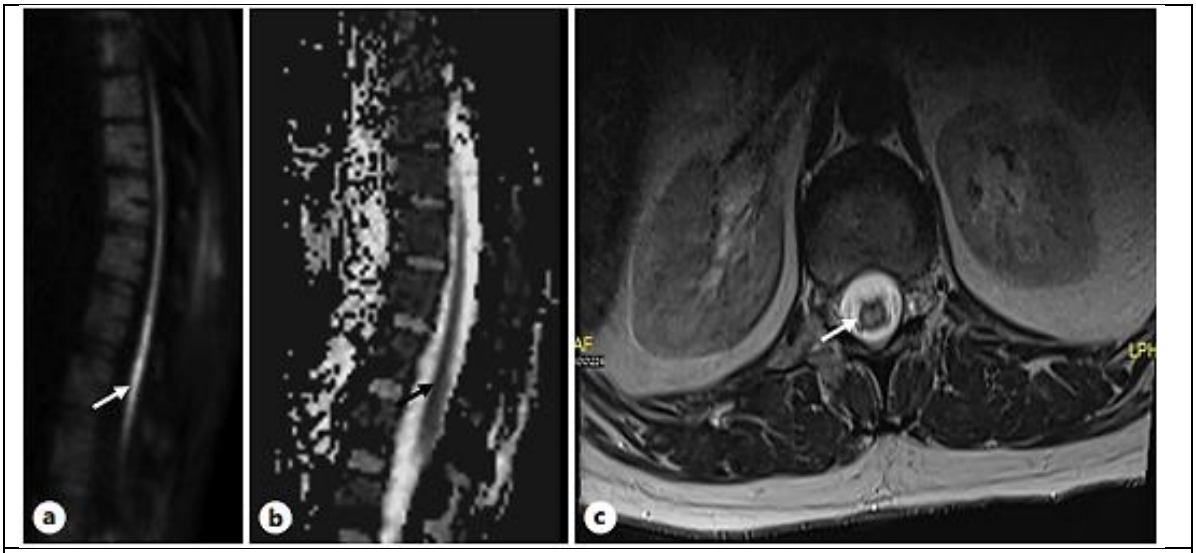

Fig 1. Spine MRI revealed an acute infarction at T12 and L1 vertebrae levels. The sagittal image showed a lesion with restricted diffusion, as evidenced by a high signal on DWI (a) and low ADC values (b). The axial image showed bilaterally symmetric pathologically increased T2WI signal (c), primarily involving gray matter.

Discussion:

SCI is a rare and frequently fatal neurovascular disease. The patient received anticoagulant treatment and underwent physical therapy to regain her independence.[2] In another study by Anant Naik et al., on non-iatrogenic spinal cord ischemia patients concluded that CSF drainage, blood pressure elevation, corticosteroids, antiplatelets/anticoagulants/thrombolytics, mannitol, naloxone, surgical revascularization, hyperbaric oxygen, and systemic hypothermia were some of the therapy options available in this patient population.[3]

Conclusion:

This case report described an unusual neurovascular condition and its rare etiology. Even in the lack of usual indications on neurological examination, it emphasized the necessity of a comprehensive and rapid investigation to rule out SCI in patients presenting with acute paraparesis. After receiving anticoagulation, the patient had remarkable improvement through physical rehabilitation.

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4. Case report on the entrapment of an embolic protection device inside a stent during carotid artery stenting

Introduction:

If complications arise during stenting, understanding the catheters, wires, and devices might assist the operator in resolving the issue using endovascular methods.

Carotid artery stenting (CAS) with an embolic protection device (EPD) is an effective treatment option for carotid artery stenosis, replacing carotid endarterectomy surgery. EPD minimized the risk of embolic complications during CAS. However, using an EPD might lead to issues such as filter blockage, internal carotid artery (ICA) dissection, difficulty retrieving or trapping of protective devices, and spasms.[1] The type and configuration of the aortic arch, vessel tortuosity, atheroma burden (in both the target and access vessels), and length of stenosis all have a significant impact on guide wire/catheter manipulation and contribute to the procedure's neurologic risks.[2] This case report described EPD entrapment within a stent during a CAS surgery, which was safely corrected with an endovascular maneuver.

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Case Presentation:

A 67-year-old male with ICA stenosis presented to a neurology outpatient clinic with right leg weakness. He had temporary right leg weakness and numbness lasting around 5 minutes for 2 weeks prior to the appointment. A neurological examination performed at the time of admission revealed no abnormalities. He had a history of hypertension and dyslipidemia. Diffusion-weighted magnetic resonance (MR) images revealed several border zone infarctions at the confluence of the left anterior cerebral artery and middle cerebral artery areas. A catheter angiography revealed 85% stenosis in the left proximal ICA. The medical professional chose to treat symptomatic stenosis with CAS. The procedure was done under local anesthesia. A 6-Fr guiding sheath was placed in the left common carotid artery (CCA) via a transfemoral technique. An Embold NAV6 filter (Abbott Vascular, IL, USA) was advanced over the stenosis and implanted at the ICA cervical portion, distant to the lesion. After deploying the EPD, a 5×40 mm Submarine Rapido balloon catheter (Medtronic Minneapolis, MN, USA) was inserted across the lesion. An Acculink 6-8×40 mm stent was implanted from the proximal left cervical ICA to the distal left CCA. After stent placement, the inexperienced interventionalist thought the surgery was successful. While pushing the retrieval catheter to remove the filter device, the operator was unaware that the filter wire had exited the catheter's side hole. As the retrieval catheter advanced, the filter was suddenly drawn back and caught inside the stent that had been inserted (Figure 1). The filter wire encountered resistance within the stent when gently tractioned. Attempts were taken to extract the filter basket using a retrieval catheter within the stent. However, the filter wire ring didn't fold. Several failed efforts were made to push the filter with the headhunter catheter. To remove the filter from the stent, a shuttle sheath was used. The filter was carefully pushed out of the stent using the shuttle sheath (Figure 2). The filter was removed easily using a retrieval catheter. A DWI collected 2 days after CAS revealed no new infarctions. A follow-up Doppler ultrasonography two days following the surgery revealed no signs of in-stent restenosis or thrombosis. The patient was discharged without any neurological deficits. The patient had positive clinical results during the 8-month follow-up.

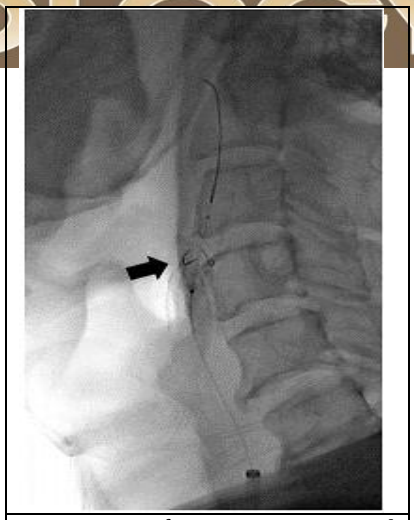

Fig 1. Left common carotid angiography revealing entrapment of the filter basket within the stent (arrow)

Fig. 2: An angiography taken after the shuttle sheath was pushed into the stent to push the filter reveals that the filter has migrated (arrow) out of the stent.

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Discussion:

The primary issue with CAS is the potential of distal embolic stroke due to plaque fragment migration. Carotid artery stenting (CAS) with an embolic protection device (EPD) is an effective treatment option for carotid artery stenosis.[1] According to a study by Nazari et al., patients who did not get EPD had a fourfold higher risk of perioperative stroke, according to data from the American College of Surgeons National Surgical Quality Improvement Program database.[3]

Conclusion:

The most crucial stage in stenting was to remain cautious until the treatment is complete. If complications arise during the procedure, a thorough understanding of the catheters, cables, and devices might aid in resolving the issue utilizing endovascular techniques.

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