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Interesting cases NEUROLOGY

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 the rare presentation of a Bilateral Brain Abscess**
2. **A Case Report of Delayed Nonarteritic Posterior Ischemic Optic Neuropathy after Herpes Zoster Ophthalmicus.**
3. **A case report of hemochorrhea brought on by a isolated temporal infarction and significant middle cerebral artery stenosis**
4. **A case report of Wernicke's encephalopathy characterised by urinary incontinence**

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. A case report on the rare presentation of a Bilateral Brain Abscess

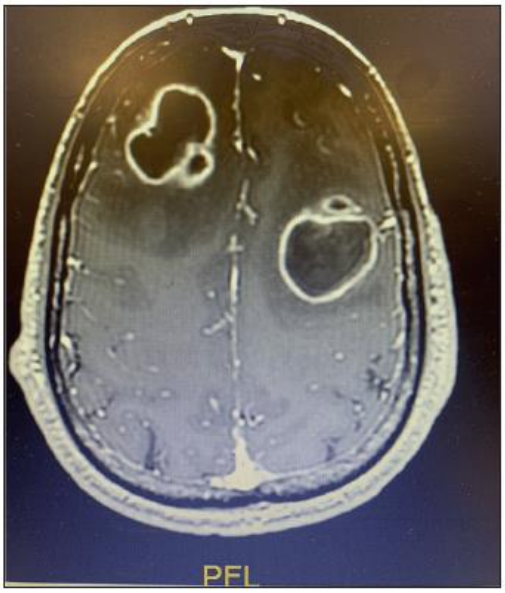
Introduction:

An brain abscess is a membrane-encircled necrosis in the brain parenchyma brought on by an infection or trauma. The organisms most frequently found in brain abscesses are *staphylococcus* and *streptococcus*.[1] Rare but dangerous infections involving the brain tissue, brain abscesses are brought on by bacteria or fungus. It is crucial to identify the issue and address it as soon as possible since they can result in major issues if not addressed right away.[2] Here is a case of a bilateral brain abscess in an adult with an unusual appearance and prognosis.

The significance of early detection and treatment is demonstrated in a 46-year-old man with bilateral brain abscesses.

Case Presentation:

A 46-year-old heavy smoker who had a 3-day cough, sore throat, fever, nausea, and disorientation appeared. The patient had a medical history that included immunisations, bronchoscopy, and pneumonia. A 14/15 Glasgow Coma Scale result and right pronator drift were found during the physical examination. CT and MRI revealed bilateral frontal intraaxial lesions with edoema, midline displacement, and uncal herniation. Amoxicillin-clavulanic acid-sensitive Escherichia coli and Staphylococcus aureus were found in cultures. A diagnosis of bilateral brain abscesses was made. Following sensitivity results, intravenous vancomycin, ceftriaxone, and metronidazole were administered for six weeks. Edoema and consequences were decreased with steroids. Cystic lesions were evacuated using frameless stereotactic aspiration. The abscesses were found by the neurosurgeon and navigation team, reducing harm. After aspirating the abscesses, saline and antibiotics were irrigated into the cavities. The outcome of the therapy and its implications were closely monitored in the critical care unit. Serial neuroimaging evaluated the clearance of the abscess. After discharge, the patient was monitored by the neurosurgeon and an expert in infectious diseases to assure healing and stop



Pre-operative transverse view of brain MRI showing bilateral abscess. Bilateral frontal intraxial space occupying lesions with intense marginal enhancement and perilesional edema. They are seen effacing adjacent lateral ventricle.

recurrence. A quick detection and treatment of the brain abscesses were essential due to the risk of herniation or death from probable consequences. Cultures revealed the etiological agents, directing antibiotic therapy. Inflammation was decreased by steroids. The abscesses were quickly treated using stereotactic aspiration, which accelerated recovery. The abscesses were found by the neurosurgeon and navigation team, reducing harm. After aspirating the abscesses, saline and antibiotics were irrigated into the cavities. The outcome of the therapy and its implications were closely monitored in the critical care unit. Serial neuroimaging evaluated the clearance of the abscess. After discharge, the patient was monitored by the neurosurgeon and an expert in infectious diseases to assure healing and stop recurrence. A quick detection and treatment of the brain abscesses were essential due to the risk of herniation or death from probable consequences. Cultures revealed the etiological agents, directing antibiotic therapy. Inflammation was decreased by steroids. The abscesses were quickly treated using stereotactic aspiration, which accelerated recovery.

Discussion:

An uncommon and fatal disease known as a bilateral brain abscess occurs when pus builds up in the brain tissue on both sides. It is brought on by bacterial, fungal, or parasitic bloodstream infections. It could be challenging to identify bilateral brain abscesses in a 46-year-old individual without risk factors. Brain surgery or trauma, a foreign body in the brain, and a weakened immune system are risk factors for brain abscesses.[2] Meningitis, hydrocephalus, and brain injury can all be brought on by brain abscesses. To avoid these consequences and succeed, early diagnosis and treatment are crucial.[3] When treating pneumonia patients who have fever, malaise, and altered mental state, healthcare practitioners should take brain abscesses into consideration.[2]

Conclusion:

The significance of early detection and treatment is demonstrated in a 46-year-old man with bilateral brain abscesses. Results from imaging and cultures aided in diagnosis and treatment. The removal of the cystic lesions and the patient's recovery demonstrate the effectiveness of the therapy.

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2. A Case Report of Delayed Nonarteritic Posterior Ischemic Optic Neuropathy after Herpes Zoster Ophthalmicus

Introduction:

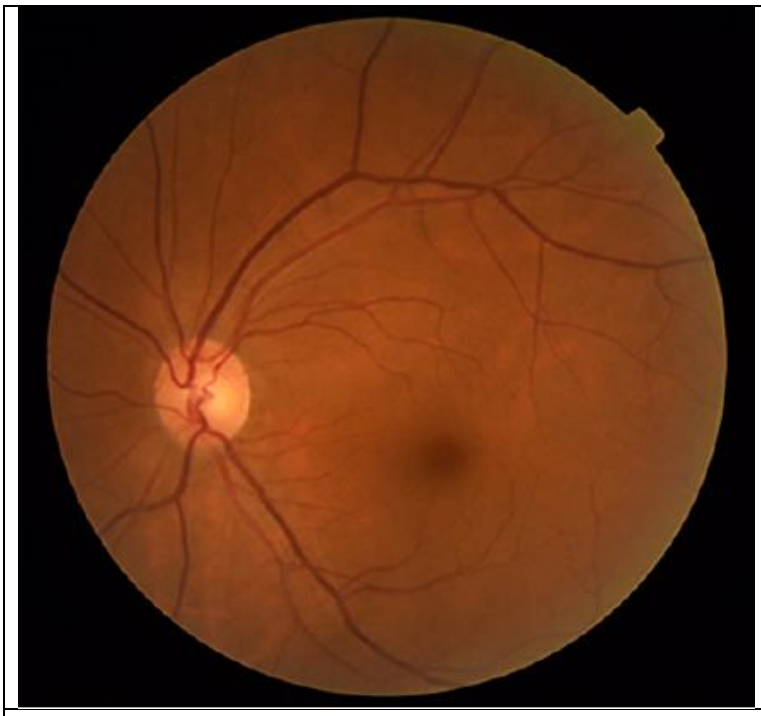
A rare but possibly fatal consequence of surgery is postoperative blindness, particularly posterior ischemic optic neuropathy (PION).There are three different aetiologies for PION, which is brought on by acute ischemia affecting the optic nerve's posterior portion. they can be classified into three etiologies: arteritic, nonarteritic, and surgical.[1] Studies on PION cases linked to Herpes zoster ophthalmicus (HZO) are few, and otic neuropathy is an uncommon herpes zoster consequence.[2] Here, researchers describe a rare instance of nonarteritic PION with a somewhat delayed presentation following HZO.

This case expanded the knowledge of herpes zoster optic neuropathy by demonstrating a delayed appearance of nonarteritic posterior ischemic optic neuropathy after Herpes zoster ophthalmicus.

Case Presentation:

A woman in her 64s showed up with a sudden, painless loss of vision in her left eye. She had no history of smoking, trauma, dyslipidemia, hypertension, diabetes mellitus, or any systemic vascular disorders. She was treated with topical acyclovir, levofloxacin, and prednisolone from an ophthalmologist as well as topical fusidic acid, betamethasone, and neomycin sulphate from a dermatologist six weeks prior to the onset of her visual loss for ipsilateral HZO, which is characterised by painful rashes on the left forehead, eyelid, and side of the nose. After receiving therapy for three weeks, her health largely improved. Her vision loss was first limited to the top part of her left visual field. The left eye's visual acuity was 0.03, while the right eye's was normal. Herpetic uveitis was identified as the cause, and acyclovir, neomycin sulphate, and triamcinolone acetonide were used as topical treatments. Later, visual perimetry revealed ambiguous superior visual abnormalities in the right eye and superior altitudinal and inferior peripheral defects in the left eye. Her left eye's vision continued to deteriorate until it was completely lost after one week. A visual acuity of 1.0 OD and OS were found during an eye test, and there were no indications of uveitis, cherry red spots, or anterior ischemic optic neuropathy (AION). With the exception of previous infarcts in the left basal ganglia, brain and orbit magnetic resonance imaging with contrast revealed no signs of an acute stroke, infiltrative lesions, or space-occupying lesions. She refused additional testing and took oral valaciclovir (500 mg three times a day) for three days following discharge, which was remarkable considering herpes zoster-associated ocular neuropathy. She was hospitalised with stationary vision two weeks after experiencing visual loss. Examination revealed a relative afferent pupillary deficit, a slow light reaction, and a visual acuity of hand motion of 30 cm in the left eye. It was 19 mm Hg inside the eyes. A light cataract, a clean anterior chamber, and no abnormalities on the

cornea were seen during a slit-lamp examination. During a fundoscopy, the retina, macula, and retinal vessels all appeared to be healthy, and the optic disc appeared normal. Except for a little cataract, the right eye was ordinary with a visual acuity of 1.0. There was an examination of optic neuropathy. There were 6,700 white blood cells, of which 68.1% were neutrophils, 20.3% were lymphocytes, 10.5% were monocytes, and 0.6% were eosinophils. The C-reactive protein level was 4.3 mg/L, and the erythrocyte sedimentation rate was 46 mm/h. Laboratory testing comprising thyroid-stimulating hormone, thyroxine, cobalamin, lipid profile, serum creatinine, and alanine aminotransferase were all within normal ranges. No pleocytosis or culture evidence of viral, TB, or syphilis infection was found, according to cerebral spinal fluid (CSF) investigations. Herpes simplex virus types 1 and 2 and varicella-zoster virus (VZV) DNA were not detected by CSF polymerase chain reaction. The carotid and ophthalmic arteries' sonograms were unremarkable. There was no indication of an obstruction in the ocular arteries, according to cerebral digital subtraction angiography. The diagnosis of nonarteritic PION raised the possibility that it was related to prior herpes zoster. Aspirin, topical brimonidine, and a further 7-day course of oral valaciclovir (1 g 3 times per day) were used to treat her. Following therapy, the affected eye's visual acuity marginally improved and, four and six weeks after the onset of vision loss, respectively, reached the counting finger of 20 cm and 0.02. Six weeks following the onset of blindness, visual perimetry indicated a complete defect in the left eye and, while being of inferior quality, a superior defect in the right eye, which was free of subjective visual issues or altered visual acuity. Optical coherence tomography revealed a loss of the retinal nerve fibre layer and a pallor in the left temporal eye (Fig. 3). Visual evoke potential data seven weeks after the visual loss showed that both sides of the P100 latency were extended, with the left side having a more severe delay (left, 146.7 ms;



Fundus examination. At 2 weeks after the visual loss, fundoscopy revealed a normal-appearing optic disc in the left eye, without disc edema, hemorrhage, retinal detachment, or maculopathy. The fundoscopy of the right eye was overall normal.

right, 119.1 ms; cutoff value, 110 ms). She reported stationary vision during the follow-up.

Discussion:

PION is a clinically and pathologically unique condition caused by poor perfusion of the posterior region of the optic nerve. The ocular division of the trigeminal nerve is involved in the disorder known as HZO, and optic neuropathy is an uncommon symptom of HZO.[3] There have been a few rare cases of PION linked to HZO prior to this case report. This study enhanced the understanding of herpes zoster optic neuropathy by demonstrating a delayed manifestation of non-arteritic PION following HZO compared to other research.[2]

Conclusion:

The presence of visual abnormalities many weeks after the zoster rash and the absence of any obvious vascular variables indicate that VZV may still be likely connected with the case's nonarteritic PION.This case revealed a comparatively delayed presentation of nonarteritic PION after HZO, extending the herpes zoster optic neuropathy spectrum.

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3.A case report of hemochorrhea brought on by a isolated temporal infarction and significant middle cerebral artery stenosis

Introduction:

Hemichorea is a condition in which one side of the body makes involuntary, continuous, irregular movements. It can also be brought on by metabolic abnormalities like hyperglycemia, while strokes are the most prevalent cause.[1] Hemichorea normally comes from a lesion of the contralateral subthalamic nuclei (STN), however it has also been noted in the cortex. There aren't any reports of hemichorea developing as a secondary condition to an isolated temporal stroke in the literature.[2] Here, is a case of an acute temporal stroke that caused hemichorea in the right extremities.

It is critical to recognise and take into account acute onset hemichorea as an initial sign of stroke to prevent misdiagnosis and delays in the delivery of effective therapy.

Case Presentation:

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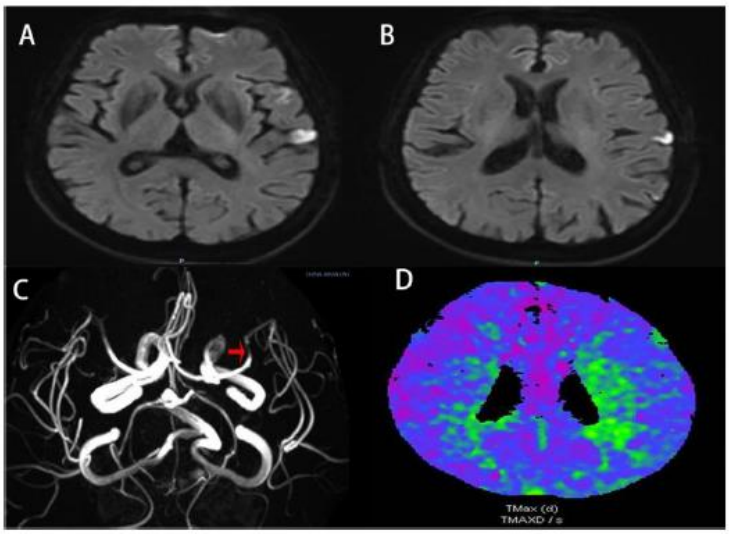
A 71-year-old woman with a history of hypertension was hospitalised to our hospital after experiencing right-extreme hyperkinesia for two days. Her right upper extremities began involuntary flailing movements, she unpredictably grabbed her right hand, and her right lower extremity began to kick suddenly. The continuous, asynchronous, varied, and worsening nature of these irrational motions in the right upper and lower extremities was exacerbated by deliberate task performance. The patient had minor dysarthria for the length of the illness, but it suddenly worsened to total aphasia with a drop in muscular strength of her right upper limb to level 3 for a period of 10 minutes, during which time her hemichorea persisted.After that, her speech and muscular strength returned to normal, and she had no further incidents. A brain computed tomography (CT) scan revealed ordinary results. Infectious, toxic, and metabolic causes of her encephalopathy were ruled out by laboratory tests and information from her medical history. Magnetic resonance angiography (MRA) of the head revealed significant stenosis in the left M1 branch, and brain magnetic resonance imaging (MRI) showed an acute ischemic lesion in the left temporal lobe. The left parietal cortex and corona radiata had delayed perfusion in the computed tomography perfusion (CTP) scan during the symptomatic period, according to the time-to-peak (TTP) measure. The patient received 100 mg of aspirin and 20 mg of atorvastatin, each administered individually, once daily, along with two injections of 100 ml of butylphthalide. She was given oral haloperidol at a dose of 2 mg twice day for three days, but no symptoms were relieved. The haloperidol dosage was increased to 4 mg in the morning, 2 mg in the afternoon, and 2 mg in the evening after seeing no improvement. She was discharged ten days after beginning the previously mentioned antithrombotic treatment and pharmaceutical management of movement disorders. Her hemichorea had significantly improved.



Brain images. DWI demonstrated high signal in the temporal region (A and B); MRA illustrated severe left M1 stenosis (arrow) (C); CTP during the symptomatic phase showed delayed perfusion in the left parietal cortex and corona radiata in the TTP measure (D). DWI = diffuse weighted image; MRA = magnetic resonance angiography; CTP = computed tomography perfusion; TTP = time-to-peak

Discussion:

Hemichorea, a very uncommon initial or only symptom of acute ischemic stroke, arises only from the cortex, particularly in the temporal lobe, which is not usually connected to motor function. In this case report, a patient with significant middle cerebral artery stenosis and hemichorea in the right extremities as the initial symptom of an isolated temporal stroke. No additional intracranial disease was identified to explain the symptoms.[2] Few cases of

hemichorea following lesions that involved the temporal lobe have been recorded, although none of them were solely related to a temporal lesion.[3,4]

Conclusion:

In the absence of any other abnormalities, this instance demonstrates that isolated temporal stroke brought on by significant MCA stenosis can be linked to hemichorea. Therefore, to avoid misdiagnosis and delaying the administration of necessary treatment, doctors should be aware that early onset hemichorea could be an initial sign of acute ischemic stroke. To better understand the underlying mechanisms of temporal stoke leading to hemichorea, more research is required.

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4.A case report of Wernicke's encephalopathy characterised by urinary incontinence

Introduction:

Acute neurological disease called Wernicke encephalopathy (WE) is characterised by the clinical characterises of confusion, ataxia, and ophthalmoparesis with nystagmus. Thiamine deficiency is the major contributing factor to this serious illness, which predominantly impacts the peripheral and central neurological systems.[1] Chronic drinking, gastrointestinal conditions, gastrointestinal surgery, and severe vomiting during pregnancy are some of the common causes of WE. WE may also result in other micturition issues, such as urine incontinence .[2] Here is a case of WE with urine incontinence as the primary symptom.

Surgeons should be mindful that urine incontinence in long-term fasting patients may be a sign of WE and that they should be given vitamin B1 immediately without extensive investigation.

Case Presentation:

A 62-year-old woman who had been experiencing intestinal blockage, nausea, and vomiting for five days was taken to the hospital. She was in good physical condition before to being sick, and she had never been diagnosed with alcoholism, liver disease, digestive problems, or undergone surgery. A mass in the right colon was discovered by

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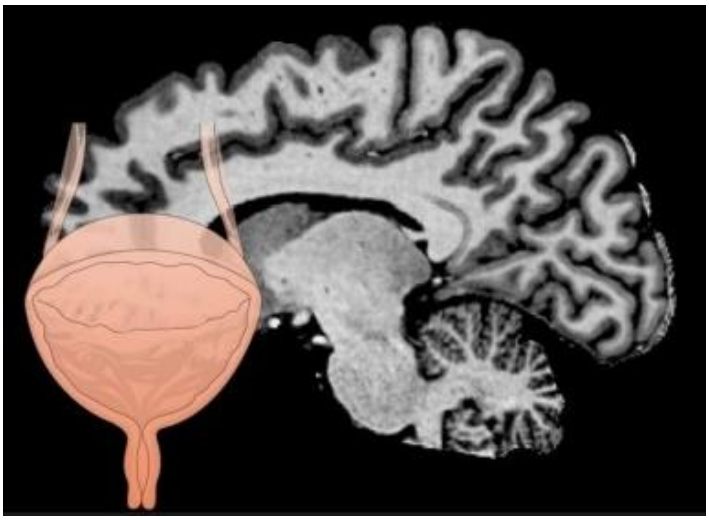
an abdominal CT scan; it was removed, and a proximal colostomy was done. There were no issues with the procedure. Continual IV nourishment was given to the patient, however vitamin B1 was not given. Following the procedure, the colour Doppler ultrasonography test revealed that the bladder was empty. She experienced total urinary incontinence three days following the general anaesthesia procedure, with no apparent cause. Her Mini-Mental State Examination (MMSE) score was 27, with problems with concentration, daydreaming, making mistakes in simple calculation, and being unable remember events that had happened ten minutes earlier. She had no confusion, could respond to questions normally, but displayed mild indifference in her mental state as evidenced by her tardiness in responding when called by name and her lack of interest in changes in the people or environment around her. Upon physical examination, the patient's temperature was measured at 37.6°C, along with their heart rate, respiration rate, and blood pressure, which were all 135/82 mmHg. She cooperated during the examination, had pupils that were 2.5 mm in diameter, were equally responsive to light, no evident ataxia, no nystagmus, and no ophthalmoplegia. Additionally, the cardiac assessment revealed no abnormalities. The results of the lab tests were normal, with the exception of slightly reduced albumin levels. Urologists were consulted when the general surgeon was unable to identify the patient's urine incontinence's underlying cause. The urologist thought about whether the incontinence may have been brought on by damage to the sacral or lumbar nerves after the appointment. The general surgeon, however, ruled out any likelihood of nerve injury. The patient had no prior history of neurological illness, thus the neurologist did not examine the likelihood of cerebral haemorrhage, cerebral thrombosis, or other central neuropathies. As a result, vitamin B1 diagnostic supplementation was advised, and the patient received 200 mg/day of vitamin B1 intramuscularly immediately. Urinary incontinence and mental apathy in the patient improved after 3 days of vitamin B1 supplementation. Within 7 days of starting medication, the patient's urine incontinence was entirely cured.



Discussion:

It is well knowledge that the central nervous system controls proper urine function. The midbrain's periaqueductal gray (PAG) receives signals from lower urinary tract afferents that synapse in the spinal cord's dorsal horn. The pontine micturition centre (PMC), located in the brainstem, is a coordinating centre for voiding and an area of convergence for multiple implications. The periaqueductal gray, which also integrates bladder

sensory input, modulates voiding and urine storage, and regulates the transition between the two phases, serves as the primary pathway through which these centres' effects on the PMC are transmitted.[3] Glucose is primarily used as energy in nerve and brain cells. A lack of vitamin B1 can result in problems with glucose metabolism and inadequate energy generation, as well as a buildup of lactic and pyruvic acids in the nerve cells of the PAG, which can induce incontinence or voiding dysfunction.[2]

Conclusion:

Researchers recommend that the likelihood of WE be taken into consideration when patients who have been on prolonged fasts and without vitamin B1 supplements have symptoms including mental apathy, urine incontinence, and faecal incontinence. Patients don't need to undergo a brain MRI, a cerebrospinal fluid analysis, or any other tests in order to receive diagnostic vitamin B1 treatment.

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