Vol 3 | Issue 14 | 2022

Interesting cases NEUROLOGY

Neurology Case Series

Vol 3 | Issue 14 | 2022

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 bilateral mydriasis as the first sign of Hodgkin's lymphoma.**
2. **A case of Myeloencephalitis caused by an Omicron SARS-CoV-2 infection.**
3. **Pituitary apoplexy-related unusual bifrontal cerebral infarction: A rare case report.**
4. **A case of Vestibular schwannoma presenting typical clinical symptoms mimicking Vestibular Neuritis.**

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,

MICRO LABS SYNAPSE
Vol 3 | Issue 14 | 2022
Interesting cases NEUROLOGY

1. A case report on bilateral mydriasis as the first sign of Hodgkin's lymphoma

Introduction:

Hodgkin lymphoma (HL) is an uncommon monoclonal lymphoid tumour that is characterized by four characteristics: HL typically manifests in young adults, is characterised by dispersed large mononuclear Hodgkin and multinucleated Reed-Sternberg cells on a background of non-neoplastic inflammatory cells, and distinctive neoplastic cells are frequently surrounded by T lymphocytes.[1] The majority of the time, severe brain stem injury or drug-induced sympathomimetic activation is the cause of bilateral mydriasis. extremely few incidences of mydriasis caused by cervical sympathetic nerve chain impairment have ever been documented as a neurologic complication of Hodgkin's lymphoma.[2] In this case, a young woman with bilateral mydriasis, the initial clinical sign of Hodgkin's lymphoma, and enlarged pericarotid lymph nodes that were the cause of an oculosympathetic spasm are presented.

Case presentation:

When other common reasons, such as brain stem impairment, are ruled out, pathologies surrounding the carotid artery that produce oculo-sympathetic spasm should be taken into consideration.

A 23-year-old woman who had previously been in good health had bilateral mydriasis (left pupil diameter: 6 mm, right pupil diameter: 8 mm, both pupils unresponsive to direct and indirect light stimuli). Six weeks before to hospitalization, the right side of the body experienced mydriasis, which has continued ever since. On the day of hospitalisation, the patient observed the mydriasis on the left side. Ocular motility testing and other neurologic testing revealed no abnormality. Brain MRI scans with gadolinium contrast performed 4 weeks before admission and on the day of admission revealed nothing abnormal. The mediastinum and supraclavicular lymph nodes on both sides were enlarged in the 15 days before to admission MRI scans of the cervical spine and chest. An analysis of a lymph node biopsy samples by a histopathologist revealed classical Hodgkin's lymphoma. Additional problematic lymph nodes were discovered during a staging 18 F-FDG PET/CT in the left axillary, right pelvic region, and retroperitoneal. The carotid artery was observed to be severely compressed on both sides by mediastinal and cervical lymph nodes. An study of the patient's cerebral spinal fluid (CSF) revealed a lymphocytic pleocytosis (10 cells/l) with normal protein and glucose levels. Low genome concentration (500 genome copies/ml) polymerase chain reaction (PCR) results for HSV-1 and HSV-2 were positive. Leukocytes from CSF underwent flow cytometric analysis, which revealed normal cell distribution and no evidence of meningeosis lymphomatosa. As a result, the patient received an 18-day course of acyclovir and an intensified BEACOPP chemotherapy regimen (bleomycin, etoposide, doxorubicin, cyclophosphamide, vincristine, procarbazine, prednisone). A second 18F-FDG PET/CT was performed for follow-up assessment and restaging after about four months and four cycles of increased BEACOPP. The results revealed a complete remission. The bilateral mydriasis showed a slight improvement.

MICRO LABS SYNAPSE
Interesting cases NEUROLOGY
Vol 3 | Issue 14 | 2022

Discussion:

Researchers discuss the case of a patient who initially had Hodgkin's lymphoma with bilateral mydriasis. According to their pathophysiological hypothesis, larger lymph nodes irritate the pericarotid sympathetic nerves, which in turn causes oculosympathetic spasm and eventually leads to autonomic dysfunction, which is clinically seen as pupillary dilatation.Bilateral mydriasis is the main clinical symptom of Hodgkin's lymphoma in the case that is being discussed here. This is the first instance of mydriasis being documented in conjunction with Hodgkin's lymphoma according to researchers.[2] In other study, a Hodgkin's lymphoma patient with enlarged mediastinal lymph nodes was described as having Horner syndrome.[3]

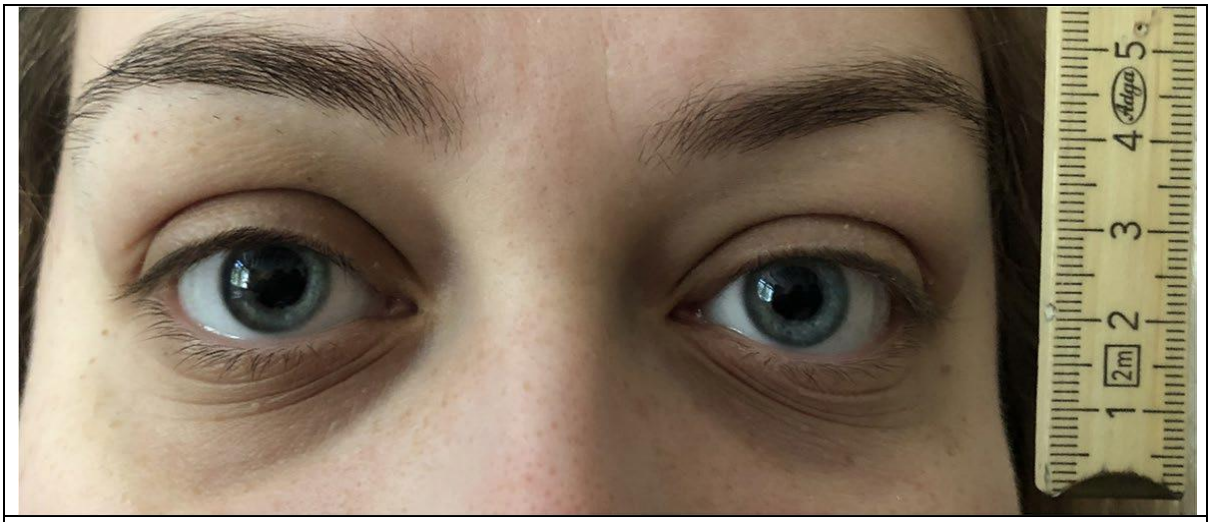

Image showing Dilated pupils on admission

Conclusion:

In conclusion, this case highlights the significance of thorough diagnostics in situations of autonomic dysfunction like mydriasis, particularly imaging of the tissues surrounding the sympathetic pathway.

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MICRO LABS SYNAPSE
Vol 3 | Issue 14 | 2022
Interesting cases NEUROLOGY

2. A case of Myeloencephalitis caused by an Omicron SARS-CoV-2 infection

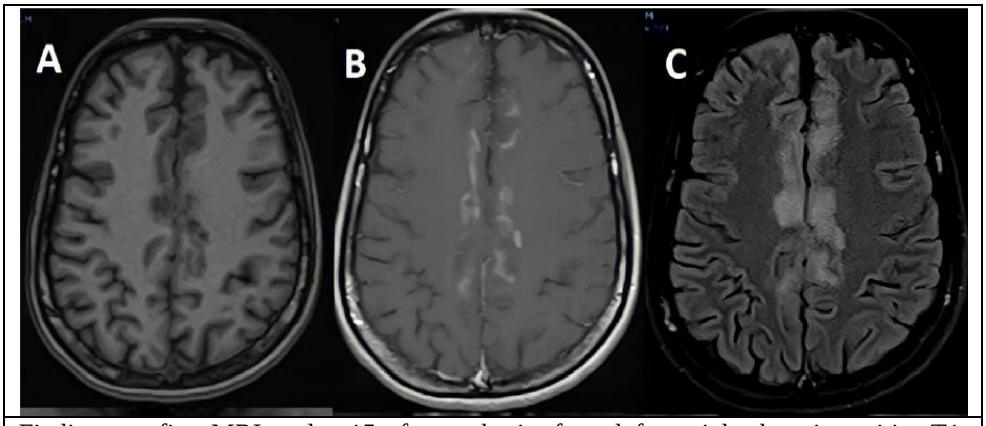
Introduction:

The primary symptom of SARS-CoV-2, which is a serious worldwide health concern, is a respiratory condition. Hypercoagulability, the cardiovascular system, and the central nervous system are all involved in further problems. About 0.04% to 0.2% of all SARS-CoV-2 patients are expected to have central nervous system (CNS) disease. A case of isolated myeloencephalitis with positive real-time PCR (RT-PCR) for SARS-CoV-2 in CSF in a young patient with no prior history is presented here by researchers. The temporal link between clinical traits, CSF parameters, and neuroimagingis also described.

Omicron SARS-CoV-2 infection without respiratory prodrome may manifest as acute myeloencephalitis..

Case Presentation:

A woman in her late 20s who had previously been in good health came complaining of paresthesia in her 2 feet 2 weeks earlier. Within 10 days, the paresthesia had extended to her lower abdomen and upper limbs and was linked with tetraparesis. Three doses of Pfizer Cominarty were listed in her past immunisation history, the last of which was administered one month prior to the onset of symptoms. No previous SARS-CoV-2 infections were found. As per the hospital's protocol, a rapid COVID-19 entry screening test was positive at the time of admission. There were no respiratory symptoms like a sore throat, fever, or coughing. On room air, the patient was totally eupneic, and pulmonary auscultation revealed no irregularities. During the neurological assessment, the patient was alert, oriented, and afebrile. Moderate lower limb weakness (3/5) and mild upper limb weakness (4/5) were present, with a modest asymetry predominating on the right side. Ankle clonus and bilateral hyperreflexia with Babinski symptoms were noted. In the lower left limb's vibration sensation and joint position sense, however they were lost in the lower right limb. Additionally seen was a slight nuchal rigidity. Both significant cognitive dysfunctions and cranial nerve impairment were absent. A clinically suspicious pyramidal and sensory syndrome with rapid advancement served as the basis for the initial diagnostic diagnosis, which included acute myeloencephalitis. At day 15 of symptoms, her first gadolinium-enhanced MRI of the brain and spinal cord was



Findings on first MRI at day 15 of paresthesia: from left to right, hypointensities T1, gadolinium-enhanced T1 lesions and hypertensities T2/fluid attenuated inversion recovery in bilateral cingulate and medial frontal cortex (A–B–C)

performed. Following that, a lumbar puncture was done to find a mild pleocytosis at 74 cells/L and a slightly high proteinorrachia. Myeloencephalitis acute was determined to be the cause. A CT of 20.52 was used to assess the presence of SARS-CoV-2 in CSF, and the results were positive. Numerous known causes of acute or subacute encephalitis were thoroughly investigated, but none were found. A concomitant neuropathy was ruled out after additional testing using nerve conduction studies and electromyography revealed no involvement of the peripheral nervous system. An electroencephalogram that lasted 30 minutes was unremarkable. Later, a thoracoabdominopelvic CT scan revealed a minor pleural effusion on both sides without any pulmonary parenchymal opacities. Eventually, a B.1.1.529 variant (Omicron) in a nasal sample was discovered using genome sequencing. As soon as the diagnosis of acute myeloencephalitis was made and bacterial and tubercular causes on CSF analysis were ruled out, a course of high-dose methylprednisolone (1000 mg daily for 5 days) was started. The nasal swab RT-PCR was positive, SARS-CoV-2 as a potential cause was suspected, and intravenous antiviral treatment with remdesivir (loading dose of 200 mg, followed by 100 mg daily) was added. The result of RT-PCR in CSF came back positive 2 days after start of antiviral therapy. She started experiencing symptoms of urine incontinence on day 18 and needed to have her bladder catheterized. At the end of the 5-day pulse corticotherapy, a second lumbar puncture revealed a recession of pleocytosis down to 16 cells/L and a persistently increased proteinorrachia. Her following nasal swabs and second SARS-CoV- 2 RT-PCR in CSF both came up negative. Seven days after the initial brain MRI, a second one was taken, and it showed less contrast enhancement. The C-reactive protein and interleukin-6 systemic inflammatory indicators linked to SARS-CoV-2 infection, as well as other parameters (liver and renal function, ionogram, coagulation), were all within normal ranges. At day 20 after beginning, her clinical condition began to improve as her paresthesia and limb motor function both gradually improved. She was later discharged at day 25 of symptoms with very little distal paresthesia, a complete resolution of her urinary incontinence, and a minor tetraparesis at 4+/5. Remdesivir was withdrawn after 9 days. There was almost full neurological recovery with very little to no pronator drift in the upper limbs and no paresthesia at the 2-week follow-up appointment.

Discussion:

With a positive RT-PCR result in CSF, this is the first case of encephalitis associated with the Omicron strain. This new variant's appearance is linked to an increase in infection rates, however it is unknown whether it may cause different neurological involvements.[1] Kato et al. described another case presenting with conscious disturbance and myoclonus, though her CSF RT-PCR result was negative.[3] Older age, critical illness with delirium, seizures, altered consciousness, and mechanical ventilation are variables that are frequently linked to SARS-CoV- 2-related encephalopathy in the pre-Omicron era; none of these factors occurred in present patient.[1] Given the extraordinary rarity and novelty of the condition, it is now unable to describe a precise clinical picture of the patient's symptoms, which are consequently uncommon.

Conclusion:

The Omicron era's first case report is this one. Clinicians should examine this virus among other potential neurotropic agents and be aware with its radiological and clinical manifestations in light of the recent global explosion of SARS-Cov-2 infections.

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1. Dang TQ, La DT, Tran TN. Myeloencephalitis as the only presentation of Omicron SARS-CoV- 2 infection BMJ Case Rep 2022;**15**:e251922.
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3. Pituitary apoplexy-related unusual bifrontal cerebral infarction: A rare case report

Introduction:

Pituitary apoplexy(PA) is a condition in which the pituitary gland hemorrhage or infarction. Pituitary apoplexy is the medical term for the pituitary gland's "sudden death," which is typically brought on by an acute ischemic infarction or haemorrhage.[1] Severe headache, blurred vision, nausea, ophthalmoplegia, hypopituitarism, and altered mental status, including coma, are the major symptoms. Vascular compression and vasospasm are two mechanisms that are mentioned in relation to the association between stroke and PA.[2] Bifrontal infarction linked to PA was recorded in only a few rare cases. Here, researchers present a new case with this presentation.

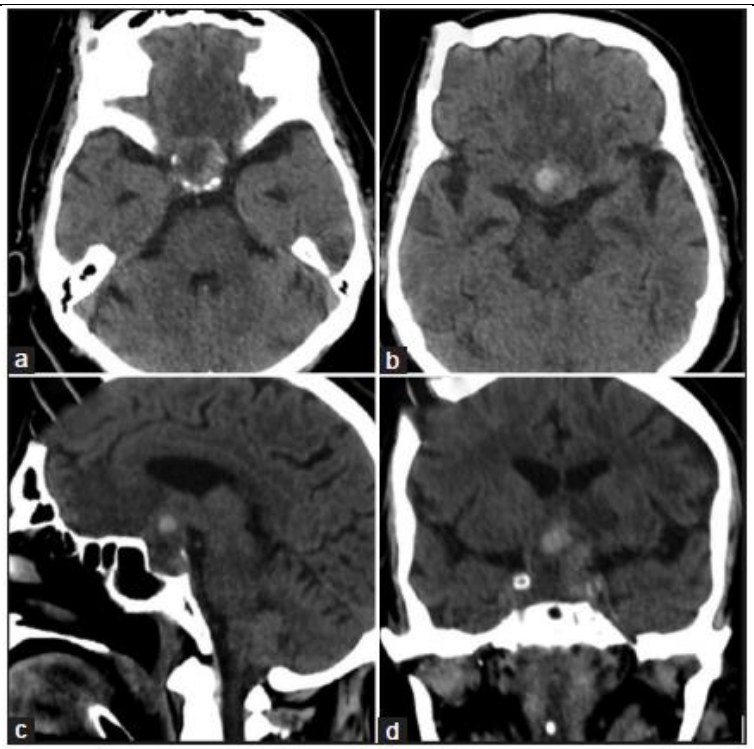
Pituitary apoplexy that results in cerebral infarction is a rare condition with high rates of morbidity and mortality. These cases must be addressed as neuroendocrinological emergencies, and surgical intervention is essential for improving the patients' prognosis.

Case Presentation:

A 65-year-old man with a history of hypertension was admitted into the hospital's emergency room with complaints of a terrible headache, nausea, and sleepiness that had started 5 days earlier. A comma and a progressive visual impairment also occurred along with these symptoms. The patient had divergent squint and anisocoria (with left mydriasis) upon clinical evaluation. Dopamine was required because of the ongoing bradycardia and hypotension. During an emergency CT scan, a sellar and suprasellar hemorrhagic expansive lesion along with a bifrontal hypodensity area were discovered. An expansive sellar and suprasellar lesion with a heterogeneous signal was visible on the MRI of the sella turcica. The lesion was 3.1x 3.0 x1.5 cm, and bilateral compression of the anterior cerebral arteries and optic chiasm was seen. In addition to the lesion, there was DWI restriction in the bifrontal region. These results supported bifrontal ischemia and PA. Levothyroxine and prednisone were administered to begin hormone replacement treatment after the endocrinological examination revealed hypopituitarism. The patient underwent an endoscopic transnasal transsphenoidal approach due

MICRO LABS SYNAPSE
Interesting cases NEUROLOGY
Vol 3 | Issue 14 | 2022

to the emergency. A red-colored lesion with an internal hemorrhagic region was seen during the procedure. Decompression of the optic chiasm with arachnoid collapse of the suprasellar cistern was seen following tumour removal. A pituitary tumour was established by the histological analysis. Surgery enhanced cardiac output, avoiding the requirement for preoperative vasoactive medication. With isochoric pupils and a slow but constant level of attentiveness, the patient made a regular recovery. Apathy was identified and linked to the ischemia area.



(a and b) Axial images which show a sellar and suprasellar hemorrhagic lesion associated with a basal bifrontal hypodense area. (c) Sagittal view. (d) Coronal view.

Discussion:

PA is a rare disease. Large adenomas, hypertension, high intracranial pressure, head trauma, cavernous sinus invasion, dopamine agonist use, anticoagulant medication use, radiation, and hormonal stimulation are the key risk factors. Despite better diagnosis and treatment options, morbidity and mortality rates remain high, at 15.3%.[3,4] Emergency neurosurgery surgery is required for PA. Early surgery reduces morbidity and mortality. [2]

Conclusion:

PA that results in cerebral infarction is an uncommon disease with high rates of morbidity and mortality. Direct arterial compression and arterial vasospasm are the two main processes involved. The symptoms of PA and stroke do not match up in the syndrome presentation. In order to improve the patients' prognosis, the situations must be treated as neuroendocrinological crises and surgical management is essential. Because hypopituitarism-related problems need to be adequately controlled to lessen their impact on prognosis, the postoperative phase is equally crucial.

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4. A case of Vestibular schwannoma presenting typical clinical symptoms mimicking Vestibular Neuritis.

Introduction:

Schwannomas are benign, well-encapsulated nerve sheath tumours with a slow growth rate that are primarily made up of Schwann cells. Any myelinated central or peripheral nerve with Schwann cells can give rise to the tumour.[1] A frequent benign intracranial tumour that constitutes around 6% of all intracranial neoplasms is the vestibular schwannoma (VS). Hearing loss, tinnitus, and vertigo are frequently present with vestibular schwannoma (VS), which is typically chronic and progresses over time. [2] Here, researchers present a case of VS in a 57-year-old man who exhibited typical clinical symptoms of vestibular neuritis (VN).

Although the dizziness associated with vestibular schwannoma is chronic and progressive, it can also manifest as an acute vestibular syndrome that resembles vestibular neuritis.

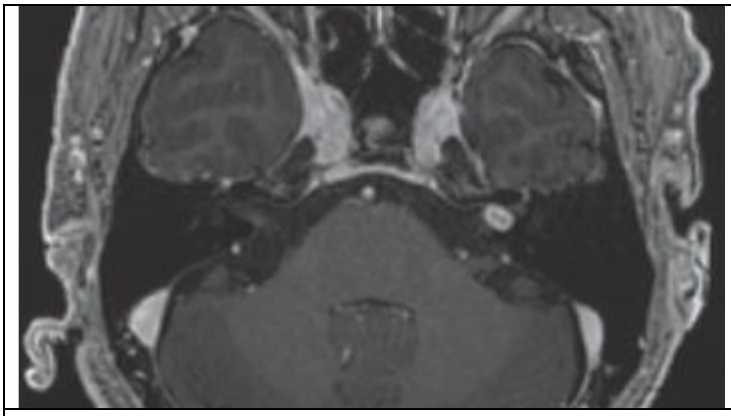
Case Presentation:

A 57-year-old man visited the emergency room after experiencing severe, continuous vertigo for three hours. He didn't express any headache, tinnitus, or hearing loss complaints. Right-beating horizontal-torsional spontaneous nystagmus was seen on video-Frenzel goggle examination, and it followed Alexander's law. Positioning techniques used in the Dix-Hallpike and head-roll tests had no effect on the nystagmus's direction. A left catch-up saccade was seen during the bedside head impulse test. There were no anomalies found during the neurological examination. Tuning fork testing of hearing were normal. After confirming a VN clinical diagnosis, the patient was admitted to the hospital. Pure-tone audiometry revealed a bilateral speech discrimination score of 92% and an average threshold of 22.5 dB. The threshold of the click-evoked auditory brainstem response was 45 dB on both sides, with no noticeable delay in latencies on the left. 72% left canal paresis was seen in the bithermal caloric test, and diminished gains in the left horizontal and anterior semicircular canals were seen in the video head impulse test. A 0.7-cm enhancing mass in the left internal auditory canal was seen using contrast-enhanced brain magnetic resonance imaging, which is indicative of VS. To treat the patient's acute symptoms, high dosage systemic corticosteroids were given to them (prednisolone 1 mg/kg/day for 4 days, then tapered off for the following 10 days). He was also given vestibular suppressants with antiemetics. Exercises for vestibular rehabilitation were suggested when the patient's condition was tolerable. The patient was referred to a neurosurgeon for follow-up after being discharged.

Discussion:

MICRO LABS SYNAPSE
Vol 3 | Issue 14 | 2022
Interesting cases NEUROLOGY

Tinnitus, hearing loss, and vertigo are some of the typical VS symptoms that often evolve over time. Even while sudden hearing loss has been a common VS presenting symptom, VN has been reported to occur far less frequently.[2] According to Sunara et al., a patient with VS who had previously had acute vertigo brought on by VN presented with additional symptoms, including deteriorating hearing loss and tinnitus. The authors hypothesised that overlapping VN in the vestibular nerve already damaged by VS could account for the patient's acute vestibular illness.[3] Researchers documented current case of VS in which the initial VN presentation without hearing loss or tinnitus.



Brain magnetic resonance imaging (MRI). T1-weighted contrast-enhanced MRI reveals a 0.7-cm enhancing mass in the left internal auditory canal (arrow).

Conclusion:

In summary, although VS dizziness is chronic and progressive in character, it can also manifest as an acute vestibular syndrome that resembles VN. A complete neurological evaluation with magnetic resonance imaging may be beneficial for a proper diagnosis, and VS should be taken into consideration as a probable cause of acute vestibular syndrome.

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