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. **Case report on traumatic optic neuropathy caused by an air gun pellet injury**
2. **An uncommon case of Klüver-Bucy syndrome after subarachnoid hemorrhage**
3. **Case Report on Migraine Symptoms Caused by an Auricular Piercing**
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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,

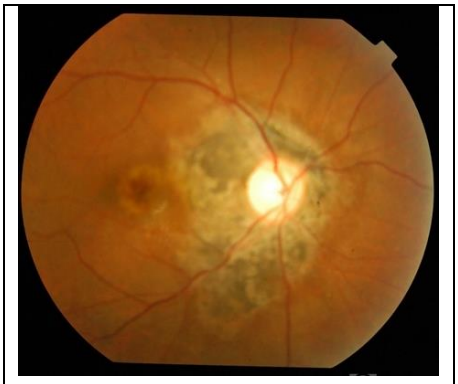
Medical Team,

1. Case report on traumatic optic neuropathy caused by an air gun pellet injury

In this case of direct TON, the patient received a high dose of steroids promptly and he reports substantial improvement in vision.

Introduction:

Traumatic optic neuropathy (TON) is a serious consequence of closed head traumas, affecting 0.5% to 5% of patients. One of the main pathophysiologic manifestations of damage is increased intracanalicular pressure.[1] TON can be classed as direct or indirect, based on the harm mechanism. Direct optic nerve injury causes acute vision loss due to external trauma, while indirect optic nerve injury causes ischemia due to disruption of the ophthalmic blood supply. Diagnosing TON is challenging and involves a thorough clinical evaluation. The degree of the injury determines the appropriate therapeutic approach. Immediate medical assistance, including surgical intervention, is necessary to prevent permanent eyesight loss and optic nerve damage.[2] This case report described an 18-year-old male who had history of an air gun being accidentally discharged into his left eye.



Traumatic optic neuropathy

Case Presentation:

A 18-year-old male was presented to the emergency room complaining of a left eye injury from an air gun that had accidentally discharged, puncturing his cornea and producing a penetrating pellet damage that had caused vision loss in his left eye. Presenting to the emergency department (ED) within three hours of the injury, he was found to be asymptomatic and vitally stable. An examination of the upper and lower eyelids revealed swelling, bruising, conjunctival chemosis, and subconjunctival hematoma (Fig. 1). The left eye's visual acuity was limited to light

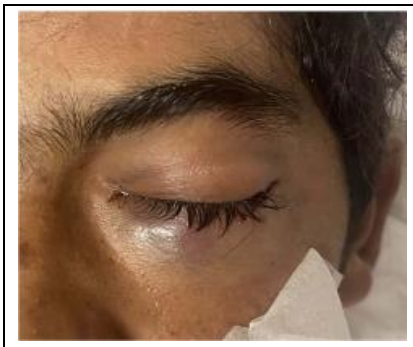


Figure 1: External examination of the left eye reveals edema and bruising on both upper and lower eyelids.

perception due to a positive relative afferent pupillary deficiency. Extraocular motions remained preserved. A fundoscopic examination indicated a normal retina with no exudates or hemorrhage. Fluorescein staining of the eye did not detect corneal or conjunctival damage or foreign substances. An unenhanced CT scan of the brain and orbit revealed shrapnel in the left maxillary sinus, the inferior part of the left orbit, and a large shrapnel in the left pterygopalatine fossa near the inferolateral recess of the sphenoid sinus (Fig. 2). The left globe and optic nerve were intact, and there was no evidence of cerebral bleeding (Fig. 3). The patient was diagnosed with direct TON and received high dose steroids intravenously. The ophthalmology team recommended Optical Coherence Tomography to diagnose a traumatic stage 1 macular hole (TMH) in the left eye. The patient was treated conservatively. The weight-based oral prednisolone treatment was continued. After a two-week follow-up, the patient reported considerable eyesight improvement.

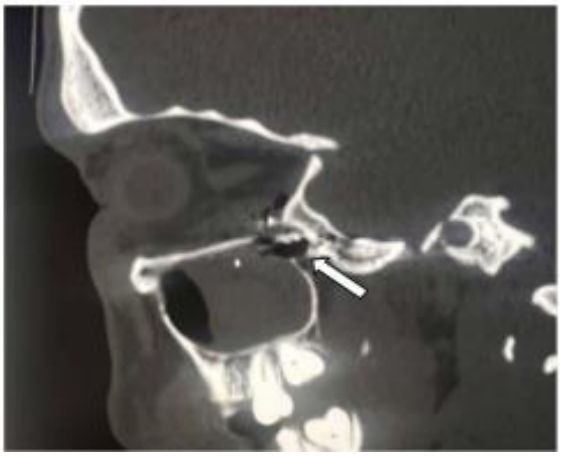


Figure 2: Sagittal section of CT-Head demonstrating several shrapnel embedded in the left pterygopalatine fossa, leading to an ocular apex fracture (white arrow).

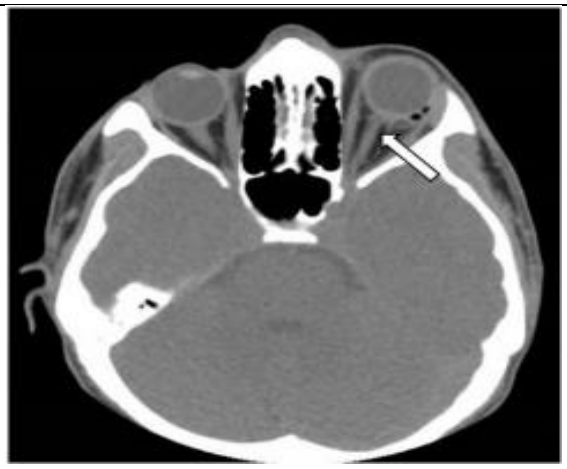


Figure 3: An axial segment of the CT-Head reveals an undamaged left optic nerve (white arrow).

Discussion:

TON, a severe complication from closed head traumas, affects 0.7%-2.5% of the population. Direct TON often causes full visual loss and has a worse prognosis than indirect TON. The direct light reflex of the eye was thought to be the most accurate during a thorough eye examination, particularly in cases where patients were unconscious.[2] This reflex to the constriction of the pupil of the affected eye in reaction to intense light, and an aberrant response may indicate damage or dysfunction of the neural pathways resulting from structural conditions such as stroke or traumatic brain injury.[3]

Conclusion:

In this case of direct TON, the patient received a high dosage of steroids immediately and patient reports significant improvement in his vision. Early detection and treatment of TON improves chances of a positive outcome.

Reference:

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2. Anis I, Baig MA. Traumatic optic neuropathy from an air gun pellet injury: a case report. Oxf Med Case Reports. 2024 Jan 27;2024(1):omad130.
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2. An uncommon case of Klüver-Bucy syndrome after subarachnoid hemorrhage

Introduction:

Klüver-Bucy syndrome (KBS) is a rare neuropsychiatric condition characterized by abnormal medial temporal lobe function (amygdala, temporal and hippocampus networks). This syndrome can manifest in several ways, including hyperorality, hyperphagia, indiscriminate dietary behavior, hypersexuality, apathy, visual agnosia, distractibility, emotional and memory impairment, and excessive visual attentiveness (known as hypermetamorphosis).[1] Humans rarely experience the entire syndrome due to less anterior temporal lobe impairment than monkeys after total temporal lobe excision. Hypersexuality was the most common symptom, followed by hyperorality.[2] This case report describes a 49-year-old female patient who acquired KBS due to subarachnoid hemorrhage.

KBS is an uncommon neuropsychiatric condition. Collaboration among the treating neurologist, psychiatrist, neurosurgeon, and radiologist is vital for determining the definitive diagnosis of KBS.

Case Presentation:

A 49-year-old female housemaid with hypertension and no history of mental illness functions well. The patient was referred to the ER when her sponsor spotted her chatting to her dead husband, obtaining her passport, and requesting to return to her home country. In addition to auditory hallucinations, the sponsor said that she attempted to seduce him, indicating hypersexuality. During the interview, she became lost to her surroundings. She also had delusions, believing her spouse who died was still alive

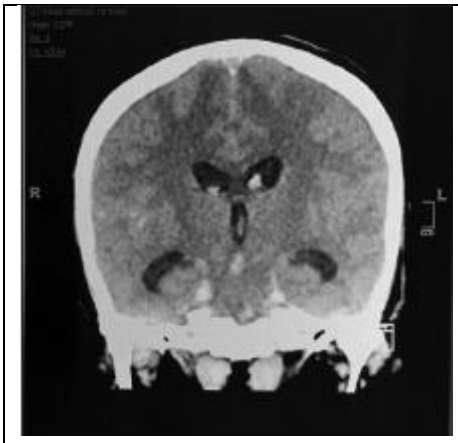

Figure 1: Calcification of the hippocampus.

and in Canada. The patient was admitted to psychiatric hospital for monitoring. A computed tomography (CT) scan revealed calcification in the hippocampus (Figure 1). She was diagnosed with a brief psychotic illness and began on 5 mg olanzapine before being discharged on April 19, 2020. The patient reported taking olanzapine for 2 weeks after. On December 26, 2020, the patient reappeared to the hospital with headaches for three days and one seizure incident. Upon evaluation, the Glasgow Coma Scale ranged from 13 to 14/15. The patient was tired and required assistance to walk. The investigation report revealed subarachnoid hemorrhage, ventricular hypertrophy, and a left posterior connecting artery aneurysm. The CSF examination revealed a pinkish/red color, 1777 red blood cells (no white blood cells), 22.8 protein, 137 glucose, and a negative Gram stain. The procedure involved inserting an external ventricular drain, coiling the aneurysm, and administering nimodipine 60 mg. Repeated CT scans revealed modest size growth in the lateral and third ventricles. The patient got a permanent shunt for hydrocephalus caused by subarachnoid hemorrhage (Figures 2 and 3). On December 31, 2020, a psychiatric consultation was conducted. The patient reported sleeplessness and persistent auditory hallucinations. She denied having depression, anxiety, or delusional symptoms. The experience was intermittent, causing confusion about time, place, and person. The provisional diagnosis was delirium, and she was administered 1 mg risperidone at bedtime. During her hospital stay, she demonstrated hyperorality by insisting on feeding while being NPO (nothing by mouth). Nurses observed that the patient exhibited childish behavior and appeared "as innocent and kind as a child," indicating a sense of calm. The sponsor observed an increase in kindness and a desire to help others, which may indicate hyperdocility. She confessed and asked for forgiveness. During the interview, the patient touched the female examiner's hand to feel its temperature and asked twice about how she was doing, despite the examiner's response the first time. This could indicate hypermetamorphosis and memory disturbance. The patient's symptoms strongly suggested KBS caused by subarachnoid

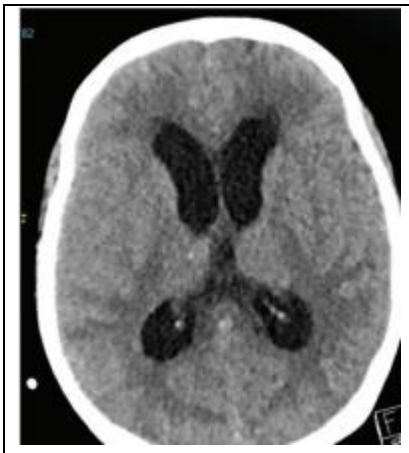

Figure 2: Computed tomography indicates hydrocephalus.

hemorrhage. On January 19, the patient was discharged with 1 mg risperidone and lost follow-up with her as she returned to her country.

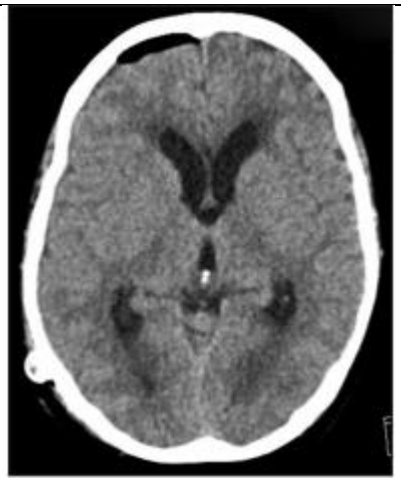

Figure 3: Computed tomography after shunt placement.

Discussion:

The exact prevalence of KBS is difficult to establish, with fewer than 200 published cases in the literature.[2] KBS is a clinical syndrome that might be missed or misdiagnosed. At least three symptoms are required to diagnose the condition. The symptoms include hypersexuality, hyperorality, placidity, visual agnosia, hypermetamorphosis, bulimia, amnesia, prosopagnosia, and hyperdocility.[3] In the present case, the author observed many symptoms of KBS including hypersexuality, hyperdocility, hyperorality, placidity, memory disturbance, and possibly hypermetamorphosis.[2]

Conclusion:

KBS is a rare neuropsychiatric illness. Collaboration among treating neurologists, psychiatrists, neurosurgeons, and radiologists is essential for determining the definitive diagnosis of KBS. The treatment of KBS is challenging due to its complex presentation, and further study was needed to provide definitive recommendations for clinical practice.

References:

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3. Case Report on Migraine Symptoms Caused by an Auricular Piercing

Introduction:

Migraine is a common neurological illness with complicated pathophysiology that affects both the central and peripheral nervous system. Migraine is currently ranked as the sixth most debilitating condition worldwide, the highest among all neurological disorders.[1] Migraines have significant direct and indirect costs. They can be tough to treat, so many people seek alternative medical treatments. One of these involves applying acupuncture to certain points on the ear that were considered to regulate migraine symptoms. Some patients have piercings in these areas in the hopes of alleviating their symptoms. Acupuncture or piercing may cause migraine symptoms, but this has not been investigated. There are no reported cases of auricular acupuncture or piercing triggering migraine symptoms in previously asymptomatic individuals.[2] In this case, a 27-year-old female with no prior migraine history experienced migraine-like symptoms following a piercing of the inferior crus of the antihelix.

Auricular piercings may play a part in the diagnosis or treatment of migraines, and physicians may find that taking the piercing out is a low-risk strategy to reduce symptoms.

Case Presentation:

A 27-year-old female with a history of cramp fasciculation syndrome, transient hemiplegic episodes, and no history of migraine with aura experienced persistent headaches, nausea, and occasional ipsilateral extremity weakness and paresthesias after piercing the inferior crus of her left antihelix (Fig. 1). She had her piercing done by a qualified practitioner and utilized ASTM F-136 grade (implant grade) titanium jewelry from Invictus Body Jewelry. Within 24 hours after the piercing, she experienced headaches and nausea that lasted many weeks. The patient had mildest (3/10 pain, 1/10 relief with acetaminophen) symptoms that she believed she had been used within a month, although they may still be present. Five months following the piercing, her symptoms increased. Her headaches and nausea had gotten worse, and her pain level had gone from an average of 3 to 6 out of 10. Her headaches started to focus behind and between her eyes. Without developing phonophobia, she experienced visual disturbances and photophobia. The frequency of the episodes increased, and they now happen almost every day for the entire day. The patient took acetaminophen for her symptoms, which helped, though she can't remember how much it alleviated her pain. After 2 months, she started experiencing occasional ipsilateral extremities weakness and numbness/tingling. Four

years earlier, the patient did experience a brief episode of hemiplegia and hemiparesthesia without headaches; at that time, the results of the CT scans of the neck, spine, brain, and carotid arteries were unremarkable, and physical therapy was effective in treating the symptoms. Given her previous normal imaging results and clinical history, more testing for her current symptoms was not required. The physical examination revealed no unusual findings and a clean piercing free of infection.

The patient kept receiving treatment to help with migraines. Finally, nine months later, the patient took the piercing out for routine cleaning and noticed that her symptoms went away in a minute. Since then, she hasn't had the piercing redone. She told the person who had done her first piercing, and they provided anecdotal reports of similar occurrences where migraines were linked to different head piercings, especially those in the auricular cartilage. Similar anecdotal reports about rook piercings have been shared to her by members of the community. The patient started experiencing similar, sporadic migraine attacks approximately 72 hours after the piercing was removed. They haven't happened as often as they did while the piercing was in. Notably, this patient did not experience any other piercing-related complications, including hematoma, chondritis, cellulitis, allergy, or keloid. In addition, the lobule, the helix, and the stem of the antihelix have all been pierced numerous times in this patient (Fig. 1). She did not experience any similar symptoms right away following any of these piercings, and her symptoms did not alter when these were put in or taken out.

Figure 1: Rook piercings include the antihelix's inferior crus (a), The inferior crus (b) is traversed vertically by the patient's piercing, The antihelix (c) and helical rim (d) superior crus. The daith piercing, located on the crus helix (e), differs from the rook piercing and may reduce migraine symptoms in some patients.

Discussion:

The trigeminal, facial, and vagus nerves, as well as the lesser occipital and greater auricular nerves, which were branches of spinal nerves C2 and C3, all contribute to the intricate innervation of the external ear. Migraines are thought to be caused by pain pathways in the trigeminal nerve. Some theories suggest that nociceptors of meningeal blood vessels, which

travel via afferent trigeminal pathways to the spinal nucleus of the trigeminal nerve, were responsible for headaches.[2] This was the idea behind the use of sphenopalatine ganglion block to relieve the symptoms of cluster headaches, migraines, and trigeminal neuralgia.[3]

Conclusion:

The association between the piercing and migraine symptoms was unclear, but it could be due to modulation of trigeminal or vagal pain pathways, as both cranial nerves innervate the auricle. However, piercings should not be disregarded as a possible contributing factor in patients presenting with migraine symptoms during the physical exam and history taking.

References:

1. Puledda F, Silva EM, Suwanlaong K, Goadsby PJ. Migraine: from pathophysiology to treatment. Journal of Neurology. 2023 Jul;270(7):3654-66.
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4. Case report on Immunoglobulin G4-related disease presenting as peripheral neuropathy

Introduction:

Immunoglobulin G4-related disease (IgG4-RD) is an uncommon and chronic condition characterized by high blood IgG4 levels and tissue infiltration by IgG4+ plasma cells. It is a fibro-inflammatory immune-mediated disorder that can affect almost any tissue or organ. IgG4-RD is known to exhibit a variety of aberrant radiography patterns and can affect the parenchyma, airways, pleura, and mediastinum. Because of this, it can be difficult to identify and distinguish it from the several illnesses that it mimics, including infections, other autoimmune diseases, and cancer. Often, a biopsy is necessary to make the final diagnosis.[1] Pathologically, IgG4-RD was linked to IgG4-positive plasma cells, storiform fibrosis, extensive lymphoplasmacytic infiltration, and obliterative phlebitis. IgG4-RD lesions have recently been reported along the spinal structure, although they don't seem common. There were few reports describing multiple spinal nerve root involvement in IgG4-RD that mimics peripheral neuropathy.[2] This case report described an uncommon instance of several sacral nerve roots involved with IgG4-RD.

Abdominal CT screening can help in the diagnosis of typical IgG4-RD-related abnormalities by ruling out IgG4-RD occurring in uncommon locations like nerve roots.

Case Presentation:

A 51-year-old man reported to the neurosurgery department with numbness in his right lower extremities that had increased over the past year. He was not on any medicine and had varicose veins, emphysema, and had undergone surgery for pneumothorax. A neurological examination found that the right Achilles tendon reflex was absent and that paresthesia affected the medial-posterior aspect of the right leg and buttock. The dermatome of left S1–S3 seems to match the paresthesia location. There were no obvious neurological abnormalities. Peripheral neuropathy may be the cause of the patient's neurological problems. No anomalies in rheumatoid factor, anti-nuclear antibodies, human immunodeficiency virus, syphilis antibodies, or malignancies were found in the blood or cerebrospinal fluid samples. Serum IgG levels were slightly higher (1974 mg/dL) than normal (870–1700 mg/dL). A pelvic magnetic resonance imaging (MRI) was done to make sure the mass wasn't the cause of the paresthesia. T1-weighted gadolinium-enhanced imaging shows a swelling and

uniformly increased appearance in the right S1 and S2 nerve roots. The dura showed signs of partial hypertrophic enhancement [Figures 1a and b]. On the basis of radiological results, sarcoidosis, lymphoma, or schwannoma were suspected.

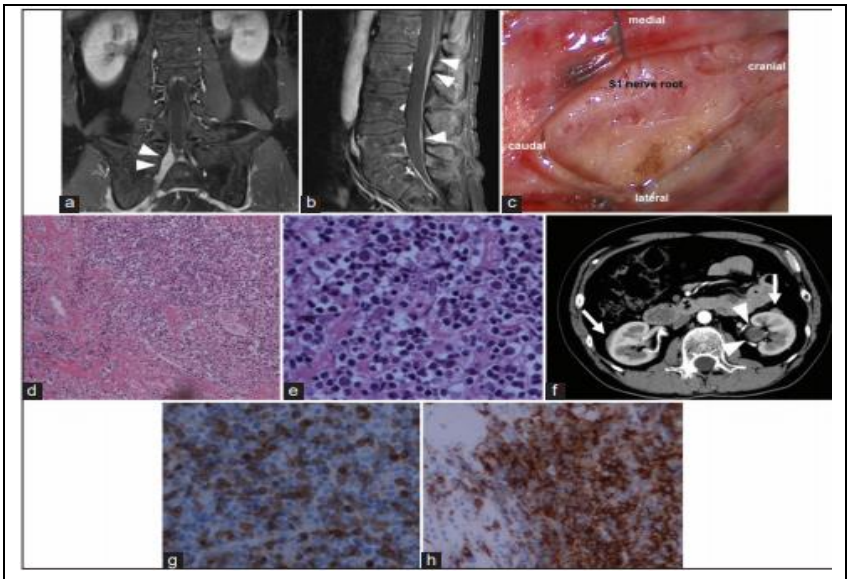

Figure 1: (a, b) The right S1 and S2 nerve roots (white arrowheads) are enlarged and amplified by the contrast agent. The hypertrophic lumbar dura (white arrowhead) is partially enhanced in (a) the coronal T1-weighted gadolinium-enhanced image and (b) the sagittal T1-weighted gadolinium enhanced image. (c) Opening the dura revealed reddish and swollen S1 nerve roots. (d and e) Lymphocyte aggregation was followed by the diffuse proliferation of fibrous tissue (hematoxylin-eosin staining at original magnification ×100 (d) and ×400 (e). (f, g) The specimen tested positive for CD3 (original magnification, ×400) and CD20 (original magnification, ×200). (h) Contrast-enhanced computed tomography shows a dilated left ureter (white arrowheads) and bilateral perirenal lesions (white arrows).

The hypertrophy dura did not compress the spinal cord, thus assumed it would not affect paresthesia. A biopsy was conducted on the enlarged S1 nerve root in order to rule out inflammation or cancer. For pathological assessment, a soft, reddish lesion of the S1 nerve root was obtained [Figure 1c]. The removed tissue tested positive for CD3 and CD20, and it had aggregated lymphocytes encircled by fibrous tissue that was expanding diffusely [Figures 1d–g]. These results are not consistent with lymphoma, which increases the possibility of another cancer or inflammatory illness. To rule out an abdominal lesion, abdominal contrast-enhanced computed tomography (CT) was used. The results showed bilateral perirenal lesions and a dilated left ureter [Figure 1h]. These results suggested the presence of IgG4-RD. IgG4 levels in the serum were 724 mg/dL (normal range: 4.8–105 mg/dL). In addition, urologists' laparoscopic examination of perirenal lesion samples showed the presence of fibrous tissue, plasma cells, and lymphocytes. There was a greater than 0.5 ratio between IgG4-positive plasma cells and IgG-positive cells. These results led to the diagnosis of retroperitoneal fibrosis caused by IgG4-RD in the perineural tissue. In order to verify this diagnosis, immunostaining was done, and fibrous structures, aggregating lymphocytes, and plasma cells were seen. Collagen fiber storiform patterns and obliterating phlebitis were seen. As a result, it was concluded that paresthesia may result from S1 and S2 nerve root involvement in IgG4-RD. Prednisolone 30

mg per day was recommended as part of an oral steroid regimen. Subsequently, the neurological symptoms decreased and the serum IgG4 level reduced. Furthermore, the follow-up MRI revealed that the enlarged S1 and S2 nerve roots had shrunk. When the symptoms did not return eighteen months after starting steroid therapy, prednisolone therapy (8 mg/day) was continued.

Discussion:

This was a rare case of IgG4-RD that affects the S1 and S2 nerve roots. IgG4-RD rarely affects the nervous system. Hypertrophic pachymeningitis, a common symptom of IgG4-RD in the nervous system, was typically found in intracranial lesions.[2] Furthermore, previous study showed that the spinal cord and nerve roots may also be affected.[3]

Conclusion:

Neurosurgeons must assess IgG4-RD, a rare condition that resembles peripheral neuropathy and has abnormal radiological characteristics. Abdominal CT screening can help diagnose common IgG4-RD-related abnormalities and rule out the possibility of it developing in rare areas like nerve roots.

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