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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 of Multiple sclerosis diagnosis after COVID-19 infection
2. Intractable Neuropathic Pain in Isaac Syndrome
3. A case report on Wernicke Encephalopathy after Bariatric Gastric Bypass surgery
4. Anti-TNF-α Agent-Induced Demyelinating Neurological Adverse Reactions

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 of Multiple sclerosis diagnosis after COVID-19 infection

Introduction:

Multiple sclerosis (MS) is a central nervous system autoimmune demyelinating disease that can manifest in a variety of ways. Symptoms of MS and those of lumbar spondylosis and/or post viral plexopathies, but in the other hand, can have a substantial overlap.[1] MS can mimic a variety of different disorders due to its wide range of neurological symptoms. Myelopathy caused by a herniated disc, spinal stenosis, or spondylosis, and, more recently,[1] COVID-19-related viral plexopathy are among them. Furthermore, evidence show that viral infections like COVID-19 can cause MS exacerbations.[6] Here's a case study of a 33-year-old woman who developed COVID-19 after lumbar surgery (anterior lumbar interbody fusion [ALIF]). The patient's postoperative symptoms/signs prompted a lumbar spinal surgery revision, however they were eventually linked to MS.

When patients come with myelopathic symptoms that are misinterpreted as "radiculopathy" due to spinal disease, the diagnosis of Multiple Sclerosis is often missed.

Case presentation:

A 33-year-old woman presented with 2 years of low back pain/radiculopathy with a medical history of hypothyroidism, polycystic ovarian syndrome, pseudotumor cerebri, pacemaker placement for bradycardia, a gastric sleeve, and a C6-7 cervical disc replacement. Patient had a considerable isolated weakness in the right lower extremity on examination, but no sensory or reflex abnormalities. The L5-S1 level had mild degenerative disc degeneration, a 3 mm minor degenerative retrolisthesis, and a right paracentral L5-S1 disc protrusion causing moderate lateral recess narrowing, according to lumbar X-rays and an MRI. Without complications, the patient underwent an L5-S1 ALIF. Patient had fevers one week after surgery, but a postoperative CT scan of the abdomen and laboratory tests were normal. Patient reported of new left leg paresthesias with weakness and diffusely decreased sensitivity to light touch two weeks after surgery. A lumbar CT scan revealed encroachment on the left L5-S1 neural foramen by the L5-S1. The patient received a steroid taper and sent for a lumbar CT scan, which "indicated" encroachment on the left L5-S1 neural foramen by the L5-S1. The MRI revealed an intramedullary "lesion" in the area of the conus medullaris at the L1-2 level, which was of unknown significance. After that, the patient had an ALIF revision for cage relocation. Symptoms continued to worsen after surgery. Multiple lab tests and MRIs of the cervical, thoracic, and brain were all normal. The patient was discharged, but was confined to a wheelchair. The L5-S1 ALIF cage was found to be in a good location on repeat X-rays. patient’s deficiency was likely consistent with transverse myelitis and/or poly-sensory neuropathy, according to an EMG of left lower extremity performed by a neurologist

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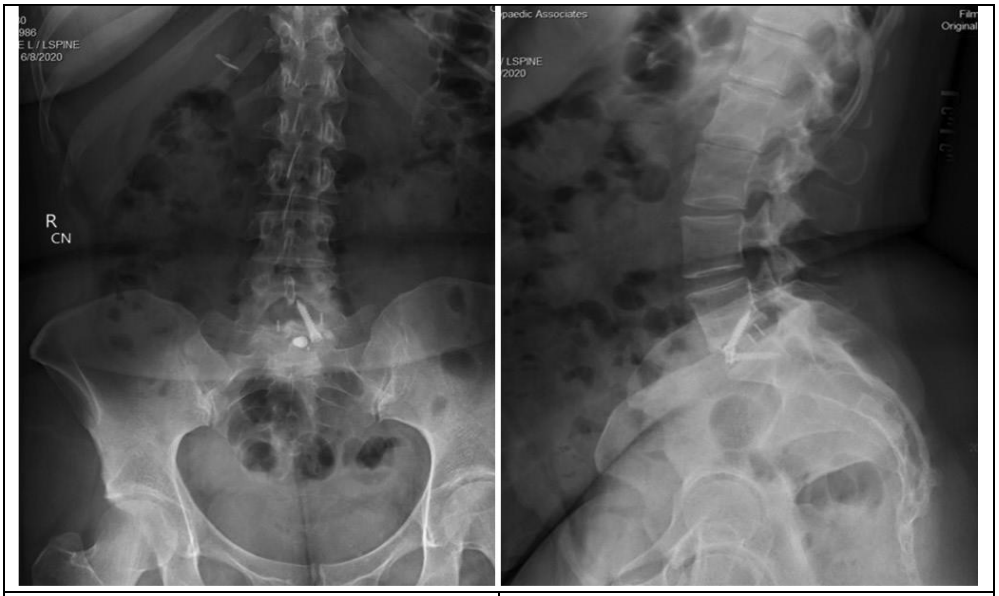
Patient received a complete spinal Myelo-CT four months later, now with further bilateral upper and lower extremities weakness/sensory loss and increased urine urgency with incomplete bladder emptying; it was negative. The cerebrospinal fluid recovered from the lumbar puncture, on the other hand, revealed results that were consistent with MS. After then, the patient received MS-modifying therapy.



Post-operative anteroposterior radiograph of the lumbar spine demonstrating anteriorly placed interbody cage at the L5-S1 level.	Postoperative lateral radiograph of the lumbar spine demonstrating anteriorly placed interbody cage at the L5-S1 level.

Discussion:

When patients come with myelopathic symptoms that have been misinterpreted as "radiculopathy" due to spinal pathology/spondylotic disease, the diagnosis of MS is frequently missed. Patients' failures to recover with surgery in these cases were eventually traced to the underlying diagnosis of MS, which could result in an unexpected aggravation of MS.[1]Further research shows that people with MS and substantial spinal pathology may benefit from surgery, despite the fact that the available data is of Low quality and contradictory. Some researchers have witnessed an increasing association between COVID-19 and the beginning of MS. [3] Furthermore, MS patients who are using anti-CD20 disease-modifying medications may be the most vulnerable to COVID -19 infections.[4] The patient's lumbar operations and suspected SARS-CoV-2 virus infection may have directly precipitated the onset/ flare of MS in this case.

Conclusion:

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When patients come with myelopathic symptoms they are misinterpreted as "radiculopathy" due to spinal pathologies, the diagnosis of MS is often missed. Furthermore, recent COVID-19 infections may act as triggers, accelerating the development of MS.

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2.Intractable Neuropathic Pain in Isaac Syndrome

Introduction:

Isaac syndrome (IS) is a condition characterised by diffuse, rapid, and asynchronous muscular contractions. Autoantibodies to the voltage-gated potassium channel (VGKC) complex are associated with this autoimmune condition.[1] Peripheral nerve hyper-excitability (PNH) disorders such IS, Morvan syndrome, and cramp-fasciculation syndrome (CFS) are associated with elevated VGKC antibody titers. Thymoma and small-cell carcinoma have both been linked to these autoantibodies.[2] this case is of A middle-aged man presented to the emergency department with a four-week history of generalised myalgia, intractable neuropathic pain, and muscular cramps. Further investigation indicated an elevated titer of VGKC complex antibodies, as well as neuromyotonic discharges on EMG, leading to the diagnosis of IS with intractable neuropathic pain, which is a rare symptom of the disease.

Case Presentation:

The combination of carbamazepine and subcutaneous immunoglobulin can relieve the Neuropathic pain in Issac Syndrome

A 45-year-old patient presented with muscle twitching, intense widespread burning pain, and episodic sweating in the limbs for four weeks. Patient was prescribed Vicodin and muscle relaxants, but they didn't help. Muscle fasciculations over the deltoids, triceps, forearm, thighs, and calf muscles were positive on examination. There was no evidence of muscular atrophy or hypertrophy, and all muscles had a strength rating of 4+. Asynchronous resting contractions involving the proximal and distal musculature were seen in these fasciculations. There were also visible asynchronous contractions in the abdominal musculature. Patient also complained of scorching pain in both of his distal extremities. A 5-day course of intravenous immunoglobulin was started for the patient (IVIg). Burning pain in the extremities necessitated massive dosages of Dilaudid and subsequently morphine, which provided only temporary relief. After receiving IVIg. Muscle fasciculations improved and eventually resolved with only a modest improvement in pain, and

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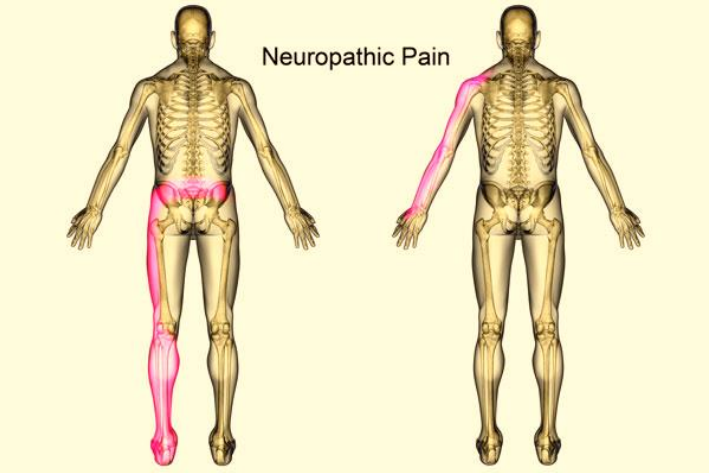
the patient was transitioned to oral Percocet and gabapentin without significant pain relief. On the pain evaluation scale, the average pain was a 9/10. The complete blood count and basic metabolic panel revealed an increased creatine kinase of 620 U/L and a white blood cell count of 9.2 10E9/L, respectively. Negative ANA and anti-dsDNA antibodies, negative GAD antibodies, negative ACH receptor antibody, negative extractable nuclear antigen panels that included anti-Jo, antiscleroderma scl-70, anti-RNP, anti-Smith, anti-ro, and anti-la, and TSH within normal levels, as well as Lyme antibodies. In the right medial gastrocnemius and left deltoid muscles, electromyography and nerve conduction investigations revealed spontaneous activity in the form of grouped +3 discharges as well as neuromyotonic discharges. The potentials of the motor units were normal. The presence of VGKC antibodies at 63 pmol/L (normal 0–31) confirmed the diagnosis of IS. The thymoma was not found on the chest CT scan. The whole-body PET scan revealed no signs of cancer. Despite utilising IVIg, carbamazepine, gabapentin, opioids, phenytoin, dantrolene, and benzodiazepines, the neuropathic pain remained. At the 1-year follow-up visit, the combination of carbamazepine and subcutaneous immunoglobulin (SCIg) provided moderate pain reduction. On average, moderate pain reduction was rated as a 5/10.



Discussion:

IS is characterised by a gradual stiffness of the generalised musculature, as well as frequent cramping and slow mobility. Hyperhidrosis, sialorrhea, piloerection, and stomach discomfort are all prevalent symptoms. Neuropathic pain is a rather uncommon symptom in IS patients. [3] The most common symptom of IS in this case was neuropathic pain. This is most likely related to an increase in the hyperexcitability of peripheral neurons. According to EMG findings, the myotonias seen in the clinical picture are caused by spontaneous depolarization of these nerves .[4] Researchers believe that, in this case, hyperexcitable nerves can cause peripheral neuropathic pain syndrome. PNH illnesses such as IS, Morvan syndrome, limbic encephalitis, and CFS have elevated VGKC antibody titers. Despite the fact that the pathogenesis of IS involves autoantibodies causing VGKC dysfunction, leading in a potassium-ion imbalance and symptoms of muscle hyperexcitability, this case brings up the question of how neuropathic pain might accompany the normal appearance.[3]

Conclusion:

Finally, this case shows a patient with IS who suffered from intractable generalised neuropathic pain. The pain was resistant to a variety of drugs, but the patient eventually found relief with a

combination of carbamazepine and SCIg. To further understand the pathophysiology of neuropathic pain in IS and to determine the appropriate therapy plan, more research is needed.

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3.A case report on Wernicke Encephalopathy after Bariatric Gastric Bypass surgery

Introduction:

Obesity is associated with a 5–10year reduction in life expectancy and a considerable increase in mortality from cardiovascular disease and cancer.[1] Bariatric surgery has long been recognised as the most effective treatment for morbid obesity, resulting in rapid weight loss, decreased macrovascular problems, and a 40% reduction in all-cause mortality . Patients with a BMI of 40.0 or 35.5–39.0 and a weight-related conditions, such as hypertension, type 2 diabetes, or severe sleep apnea, are candidates for gastric bypass surgery . Over a third of patients after gastric bypass surgery have nutritional deficits as a result of reduced absorption [1]. Thiamine (vitamin B1) deficiency, which produces Wernicke encephalopathy , is one nutritional cause of neurologic problems following gastric bypass surgery. Thiamine is absorbed in the duodenum, which is skipped in the new GI tract, and thiamine deficiency causes cognitive impairments. Ophthalmoplegia, ataxia, and encephalopathy are the classic symptoms of Wernicke encephalopathy. Wernicke- Korsakoff syndrome, which includes irreversible short-term memory loss and confabulation, can develop if symptoms are not treated quickly. FLAIR imaging of Wernicke encephalopathy reveals bilateral hyperintensities in the mammillary bodies, thalami, and periaqueductal region.[1,2] The classic triad of Wernicke encephalopathy symptoms is the focus of most reports about thiamine deficiencies from gastric bypass surgery.[1,2] Following gastric bypass surgery, this patient developed Wernicke encephalopathy with concurrent polyneuropathy, vitamin A insufficiency, and a flat affect. Furthermore, symptoms were present and progressed before imaging evidence of Wernicke encephalopathy appeared.

Wernicke encephalopathy is becoming a more recognized nutritional and neurological complication of bariatric surgery that can have devastating effects if it is not recognized quickly

Case Presentation:

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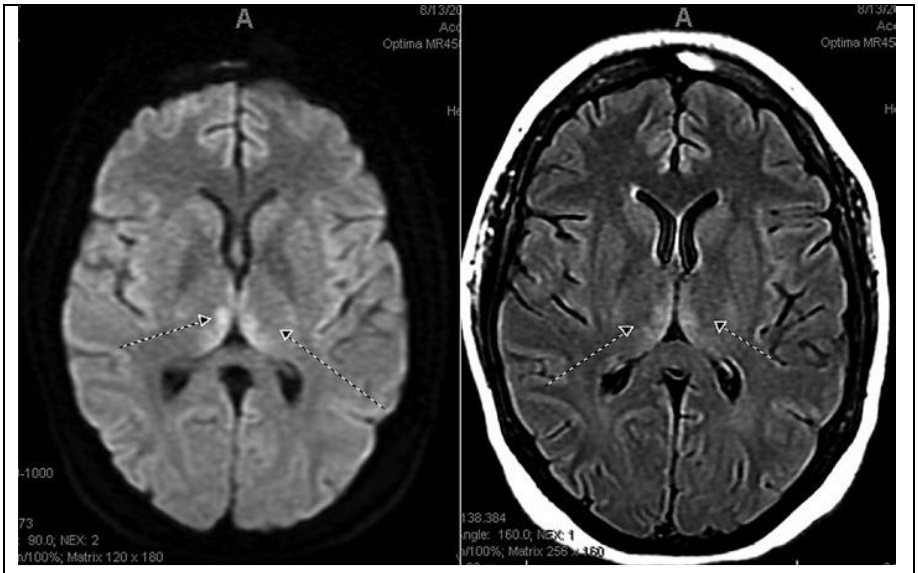
A 40-year-old Patient was transferred from another hospital due to worsening balance and sensory changes. Patient had previously been diagnosed with hypertension, hyperthyroidism, gastroesophageal reflux disease, myocardial infarction, and morbid obesity (BMI: 48.9). Two months prior to admission, the patient had Roux-en-Y gastric bypass surgery and had dropped 80 pounds. The patient had complained of nausea and hyperemesis of a claimed GI disease for weeks prior to admission to an outside hospital. Patient then started to get numbness in her inner thighs, which progressed to poor balance and sensory alterations, which characterised as bilateral numbness from nipple line to ankle. Upon arrival, a physical examination is performed. There was no nystagmus and dysesthesia from the nipple line to the ankles. Head CT and MRI scans, lumbar puncture, and MRI scans of the cervical, thoracic, and lumbar spine were all part of the first workup at the outside hospital. An MRI of the cervical spine revealed mild disc bulging at C5–6, but no demyelination. At T6–7 These findings did not correspond to patient's current symptoms, indicating that they were most likely coincidental. The rest of the procedure went without a hitch. The patient developed diplopia in addition to existing symptoms on her fourth day in the hospital, which was reevaluated by neurology.



Brain MRI demonstrates symmetrical hyperintensities of the mammillary bodies and dorsomedial thalami (indicated by arrows). MRI; magnetic resonance imaging.

Multidirectional horizontal and vertical nystagmus, decreased facial expression, dysesthesia from the nipple line to the ankles, bilateral lower extremity weakness, more prominent on the left, diminished brachioradialis reflex, and absent knee and ankle reflexes were all observed during the physical examination. These findings triggered a more serious examination. The next day, a repeat MRI revealed symmetrical T2/FLAIR signal in the mammillary bodies and dorsomedial thalami, which was compatible with Wernicke's encephalopathy. EMG revealed acute axonal polyneuropathy, which could be due to infiltrative, toxic, or nutritional neuropathies, or a rare variant of Guillain-Barre syndrome called acute motor axonal polyneuropathy. In a situation of hypoglycemia previous to the procedure, a repeat lumbar puncture revealed normal white blood cells and protein, as well as

low glucose levels. Significant thiamine (30 nmol/L) and vitamin A (37 g/dL) deficits were discovered in the patient. The neurologic symptoms improved over the next several days after the high-dose intravenous thiamine supplementation. Patient was given oral thiamine, vitamin A, and multivitamin pills before being discharged to an inpatient rehabilitation clinic.

Discussion:

Roux-en-Y is the most common gastric bypass surgery, and it has been linked to nutritional inadequacies as a result of the decreased stomach size and bypass of absorptive small intestine sections.[1] Nutritional deficiencies, particularly B vitamins, are infrequent causes of neurological problems after surgery.[3] Thiamine (vitamin B1) insufficiency, which causes Wernicke encephalopathy, is becoming a more well-known consequence following gastric bypass surgery. Early detection is critical since this disease can lead to Wernicke-Korsakoff syndrome, which includes short-term memory loss, psychosis, confabulation, and gait problems.[1] These impairments can be irreparable and even fatal, therefore it's vital to stop them from getting worse. The classic trio of signs of Wernicke encephalopathy are gait ataxia, altered mental status, and nystagmus. Wernicke encephalopathy was not first suspected in this case because the patient only noted one of the three characteristic symptoms, gait ataxia. This patient had flat affect and proptosis rather than changing mental status. The presence of visual symptoms is the least likely of the three classic signs, although it can suggest a complete thiamine deficit.[4]

Conclusion:

Wernicke encephalopathy is becoming a more well-known nutritional and neurological consequence of bariatric surgery that can be fatal if not diagnosed early. In order to avoid irreversible complications, healthcare providers should be able to quickly recognise the signs and symptoms. Even if the initial workup was negative, a high clinical suspicion should result in a complete evaluation.

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4.Anti-TNF-α Agent-Induced Demyelinating Neurological Adverse Reactions

Introduction:

Tumor necrosis factor (TNF-α) is a pleiotropic cytokine with multifunctional proinflammatory features that plays a vital role in host defence systems.[1,2] The pathogenesis of a variety of disorders, including rheumatoid arthritis, Crohn's disease, atherosclerosis, psoriasis, sepsis, diabetes, and obesity, has been related to abnormal TNF-α development and TNF receptor signalling. As a result, TNF-α and its receptor have been considered as a therapeutic target for a variety of illnesses. Anti-TNF-α agents have changed the treatment of rheumatoid arthritis, ankylosing spondylitis, psoriasis, psoriatic arthritis, juvenile polyarticular rheumatoid arthritis, and inflammatory bowel disease due to their efficacy and safety. These agents work by inhibiting soluble TNF-α from attaching to the TNFR1/TNFR2 receptors .[1] Many anti-TNF-α agents are currently approved for the treatment of inflammatory illnesses such as rheumatoid arthritis and inflammatory bowel disease. Aside from the usual presumed infectious difficulties, rare cases of neuroinflammatory demyelinating disorders, such as multiple sclerosis (MS), myelitis, and optic neuritis, have been documented with the use of anti-TNF alphas. Two cases of demyelination have been reported as a result of anti-TNF-α treatment. These cases were created to raise physician knowledge of the anti-TNF-α therapy's side effects.

Anti-TNF-α agents cause demyelination, Doctors should be aware of the event of any neurological side effects

Case Presentation:

Case 1: Adalimumab was prescribed to a 21-year-old Patient with HLA-B27-negative peripheral spondyloarthritis. Patient got severe headaches, urine incontinence, and bilateral lower limb numbness after being on adalimumab for two years, which moved towards the middle of the trunk over several days. Neurological examination revealed dysesthesias below the dermatome T4 and brisk tendon reflexes, with the rest of the

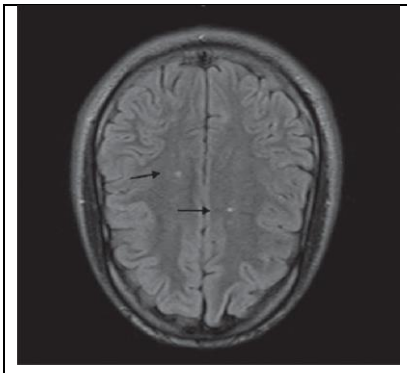

Figure 1: (a) there are few small foci of T2 and FLAIR hyperintensity noted in the periventricular white matter (bilaterally), perpendicular to the ventricular margin.

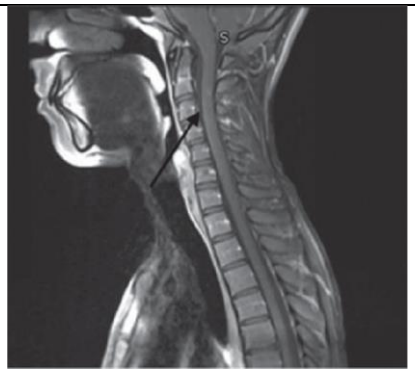

Figure 1: (b) there is a small area of T2 hyperintensity with corresponding postcontrast enhancement noted in the cervical spinal cord at the C2-C3 level

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examinations being normal. Multiple hyperintense lesions on brain MRI in the left paramedian anterior pons, left posterolateral medulla, and bilateral periventricular white matter, some of which involve the optic radiations, were used to confirm the diagnosis of multiple sclerosis (MS). Figures 1(a)–

1(d) reveal similar lesions in the right lateral aspect of the corpus callosum splenium and the cervical spinal cord at the C2-C3 level, as well as a tiny area of T2 hyperintensity in the thoracic spinal cord at the T5 level. After stopping the adalimumab, the neurological problems disappeared completely within one month. As a result, no pulse steroid was administered, and the MRI was not repeated. He did, however, suffer abdominal pain and loose bloody motions, which led to an ileocolonoscopy and biopsy, which confirmed the diagnosis of Crohn's disease. Surprisingly, researchers discovered that he had a strong family history of inflammatory bowel disease. Ustekinumab helped him control his inflammatory bowel disease and peripheral arthritis, and a four-year follow-up revealed no relapses.

Case 2: a 19-year-old Patient had new-onset dizziness and hand tremors after two years of taking adalimumab for juvenile idiopathic arthritis and chronic anterior uveitis. Intention tremors, ataxia, dysmetria, and dysdiadochokinesia were found on neurological examination, along with normal tone, power, and sensibility; reflexes were slower. Patient had never been diagnosed with MS. Multiple hyperintense white matter lesions were found on MRI, particularly in the bilateral cerebellar peduncles, periventricular, subcortical and juxtacortical, brain stem, corpus callosum, and left anterior thalamus, as well as multiple patchy T2 hyperintensities in the cervical and midthoracic spinal cords, with foci of enhancement in the midthoracic spinal cord, consistent with active lesions as shown in Figures 2 (a) & 2 (b).The existence of CSF distinct oligoclonal bands was discovered during CSF investigation. Patient's symptoms improved almost completely once stopped using adalimumab; Patient was given a pulse steroid, but Patient declined, and arthritis and uveitis were well controlled with methotrexate. After a year, a repeat MRI revealed that the prior lesions had receded, but it also revealed new lesions in various regions. To Treat the arthritis and demyelinating illness, the patient was switched to

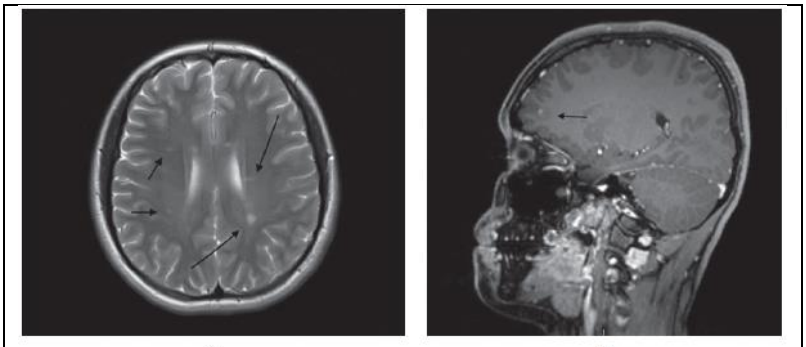

Figure 2:(a) there are multiple periventricular, subcortical, and juxtacortical white matter foci of T2 and FLAIR hypersignal in bothcerebral hemispheres, brain stem, bilateral middle cerebellar peduncles, corpus callosum, and left anterior thalamus. (b) *ere is focal enhancement noted in the left peritrigonal white matter lesion.

rituximab. Another MRI was performed a year later, and the earlier demyelinating lesions appeared to be stable, with no evidence of disease activity.

Discussion:

Anti-TNF medication has been demonstrated to cause CNS demyelination in multiple publications from different countries.[3] However, it is unclear whether these demyelinating occurrences are merely coincidental or causally linked to the administration of TNF-αantagonists .[1] Clinical trials using TNF-α blockers in MS patients revealed a rise in demyelinating episodes , as well as complete symptom remission after discontinuation of this treatment .[1] The temporal relationship, as well as the rechallenge phenomena , support a causal relationship.[1]

Conclusion:

Despite the limited number of patients, this study contributes to the growing body of evidence that anti-TNF-α agents cause demyelination, which doctors should be aware of. In the event of any neurological side effects, discontinuing TNF-α blockers as soon as possible and getting an urgent MRI scan to confirm the diagnosis are crucial.

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