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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.

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MICRO LABS SYNAPSE
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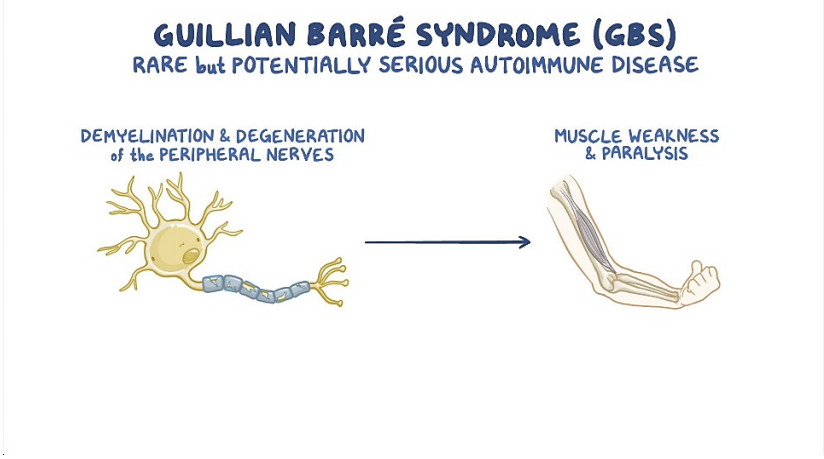
1. Correlation between acute brucellosis and a presentation resembling Guillain-Barré syndrome- A case report

Introduction:

Brucellosis is a zoonotic illness that has been documented since the past; nevertheless, the cause was established only in the nineteenth century. Brucella species that cause human brucellosis are B. melitensis, B. abortus, B. suis, and B. canis. In humans, Brucellosis infection can cause a variety of symptoms, including osteoarthritis, sacroiliitis, hepatitis, and neurological problems. Neurological symptoms occur in 3-5% of individuals and might include meningoencephalitis, cerebellitis, myelitis, cranial neuropathy, and peripheral neuropathy. Peripheral nervous system involvement was detected in 7% of neurobrucellosis patients; this involvement can take the form of chronic polyradiculoneuritis or acute neuropathy with Guillain-Barre syndrome (GBS)-like symptoms, although it is rarely recorded.[1] It is spread to humans by direct contact with an infected animal or through the consumption of unpasteurized milk products. Hyperthermia, exhaustion, weight loss, lack of appetite, and acute pain in the joints, spine, and lower back are the most prominent symptoms. Neurobrucellosis (NB) affects both the central and peripheral nerve systems and can occur at any stage of the illness. The frequency of NB in children ranges from less than 1.0% to 2.2%, with a death rate ranging from 0% to 27% with a little male predominance. Microbiological and/or biochemical evidence from cerebrospinal fluid (CSF) is used to make the diagnosis.[2] This case report describes a child who had acute brucellosis together with GBS that was detected both clinically and by laboratory testing.

It is important to check for brucellosis in all patients with acute paralysis, especially if they reside in an area where the illness is common.



Guillain-Barré syndrome

Case Presentation:

A 4-year-old boy was brought to the paediatric unit after he had a 15-day episode of a persistent fever that had been recorded at 38.5–39 °C four or five times a day. The fever was partially treated with antipyretics, and the boy also had weakness in his lower limbs, lethargy,

and headaches. Nonsteroidal anti-inflammatory drugs (NSAIDs) were used to treat the child, but there was no improvement and the child's weakness increased. The patient described consuming unpasteurized dairy products, living in a community in a region where brucellosis is widespread, and having direct contact with infected animals. Wright serologic tests showing brucellosis (more than 1/320) led to the diagnosis, and he was given oral medication for 45 days without finishing the course of therapy. Mild paresthesia and muscular weakness were discovered during a physical assessment that also included a neurological test. His lower extremities lacked deep tendon reflexes (DTRs), and he had a stiff neck with Kernig's sign. There were no abnormal plantar reflexes. There were no cutaneous reflexes in the abdomen, and the sphincter was unaffected. During the physical examination of the abdomen, the spleen was palpable two centimetres below the costal border, but everything else was normal. The laboratory study revealed that there was an increase in both creatine phosphokinase (CPK) and erythrocyte sedimentation rate (ESR) (Table 1). Typhoid was found to be absent from the Widal test (O: 1/80; H: 1/80), but present in the Wright test (more than 1/320). CSF study revealed albumino-cytological dislocation. Ultrasound of the abdomen revealed moderate splenomegaly with no signs of space-occupying lesions. Axonal and demyelinating polyneuropathy were detected by a nerve conduction study (NCS) and needle electromyography (EMG). There was a reduction in the leg's sensory nerve transmission velocity. The patient's diagnosis of acute brucellosis with a GBS-like presentation was made based on previous medical information. Sulfamethoxazole+ trimethoprim (10 mg/kg/day), rifampicin (15 mg/kg/day), gentamicin (5 mg/kg/day), and dexamethasone (0.5 mg/kg/day) were the medications used to treat the patient. Following seven days of therapy, the child's symptoms which included a fever, headache, and backache were improved. However, the child's lower extremity discomfort persisted, along with the lack of tendon reflexes, and he was still unable to walk. After 15 days, the patient was discharged with instructions to continue therapy with sulfamethoxazole+ trimethoprim (10 mg/kg/day), rifampicin (15 mg/kg/day), and vitamin supplements for 6 months. After being discharged from the hospital, the child gradually became better. A month after, his lower limb muscle weakness improved and he began to walk with assistance. After two months, he was walking on his own. The child is healthy right now, moving normally and growing at the normal pace.

Table 1. Laboratory data of the case

Test	Result	Normal range	Test	Result	Normal range
WBC (10^3/μL)	11.9	6.2–17	AST (U/L)	36	5–40
Neutrophils (%)	37	40–60	ESR (1 hour)(mm/hour)	30	0–10
Lymphocyte (%)	67	20–40	Glucose (mg/dL)	100	70 -100
Hb (g/dL)	11.5	11–13	CPK (U/L)	852	21–230
MCV (fl)	66	70–85	Urea (mg/dL)	33	15–36
PLT (10^3/μL)	322	150–450	Creatinine (mmol/L)	0.2	0.5–1.3
CRP (mg/dL)	3	<5	K (mmol/L)	3.9	3–4.5
ALT (U/L)	39	7–55	Na (mmol/L)	133	135–145

WBC white blood cell, HB hemoglobin, MCV mean corpuscular volume, RDW red cell distribution width, PLT platelets, CRP C-reactive protein, ALT alanine aminotransferase, AST aspartate aminotransferase, ESR

erythrocyte sedimentation rate, CPK creatine phosphokinase.

Discussion:

Hughes published the first report on NB in 1896. It is uncertain how a Brucella infection induces polyradiculopathy during the acute stage of sickness and sets off an immune response against autoantigens. Sulfamethoxazole+ trimethoprim (10 mg/kg/day), rifampicin (15 mg/kg/day), gentamicin (5 mg/kg/day), and dexamethasone (0.5 mg/kg/day) were the medications used to treat NB.[2] Similarly, in a study by Dammak M et al showed that the combination of two or three appropriate antibiotics (doxycycline, rifampicin, trimethoprim–sulfamethoxazole, streptomycin, or ceftriaxone) for extended periods of time (ranging from two to fifteen months, though at least six months is advised) with intravenous immunoglobulin or plasma exchange sessions is the commonly used treatment for NB (GBS with brucellosis).[3]

Conclusion:

The present case illustrates a unique neurologic presentation of brucellosis. All patients with acute paralysis should be evaluated for brucellosis, particularly if they live in a region where the disease is prevalent.

Reference:

1. Alanazi A, Al Najjar S, Madkhali J, Al Malik Y, et al. Acute brucellosis with a guillain-barre syndrome-like presentation: a case report and literature review. Infectious Disease Reports. 2021;13(1):1-10.
2. Doya LJ, Haidar I, Sakkour S. The association between acute brucellosis with a Guillain-Barré syndrome-like presentation: a case report. J Med Case Rep. 2023;17(1):25.
3. Dammak M, Kacem I, Farhat N, Gouider R, Mhiri C. Neurobrucellose. Revue Neurologique. 2018;174(3): S162

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2. Moersch-Woltman Syndrome with Positive Anti-GAD Antibodies- An unique Case Report

Introduction:

Moersch-Woltman Syndrome (MWS), commonly known as Stiff Person Syndrome (SPS), is a rare, progressive disorder characterised by excessive muscle activity caused by a loss of inhibition in the brain and spinal cord. It appears as progressive muscular stiffness, most often in the axial muscles.[1] It was described for the first time in 1956. Stiff-person syndrome (SPS) is an uncommon illness with an estimated frequency of 1-2 cases/1,000,000 in the general population. Females are 2-3 times more likely to experience it, with symptoms often appearing between the ages of 20 and 50. Although antibodies against glutamic acid decarboxylase (GAD) and amphiphysin are related with stiff-person syndrome, their presence is not required for diagnosis. The therapy should be multidirectional, including immunomodulation, symptomatic treatment, monitoring and treatment of overlapping autoimmune diseases, and surgery.[2] The aetiology of this disease is rarely identified, making disease development difficult to treat. The cause of GAD autoimmunity manifestation in MWS patients is unknown, and whether MWS fits the criteria for classification as a neuro-autoimmune disorder is controversial. It has not been proven that GAD antibodies are the only cause of MWS, and the potential of GAD functioning as a biomarker for the disease is very questionable.[1] The purpose of this case report is to demonstrate the relevance of the diagnostic problems associated with MWS, as well as the significance of glutamic acid decarboxylase (GAD) antibodies.

> Combining immunotherapy with GABA-enhancing medications is the usual and rational treatment approach for Moersch-Woltman Syndrome. This is because these two therapeutic approaches operate in distinct ways: One deals with autoimmune responses, while the other with the underlying causes of illness.

Case Presentation:

A 57-year-old female presented to the neurology clinic with the main complaints of persistent, slowly worsening generalised muscular stiffness, pain, and spasms in her head, neck, and back during the previous 15-20 years. Her everyday activities have been greatly affected by these persistent and increasing symptoms, which made worse by lack of sleep and improved by rest and exercise. She recalls having a brain injury in the 1990s, but there was no underlying weakness. Her present symptoms include burning fingers, stiffness, tingling in both arms, numbness in the glutes while sitting for extended periods of time, spasms in her muscles without stable posture, stiffness in her spine, loss of balance, scattered spasms in her muscles, and pain in her entire body. She also has trouble sleeping, bending over, and turning,

as well as autonomic signs like constipation and urgency in the urine. Prolonged postures exacerbate her discomfort, and drugs only give temporary relief. The patient is known to be allergic to erythromycin, fish product derivatives, iodine-containing contrast agents, porcine derivatives, and shellfish. In the past, her mother experienced osteoarthritis and colon cancer, and also had age-related worsening of her chronic pain. She has a medical history of depression, anxiety, and migraine headaches. She takes a number of drugs to treat her symptoms, such as fovatriptan, gabapentin, ibuprofen, magnesium oxide, naproxen, ondansetron, pantoprazole, polyethylene glycol, polyvinyl alcohol, ropivacaine, triamcinolone, epinephrine, and zolmitriptan. She seemed stiff and moved tightly during the test, with no trunk twisting. Due to her stiffness, she preferred standing. Her arms and legs were weak, she had trouble elevating her leg when walking, and she dragged her feet occasionally. Upon doing a neurological examination, it was found that the patient had broad-based gait, 3/5 graded muscular tone, reduced distal muscle strength, stiffness in the limbs, and normal reflexes. All of the neurological evaluation's findings were normal, including the eye examination. Laboratory tests, MRI scan that showed no abnormalities, and an NCS and EMG were among the further examinations. Notably, the patient's positive result from the GAD antibody test supported the diagnosis of MWS. Clonazepam and Baclofen are used as part of the therapy plan. She reported a 15-20% reduction in spasms and an improvement in her everyday activities during a three-week follow-up. For immediate alleviation, an intravenous immunoglobulin treatment is planned. She noticed a 30–40% increase in her ability to do daily tasks and her spasticity after two months.

Table 1. Summary of the differential diagnosis of Moersch-Woltman Syndrome.

Serial number	Diagnosis	Differentiating Presentation	Diagnostic tests	Comparison with MWS
1.	Myelopathies	Upper motor neuron, lower motor neuron signs, sensory deficits	MRI confirms the diagnosis.	In MWS, MRI is normal.
2.	Dystonias	Variable abnormal posturing, significant muscle pain, and cramping.	(EMG shows pulsating nerve signals being transmitted to the muscles even at rest.	EMG shows continuous motor unit activity in agonist and antagonist muscles in MWS.
3.	Spinocerebellar Ataxia	Hypermetric, slow saccades, nystagmus, areflexia, tremors, intellectual disability	Genetic testing confirms the diagnosis.	No genetic testing is required. Preserved intelligence in MWS.
4.	Primary Lateral Sclerosis	Onset after 50 years of age, Spasticity, Hyperreflexia, Babinski sign positive.	Pringle's Criteria for diagnosis.[13]	Anti-GAD for diagnosis of MWS.
5.	Neuromyotonia	Hyperhidrosis, muscle fasciculations, quivering of the muscle, myoclonic jerks, and myotonia-like symptoms	Fibrillation potentials and fasciculations on EMG.	EMG shows continuous motor unit activity in agonist and antagonist muscles in MWS.
6.	Multiple Sclerosis	Off and on UMN symptoms, Temperature sensitivity, Optic neuritis	MRI for diagnosis	MRI normal in MWS, no temperature sensitivity
7.	Parkinson's disease	Tremor, Rigidity, Akinesia	Clinical, DaT scan, MRI	DaT Scan is normal, no tremors are present in MWS.

Legend: MRI: Magnetic Resource Imaging, MWS: Moersch-Woltman Syndrome, DaT: Dopamine transporter, EMG: Electromyography, GAD: Glutamic Acid Decarboxylase; UMN: Upper Motor Neuron.

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Discussion:

MWS is an uncommon central nervous system condition characterised by proximal and axial muscular stiffness and painful, stimulus-triggered muscle spasms. A wide range of conditions affecting the brain, spinal cord, and muscles, such as myelopathies, are included in the differential diagnosis for MWS. Differential diagnosis of Moersch-Woltman Syndrome is mentioned in table 1. Immunotherapy and GABA-enhancing drugs are used in combination as the standard and sensible therapeutic strategy. This is due to the different methods by which these two therapy modalities function: one targets autoimmune reactions, while the other treats underlying disease processes.[1] According to Dalakas et al., antispasmodic medicine should be used in combination with antibody therapy as soon as possible.[3] Furthermore, a study by Galli JR et al. demonstrated since there is no therapy for MWS, working with a professional and keeping symptoms under control can make it easier to live with the condition.[4]

Conclusion:

The wide range of differential diagnoses for MWS emphasises the significance of a careful assessment and the exclusion of other possible illnesses. To improve knowledge and enable the development of more efficient therapies for this difficult disease, more research and awareness are required. Accurate diagnosis, subsequent management and treatment plans can be achieved by identifying the possible existence of MWS.

References:

1. Pitliya A. A Rare Case Report of Neurological Condition: Moersch-Woltman Syndrome with Positive Anti-GAD Antibodies. International Journal of Medical Students. 2023;11(3):233-6.
2. Jastrzębski K, Romanowska N, Rek-Pacześ M, Kępczyński Ł, et al. Stiff-person syndrome with sensorimotor polyneuropathy–case report. Current Neurology. 2021;21(1):47.
3. Dalakas MC. Therapies in Stiff-Person Syndrome Advances and Future Prospects Based on Disease Pathophysiology. Neurol Neuroimmunol Neuroinflamm. 2023;10(3):e200109.
4. Galli JR, Austin SD, Greenlee JE, and Clardy SL. Stiff person syndrome with anti-GAD65 antibodies within the national veterans affairs health administration. Muscle Nerve. 2018;58(6):801–4.

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3. Case Report on Spinal Metastasis Inducing Dropped Head Syndrome at a Chiropractic Office

Introduction:

Dropped head syndrome is something that clinicians who treat spinal symptoms should be aware of since it can be caused by serious disease, including a vertebral fracture associated with an underlying malignancy.

Metastatic illness frequently affects the spinal column and can result in pathological vertebral fractures, which can cause pain, incorrect posture, and neurological consequences such as paralysis or poor balance.[1] Systemic therapeutic advances have increased the number of patients requiring treatment for spinal metastases each year, with an estimated 100,000 additional patients each year. In cases of metastatic spine disease, mechanical instability and neural element compression are the two primary reasons for surgery.[2] Dropped head syndrome is an uncommon condition that causes a significant kyphotic malformation of the cervicothoracic spine in both sitting and standing postures. The syndrome is most common in older persons and can be caused by a number of disorders, including myopathy, spondylosis, spinal cord damage, and, in rare cases, vertebral fracture, which can be caused by underlying spinal metastasis.[1] In this case study, a 79-year-old man who had a history of degenerative cervical spondylosis was unable to hold his head up or stand up straight for two years.

Case Presentation:

A 79-year-old man with a history of degenerative cervical spondylosis presented to a chiropractor with a two-year history of difficulty holding his head erect and standing up straight. He stated that his symptoms began progressively as tiredness in his neck and upper back. He also had terrible balance and needed a cane and the aid of a family member to walk. The patient reported only little neck and back discomfort, graded 1/10 on the numeric pain scale, and denied any radiating pain, paresthesia, or numbness in the upper and lower limbs. The patient consulted his family doctor six months before seeing the chiropractor for symptoms that were comparable but a little less severe. The healthcare practitioner suggested that he do muscle-strengthening activities with a physiotherapist. Nevertheless, his posture continued to worsen. As the patient's problems grew worse over time, they decided to get a second opinion from a chiropractor. At the time of presentation, the patient was seen to be seated and standing with a chin-on-chest (dropped head) position (Figures 1A and 1B). After resting supine, the dropped-head posture improved to some extent. Upon nuchal palpation, grating sensations and decreased muscle tone were detected. The patient's cervical extension was constrained to 0° in its active range and to 20° in its passive range, which resulted in crepitus. Diffuse weakness was found during manual muscle testing of the upper limbs, with each motion receiving a 4 out of 5 rating on the Medical Research Council scale. Testing of the upper and lower extremities indicated bilaterally reduced (1+) Achilles stretch responses as well as a lessened left patellar reflex. There were no tremors, stiffness, hyperreflexia, dysarthria, or abnormal reflexes (such as Babinski or Hoffmann) during the testing of the cranial nerve.

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Given the patient's deteriorating posture, generalised weakness, lack of response to conservative therapy, and advanced age, the chiropractor regarded thoracic spinal compression fracture as the working diagnosis, along with cervical and lumbar radiculopathy. The chiropractor suggested magnetic resonance imaging (MRI) to assess the entire spine, but the patient selected computed tomography (CT) because to its lower cost. CT indicated widespread mixed lytic and sclerotic alterations across the axial skeleton, including all spinal areas and many ribs, indicating widespread bone metastases. There was also a minor left pleural effusion, fibrotic alterations at both lung bases, and an enlarged prostate. There was no cervical fracture, but there was cervical spondylosis and lytic/sclerotic alterations. Because of the widespread mixed lytic/sclerotic alterations and fractures, the chiropractor suspected metastases and quickly sent the patient to an oncologist at a neighbouring hospital. The oncologist performed a PSA test, which came out normal. However, a prostate sample revealed cancer, prompting the oncologist to establish the probable diagnosis of prostate metastasis to the spine. The patient was taken to a local hospital one week following the chiropractic appointment on suspicion of pneumonia. MRI confirmed the diagnosis of spinal metastasis. The patient's condition progressively deteriorated, and he died three weeks later.

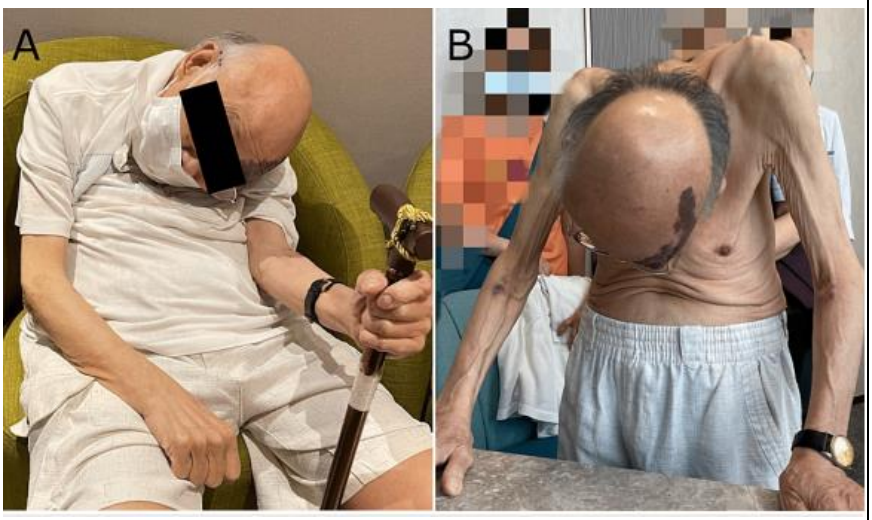

Figure 1. The patient had a chin-on-chest/dropped-head posture in both sitting (A) and standing (B) positions.

Discussion:

When a CT scan revealed many collapsed thoracic vertebral bodies and other metastasis-related abnormalities, the chiropractor immediately sent the patient to an oncologist, who performed a prostate biopsy and diagnosed him with metastatic adenocarcinoma of the prostate. The underlying cause of the patient's lowered head condition was pathologic thoracic fractures and greatly exacerbated kyphosis.[1] In a similar study by Kudo Yoshifumi et al. concluded that thoracolumbar spine deformity is recognised as a likely major source of typical DHS symptoms, and thoracolumbar corrective surgery is an effective means of treating DHS patients with thoracolumbar deformity.[3]

Conclusion:

This is a case report on an older man who presented to a chiropractor with dropped head syndrome and was later found to have prostate cancer metastasis to the spine. Clinicians who handle spinal symptoms should be knowledgeable of dropped head syndrome since it can be brought on by significant pathology, such as a vertebral fracture linked to an underlying cancer. When required, they should also do the appropriate examinations. Upon finding evidence of cancer through imaging or testing, healthcare professionals should quickly refer the patient to an oncologist.

References:

1. Chu EC, Trager RJ, Lee WT, Tam DM, et al. Spinal metastasis causing dropped head syndrome in the chiropractic office: a case report. Cureus. 2023;15(2): e34796.
2. Sciubba DM, Pennington Z, Colman MW, Goodwin CR, et al. Spinal metastases 2021: a review of the current state of the art and future directions. The Spine Journal. 2021;21(9):1414-29.
3. Kudo Y, Toyone T, Okano I, Maruyama H, et al. Dropped Head Syndrome Caused by Thoracolumbar Deformity: A Report of 3 Cases. JBJS Case Connector. 2022; 12(4):e22.00280.

4. Spontaneous basal ganglia hemorrhage with contralateral extension utilizing the canal of Gratiolet- A case report

Introduction:

Stroke is the second most common cause of death and disability worldwide. Twenty percent of all strokes are caused by spontaneous intracerebral haemorrhage (ICH), the most dangerous acute cerebrovascular illness. The basal ganglia are typically the site of spontaneous intracerebral haemorrhage (ICH), which is extremely deadly and incapacitating. After six months of treatment, only 20% of patients are able to return to independent functioning, and within a year, the death rate approaches 60%.[1] In 50% of instances, the most prevalent location for cerebral haemorrhage is the basal ganglia. Although the precise aetiology of these haemorrhages is frequently unclear, hypertensive haemorrhage is the most prevalent cause, followed by alcohol poisoning.[2] This is a case report of spontaneous bilateral BGH and discuss about the anatomy in relation to the imaging pattern of haemorrhage.

Imaging findings provide a fresh perspective on the structure and fibre distribution of the anterior commissure (AC) in a clinical context, and the extension of spontaneous bleeding through the Canal of Gratiolet over the AC has been specifically reported in this case report

Case Presentation:

A 69-year-old woman who had experienced anxiety, hypothyroidism, and at least 32 pack-year of smoking without any relevant family history reported experiencing a rapid onset of headache and lethargy while she was out of the house. When she first arrived at the hospital, her blood pressure was 173/98 mmHg. A physical examination revealed moderate

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encephalopathy, dysarthria, left facial droop, and weakness in her left arm. After obtaining non-contrast head computed tomography (CT), irregular hyperdensities were seen extending into the contralateral globus pallidus and contiguous along the right basal ganglia's anterior commissure (AC). Intraventricular extension into the third ventricle, bilateral foramina of Monro, the fourth ventricle were observed. A CT scan was also performed, however it revealed no signs of a vascular abnormality or aneurysm. Hemosiderin staining and vasogenic edema were seen along the AC's white matter pathway in the magnetic resonance imaging (MRI). Additionally, an MR venogram was obtained without any indication of cerebral venous blockage. A second catheter-based cerebral angiography revealed no evidence of vascular lesions. The patient was hospitalised and kept under observation. The patient was allowed to resume taking medicine at home as their systolic blood pressure objectives were less than 140 mmHg. The work-up revealed no evidence of toxicologic aetiology or coagulopathy. The patient had a prolonged pronator drift and somewhat diminished left upper extremity strength at the time of discharge. Patient showed mild dysmetria of the left upper extremities and a mild strength deficiency in the left elbow function at the time of the previous neurological test, which was performed seven months after the haemorrhage. Seven months later, follow-up imaging showed intraventricular extension and BGH resolution (Figure 1).

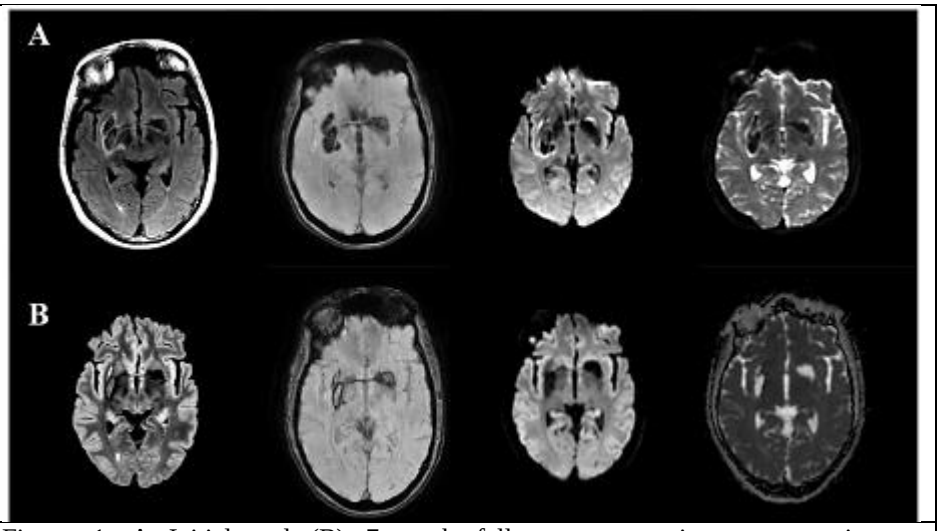

Figure 1. A Initial and (B) 7-month follow-up magnetic resonance images demonstrating the extent of right basal ganglia hemorrhage, contralateral extension and delineation of the AC tracts.

Discussion:

This case report describes a rare instance of spontaneous bilateral BGH, most likely originating in the right basal ganglia and spreading via the Canal of Gratiolet across the anterior commissure to the contralateral basal ganglia. In present case, intraventricular extension of the haemorrhage into the third ventricle, bilateral foramina of Monro and the fourth ventricle where the third ventricle had the highest density of blood components. This

pattern of bleeding indicates that blood products are extended intraventricularly through the anterior commissure's exposed surface at the anterior surface of the third ventricle. The frequency of bilateral BGH with intraventricular extension is unclear since this clinical condition is uncommon.[1] In a previous study by Shaheed TA et al. reported a case of spontaneous bilateral BGH, non-contrast CT and MRI revealed intraventricular extension into the right frontal horn.[3]

Conclusion:

This is the first instance in which the extension of spontaneous haemorrhage through the Canal of Gratiolet over the AC has been specifically described, and imaging results offer a new picture of the architecture and fibre distribution of the AC in a clinical setting. The mechanism behind this uncommon clinical condition may be explained by these results.

References:

1. Shi J, Liu Y, Wei L, Guan W, et al. Admission neutrophil-to-lymphocyte ratio to predict 30-day mortality in severe spontaneous basal ganglia hemorrhage. Frontiers in Neurology. 2023 Jan 10;13: 1062692.
2. Ismail R, George DD, Kohli GS, Khan MW et al. A case of spontaneous basal ganglia hemorrhage with contralateral extension utilizing the canal of Gratiolet. BMC Neurol. 2023; 23(1):190.
3. Shaheed TA, Glover N, Alboiny S. Nontraumatic spontaneous bilateral basal ganglia hemorrhage: a rare case report. Cureus. 2020;12(11): e11299.

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