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Case Study

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A RARE CASE OF RABIES INDUCED ENCEPHALITIS OR VACCINE INDUCED GUILLAIN BARRE SYNDROME – A CASE REPORT

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ABSTRACT

Guillain–Barre syndrome is a rare but fatal autoimmune disease. The most common known etiology of Guillain–Barre syndrome is infectious disease notably caused by Campylobacter jejuni. A very small fraction of people can develop vaccine induced Guillain- Barre Syndrome due to vaccinations like a meningococcal vaccine, poliovirus vaccine, influenza vaccine, and rabies vaccine. Amongst these vaccines, rabies is invariably fatal. However, it can be preventable by early diagnosis and treatment according to the WHO guidelines. Older formulations of rabies vaccines are cultured in the neural tissues and have been found to be associated with an increased risk of Guillain–Barre syndrome. Most of the cases of Guillain–Barre syndrome due to vaccination are either undiagnosed or misdiagnosed. Here, we report a case of vaccine-associated Guillain–Barre syndrome which developed 4 weeks after anti-rabies vaccine which was given after a dog bite in a 39 year old female who presented with lower limb weakness and difficulty in swallowing creating a diagnostic dilemma as a case of paralytic rabies or vaccine induced GBS.

KEYWORDS: Rabies vaccination, Guillain Barre Syndrome, Campylobacter jejuni, Paralytic rabies.

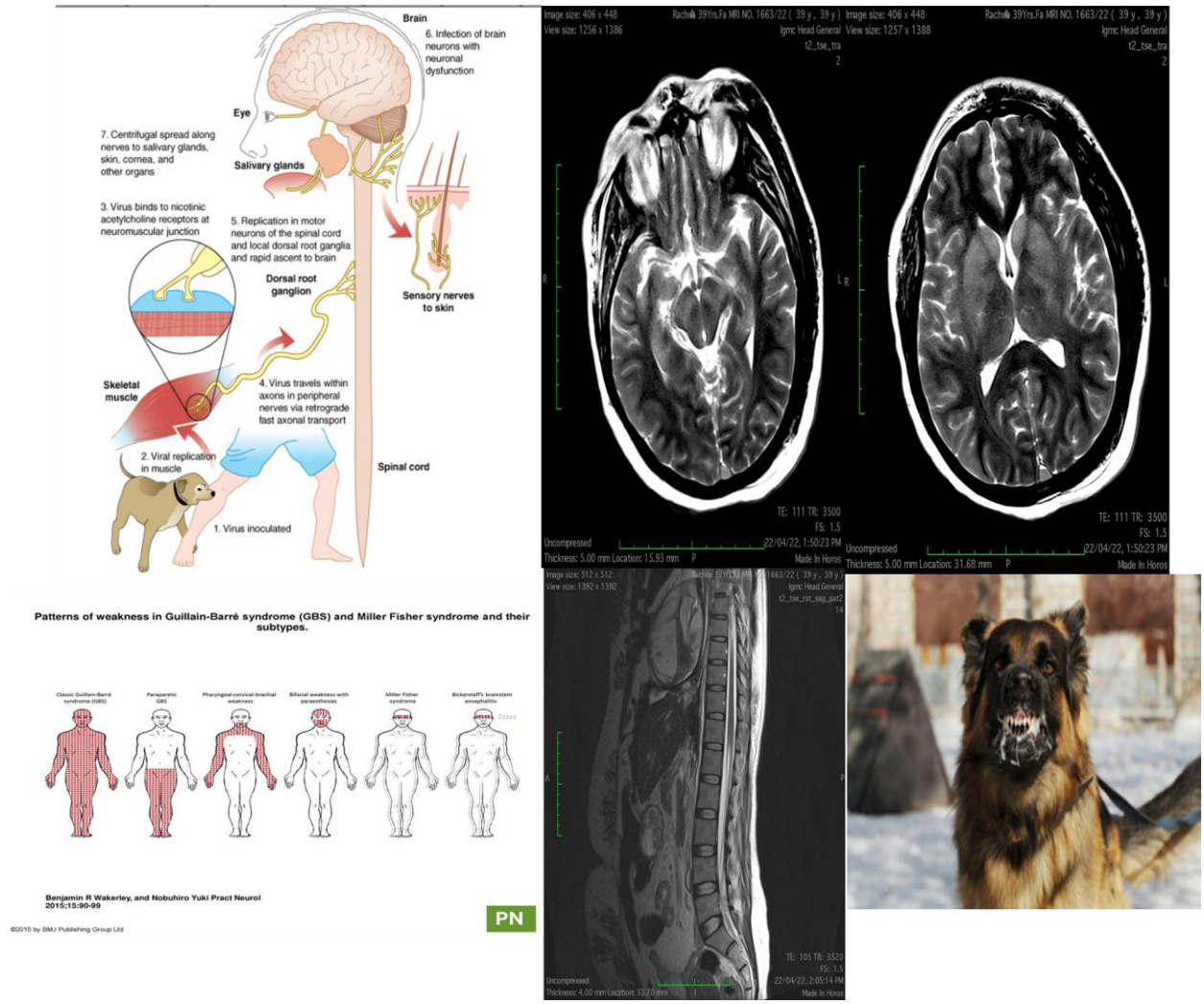
INTRODUCTION

Guillain-Barre syndrome (GBS) can be described as a collection of clinical syndromes that manifests as an acute inflammatory polyradiculoneuropathy with resultant weakness and diminished reflexes. Autoimmunity- molecular mimicry plays a role in its pathogenesis. An immune response triggered by a prior infection cross-reacts with the peripheral nerve components and mistakenly damaging their own myelin sheath.[1] Guillain-Barre syndrome (GBS) is the leading cause of acute flaccid paralysis. The role of vaccination in the development of Guillain-Barre syndrome (GBS) is controversial, although cases of GBS have been reported following a wide range of vaccines.[2] The diagnosis of rabies is not very difficult when associated with the classical features of excitations or phobias. But it can be quite challenging when presented as acute flaccid paralysis. Paralytic rabies presents with prodrome of fever in one-third cases, phobic spasms in half of cases, altered sensorium, ascending paresis and may involve respiratory and bulbar muscles with a rapid devastating course, culminating in death.[3] It is more common in persons who have received postexposure anti-rabies vaccination. Up to two thirds of patients report an antecedent bacterial or viral illness prior to the onset of neurologic symptoms. Vaccine associated Guillain Barre syndrome (GBS) is defined as the onset of GBS within 6 week period after receiving the anti-rabies vaccine.[4]

CASE REPORT

A 39-year-old female presented to the emergency department with a history of fever, progressive upper and limbs weakness and difficulty in swallowing which was sudden in onset. She had no associated symptoms like rash, headache, backache or blurring of vision. No antecedent respiratory tract infection or diarrheal illness. Past medical history was insignificant. About 17 days prior to these symptoms, she received subconjunctival immunoglobulin and anti-rabies vaccine following a dog bite. On physical examination, the patient was not in acute distress. Her vitals were stable. Neurological examination revealed markedly decreased deep tendon and upper limb reflexes. Pupils were bilaterally reactive to light. Other systemic examinations were insignificant. Laboratory investigations showed lymphocytosis with a wbc count of 14,000 while other parameters were within normal limits. Cerebrospinal fluid examination on microscopy revealed pleocytosis with a count of 45,000 cells/mm^3. No micro-organism was detected on gram stain. NCCT head showed a grossly normal study. Patient was started on IV immunoglobulins considering it to be a pharyngeal cervical brachial GBS with pyrexia with an antibiotic coverage of ceftriaxone and metronidazole. 4 days post admission patient started deteriorating further with complete dysphagia and respiratory distress with rapidly falling oxygen saturation post which the patient was intubated. Patient was shifted

to ICU for ventilatory assistance. MRI head done shows hyperintensity involving b/l basal ganglia, substantia nigra, hippocampi, posteromedial part of b/l thalami, dorsal part of mid brain, pons and medulla which suggested that the possibility of rabies encephalitis could not be ruled out. The patient however succumbed to death on the 5th day post admission. Based on the physical examination done earlier, a provisional diagnosis of rabies induced encephalitis or vaccine induced Guillain–Barre syndrome (PCB variant) was made which was left unconfirmed due to patient's death.



DISCUSSION

Guillain–Barre syndrome is a rare but fatal autoimmune disease. characterized by progressive ascending paralysis with varying degrees of weakness, sensory abnormalities and autonomic dysfunction. One of the suggested mechanisms for the development of Guillain–Barre syndrome is molecular mimicry. Despite multiple proposed mechanisms behind the development of Guillain–Barre syndrome, the exact cause of Guillain–Barre syndrome is still unknown, and it may or may not have some triggering factor.[5] Among the triggering factors, vaccines, which include meningococcal vaccine, poliovirus vaccine, influenza vaccine, and rabies vaccine, are reported to be associated with the onset of Guillain–Barre syndrome.[6] The Pharyngeal Cervical Brachial (PCB) variant of GBS is characterised by acute weakness of the oropharyngeal, neck, and shoulder muscles with swallowing dysfunction.[7] The condition involved the bulbar muscles along with upper limbs and spared the lower limbs, so it is called the PCB variant of GBS. Its incidence is approximately 0.07-0.25/100,000. The manifestations are severe bulbar palsy, slowly progressing to facial palsy, weakness of neck flexors and proximal muscles of upper limbs. Acute GBS and its PCB variant share other diagnostic features- including areflexia, raised CSF protein, nerve studies revealing demyelinating disease. Serum anti-GT la IgG antibody has been detected in about 50% of the PCB variant cases of GBS.[8] So far, there has been only two case reports showing isolated acute bulbar palsy due to the PCB variant of GBS. Out of all rabies cases, 20% cases include atypical paralytic rabies, where patients often lack hydrophobia and may present with an acute flaccid quadriparesis mimicking Guillain–Barre syndrome as was seen in our case. Early suspicion and diagnosis of rabies in such cases is essential to avoid treating them as

Guillain–Barre syndrome. Presence of fever along with distal paraesthesia in the limb carrying bite wound at the onset of illness, bladder involvement, early bulbar symptoms, impaired mentation and cerebrospinal fluid pleocytosis can alert the possibility of an infectious disorder rather than Guillain–Barre syndrome. A significantly rapid rise in rabies virus neutralizing antibody titre by Rapid Fluorescence Focus Inhibition Test in cerebrospinal fluid and Serum could have confirmed rabies encephalitis in our case.

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