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Guillain Barre Syndrome.

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Abstract :

Objective to report 2 cases of Guillian barre syndrome with different manifestations

Guillain Barre Syndrome consists of a group of neuropathic conditions characterized by progressive weakness and diminished or absent mitotic reflexes.
The estimated annual incidence in the United States is 1.65 to 1.79 per 100,000 persons.

Guillain Barre syndrome is believed to result from an aberrant immune response that attacks nerve tissue. This response may be triggered by surgery, immunizations, or infections. The most common form of the disease, acute inflammatory demyelinating polyradiculoneuropathy, presents as progressive motor weakness, usually beginning in the legs and advancing proximally. Symptoms typically peak within four weeks, then plateau before resolving.

More than one-half of patients experience severe pain, and about two-thirds have autonomic symptoms, such as cardiac arrhythmias, blood pressure instability, or urinary retention. Advancing symptoms may compromise respiration and vital functions.

Introduction:

Guillian Barre Syndrome GBS is rapidly progressive disorder of Muscle weakness sometimes progressing to complete paralysis , this Syndrome effect approximately only one or two people each year in every 100,000 population . Although its precise cause is unknown .

About half the patient have a gastrointestinal or respiratory infection a few days before the onset , there is strong evidence that the immune response to the infection produces autoimmune response which damage the nerve causing weakness and loss of sensation . (1)

In milder disease the damage only effect the nerve sheath and blockage the passage of nerve impulses . this can recover in a few weeks .

in more severe disease the autoimmune response damage and causing delay in recovery so may develop to more permanent weakness.(2)

to diagnosis of GBS is only one of many causes of acute weakness and numbness.

The diagnosis requires recognition of the characteristic symptoms and signs. Because it is rare this may be difficult for the non-specialist and referral to a neurologist is usually appropriate. Clinical examination shows loss of the tendon reflexes and supports the diagnosis of peripheral nerve disease. Two tests are commonly used to support the diagnosis:

Lumbar puncture to examine the fluid which bathes the spinal cord and nerve roots CSF: nerve conduction tests often called EMG, short for electromyogram which is to say recording of muscle activity .(2)

The doctor needs to exclude many other causes of neuropathy such as alcohol poisons, drugs, vasculitis and cancer.

Treatment for Guillain Barre Syndrome can help reduce the symptoms and speed up recovery**.** most people are treated in hospital and usually need to stay in hospital for a few weeks to a few months .

The main treatments are: intravenous immunoglobulin : the most commonly used treatment for Guillain-Barré Syndrome is intravenous immunoglobulin When you have Guillain Barre Syndrome the immune system produces harmful antibodies that attack the nerves. intravenous immunoglobulin is a treatment made from donated blood that contains healthy antibodies these are given to help stop the harmful antibodies damaging your nerves is given directly into a vein most people need treatment once a day for around five days.(2)

Plasmapheresis: is sometimes used instead of Intravenous immunoglobulin, this involves being attached to a machine that removes blood from a vein and filters out the harmful antibodies that are attacking your nerves before returning the blood to your body most people need treatment every other day for a week or two.(3)

other treatments while in hospital you'll be closely monitored to check for any problems with your lungs, heart or other body functions given treatment to relieve your symptoms and reduce the risk of further problems this may include: ventilator if you are having difficulty breathing , a feeding tube if you have swallowing problems , painkillers if you are in pain , being gently moved around on a regular basis to avoid bed sores and keep your joints healthy , catheter in your urethra if you have difficulty peeing , laxatives if you have constipation.[2]

In this report I would talk about Guillian Barre Syndrome its epidemiology and compare between two cases of GBS with different presentation.

Material and methods :

Case 1 : 11-year-old boy was referred to the emergency department with complaints of generalized body pain and gate problems. He revealed a history of upper respiratory tract infection in the preceding two weeks and mentioned generalized body pain three days prior to admission and gate problems from the day before no past medical history or family history were mentioned In a systemic examination his vital signs were stable and generalized muscle tenderness existed.

Other systemic examinations were normal :neurological examination showed normal mental status, cranial nerve, and sensory examination , the deep tendon reflex in upper limbs and lower limbs were diminished (+1) and the proximal and distal muscle force in the upper and lower limbs were 4/5 he could not walk due to severe pain the patient mentioned pain as chief complaint the clinicians performed 2 electromyography and nerve conduction velocity tests to ensure the diagnosis our laboratory results are as follows:

WBC: 9000 Lymphocyte: 60% Hb: 11 CRP: +1 RF :negative Wright Test: negative ESR: 16 LDH: 290 Repeated LDH: 300.

CSF : exam: WBC:0 ,RBC:0, Protein:55,Glucose:80 concomitant blood sugar:100

Lumbosacral MRI: normal , Technetium bone scan: normal

EMG NCV: Diagnostic for Guillain Barre (Demyelinating polyneuropathy)

Repeated EMG NCV: Diagnostic for Guillain Barre Syndrome.

From the laboratory results and positive EMG NCV tests, Guillain Barre was diagnosed and he was treated with standard drugs and released after 10 days.

In a follow up, he mentioned no weakness, he could walk, and his pain was decreased.[4]

Case 2:

A 48-year-old man presented to the accident and emergency department complaining of acute abdominal pain which was characterized by exacerbations and remissions, and nausea. The patient was able bodied. he was not a smoker neither mentioned any previous health problems on examination the chest was clear, but the lower half of his abdomen was tender to deep palpation the patient did not have a neurologic examination upon arrival to the emergency room since the primary focus at the time was his acute abdominal pain, Blood pressure was 140/90 mmHg, and heart and respiratory rate were 95 and 16/min, respectively. Laboratory tests showed a mild leukocytosis with polymorphonuclear type. Erythrocyte sedimentation rate and C-reactive protein were within normal limits. Blood glucose, renal and liver function tests, and urine examination were normal. Blood and urine amylase were negative for pathological findings. Electrocardiography, chest x-ray, ultrasound and computed tomography (CT) of the abdomen were normal.

The CT angiogram that was performed to exclude a partition aneurysm was also normal. The patient was admitted to the surgical department for further investigation of his abdominal pain.

On the day after admission, the patient developed weakness and inability to walk and stand on clinical examination, he looked frail, had tachypnea and was heavily perspiring his consciousness level was excellent the upper extremities were normal, but there was numbness and severe weakness in both lower extremities while tendon reflexes were not present , the patient was unable to walk unaided.

When asked about any recent infections, he stated that ten days before he had an gastroenteritis, and fever up to 38.5°C that lasted 48 hours.

The head CT was normal, and the lumbar puncture was negative for the presence of either protein (35 mg/dl) or cells (3/mm3) The likelihood of GBS was raised.

The patient was urgently transferred to the neurology department of a tertiary hospital in Athens . During the transport, he presented weakness of both upper extremities and urinary retention. within a few hours after his arrival, he developed acute breathing difficulty, was intubated , and required mechanical ventilation for two weeks. the further diagnostic tests (repeat lumbar puncture, nerve conduction studies, electromyography) performed corroborated the diagnosis of GBS.

acute axonal neuropathy; consistent with the rapid clinical deterioration of the patient. the patient was treated with a combination of plasma exchange followed by a course of intravenous immune globulin .

During his hospitalization, he had a tracheotomy and developed tetraplegia. he was discharged one month later and had to remain in a rehabilitation center for four months.[3]

Case 1 was performed in the pediatric hospital and we assist the clinical manifestations and result of electro diagnostic test , functional status , treatment instituted and outcome of this treatment .[4]

Case 2 was performed in medical department clinical and laboratory finding had final diagnosis of GBS electromyogram and CSF analysis recorded .[3]

Result :

The 2 cases are diagnosed as Guillian Barre Syndrome but has different causes and different clinical presentation.

Discussion:

One proposed mechanism for Guillian Barre Syndrome is that an antecedent infection evokes an immune response, which in turn cross reacts with peripheral nerve components because of the sharing of cross reactive epitopes (molecular mimicry) . The end result is an acute polyneuropathy, this immune response can be directed towards the myelin or the axon of peripheral nerve . [2]
Immune reactions directed against epitopes in Schwann cell surface membrane or myelin can cause acute inflammatory demyelinating neuropathy ,the pathology is that of multifocal inflammatory demyelination starting at the level of the nerve roots , The earliest changes are frequently seen at the nodes of Ranvier , both the cellular and humeral immune responses participate in the process.[5]

Invasion by activated T-cells is followed by macrophage-mediated demyelination with evidence of complement and immunoglobulin deposition on myelin and Schwann cells ,no specific myelin antigens have been identified.[2]

although, in some of these investigations pain was indicated as a GBS feature, It should be mentioned that in these cases, patients who encountered pain that was accompanied by other frequent symptoms of GBS and Guillain Barre syndrome pain involvement had not been considered as a major clinical manifestation for diagnostic criteria among children, which differs with our study. Our study shows no other symptoms except pain.[6]

myalgia had not been considered a chief and common complaint of GBS, often patients report weakness with or without myalgia ,however results showed that if myalgias were the chief complaint and weakness was mentioned as a less important symptom, clinicians should consider GBS after ruling out other reasons for myalgia especially inflammatory myositis , a thorough assessment of effective factors that can treat weakness and gate disability sooner and preserve patient life better is of clinical significance. [6]

Conclusion:

Guillain Barre Syndrome is a neurological disorder resulting primarily in muscle paralysis that in most cases is symmetrical.

Patients may have mild or severe involvement which may in a small percentage lead to death, patients may present with history of upper respiratory tract infection or gastrointestinal infection preceding the disease which cause neuronal sheath damage leading to loss of sensation and weakness of affected site.

Future work :

Maybe to know or identify Guillian Barre Syndrome effect which antigen to prevent this disease from occurring.

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