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CASE REPORT

Guillain Barré Syndrome after Thrombolysis With Streptokinase for Acute Myocardial Infarction: A Case Report

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Abstract:

We would like to report on a patient, a 52-year-old man with acute neurologic disorder, Guillain Barré Syndrome. He was successfully treated by intravenous immunoglobulin. The patient suffered from acute extensive anterior MI. 2 weeks after thrombolytic therapy with streptokinase, he developed GBS.

Key Words: *Guillain Barré Syndrome, Myocardial Infarction, Streptokinase etc.*

Introduction:

Guillain Barré Syndrome is an acute immune mediated peripheral neuropathy. It is a rapidly evolving polyradiculoneuropathy preceded by a triggering event, most often respiratory or gastrointestinal illness. There are reports of some cases of GBS after intravenous streptokinase administration[1-4]. In 1983, journal of the American Medical Association published an article "Possible association of Guillain Barré Syndrome with thrombolytic therapy". In 1992, British Medical Journal also reported "Guillain Barré syndrome after treatment with streptokinase". International Journal of Cardiology reported a case titled "Guillain Barré Syndrome after myocardial infarction" in the year 2003. Here we will describe about a patient of GBS who was admitted in BSMMU, Dhaka.

Case Report:

The patient was a 52 year old man. On 19th January, 2016, he had severe central chest pain for 1 hour, sweating and nausea but no vomiting. He was taken to a tertiary level hospital where ECG was done and it revealed evidence of extensive anterior myocardial infarction. After initial resuscitation, he was sent to a specialized hospital, National Institute of Cardiovascular Disease (NICVD) for better management. Troponin I was positive and CK-MB level was significantly elevated. On that day (19th January), the patient received streptokinase, gradually clinical and biochemical and other investigations revealed signs of improvement.

On 24th January, 2016, he was discharged from NICVD and advised for coronary angiogram. On 29th January, 2016 he was admitted in Mirpur Heart Foundation Hospital. Two days later, coronary angiogram was done and it revealed Triple Vessel Disease (TVD). He was advised for reperfusion therapy in the form of PCI or CABG. But on 2nd February, he experienced a new problem; distal paresthesia in his arms and legs. There was progressive weakness of both of his lower limbs

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and later in an ascending manner, it involved his upper limbs also. Clinically muscle power was MRC grade 0, both in upper and lower limbs, proximal and distal. There was areflexia and bilateral facial palsy. Sensory function was intact and no sphincter disturbance was present. Nerve conduction study was done. It revealed demyelinating sensory motor polyneuropathy compatible with AIDP. At the same time, there was difficulty in breathing. He was shifted to ICU. After resuscitation, he was improved.

There was no history of drug or toxin exposure. There was no upper respiratory or gastrointestinal infection within the last 2 months. No evidence of arterial or venous embolism, vasculitis and connective tissue disease. He was a diagnosed case of DM and was on medication.

The patient received 5 doses of IVIg (30 gm). His condition gradually improved.

Discussion:

The precise cause of GBS is not yet known, but it has been reported to be associated with history of gastrointestinal or upper respiratory infection. Underlying mechanism is probably immunological. Clinical symptoms are thought to result from streptokinase antibody complex mediated damage to the local blood nerve barrier

Streptoknase is a single chain polypeptide derived from group C beta hemolytic streptococci. The protein nature of this drug makes it antigenic in the body. It stimulates immunologic reactions[6]. This is probably the pathophysiologic basis.

There are also reports of GBS after myocardial infarction in patients without thrombolytic therapy[7]. High creatine kinase from significant muscle injury might be a possible immunological precipitant. Therefore it is not yet determined whether process of MI itself is the promoter of polyneuropathy or thrombolytic agents are the initiators of this conditions[8].

In developing conuntries and in some developed conuntries, there are institutions who lack interventional capability (ie PCI & CABG). For acute ST-elevation MI, they use fibrinolytic therapy. GBS after acute MI treated with reteplase has also been reported[9].

GBS can be seen in the late course of acute MI by the process of triggered autoimmune mechanism. A patient developing paresthesia and progressive muscular weakness after 10-30 days of myocardial infarction, especially if thrombolysed with streptokinase, high degree of suspicion of GBS could be considered. At the same time, other likely causes must be excluded. To be epidemiologically significant, many other reports around the globe should be analyzed.

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