Guillain-Barré Syndrome after Streptokinase Therapy for Acute Myocardial Infarction

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Abstract

Some drugs including streptokinase have been reported to precipitate Guillain-Barré syndrome. We report a 70-year-old man with acute anterior myocardial infarction who developed Guillain-Barré syndrome seven days after thrombolytic therapy with streptokinase.

Key words: Guillain-Barré syndrome, myocardial infarction, streptokinase

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Introduction

Guillain-Barré syndrome is an acute immune-mediated polyneuropathy caused by infection, inflammation and surgery (1). Some medications including streptokinase have been reported to precipitate Guillain-Barré syndrome. Here, we report a case of Guillain-Barré syndrome seven days after receiving streptokinase for acute myocardial infarction (MI).

Case Report

The patient was a 70-year-old obese man presented to our emergency room with chest pain radiating to the left shoulder and accompanied with dyspnea. Electrocardiogram showed ST elevation in V₂-V₄ precordial leads. Troponin-I was positive and creatine kinase-MB level was elevated to 5.4 ng/mL. The patient received 1.500000 units of streptokinase and was discharged, in good general condition 5 days after CCU admission.

Seven days after discharge, he returned again complaining of general lethargy and the sensation of pins and needles in his hands and feet from the previous day. The patient then developed progressive muscle weakness of his lower extremities as well as distal sensory impairment. On physical examination the muscle power of extremities was decreased and the patient had areflexia. The patient was conscious and

alert and fundoscopy was normal. Blood cell counts and results of biochemistry tests were within the normal range. Brain computed tomography was normal. The patient was admitted in the intensive care unit (ICU) with clinical suspicion of Guillain-Barré syndrome. Cerebrospinal fluid (CSF) analysis revealed a cell count 4 cells/mm³, protein 90 mg/dL and CSF-to-serum glucose ratio of 0.65. The patient then developed respiratory failure and was intubated and connected to a ventilator. Nerve conduction velocity showed these findings: 1. Absent bilateral H. reflex and low frequency F. waves, 2. Absent sensory nerve action potential (SNAP) of bilateral median, ulnar and superficial peroneal (SPN) nerves, and 3. Absent nerve conduction velocity of deep peroneal (DPN) and ulnar nerves. These findings were in favor of acute generalized peripheral sensory motor polyneuropathy. The patient was treated with plasmapheresis every other day for 5 times. He also received 5 doses of intravenous immunoglobulin (IVIG). His condition gradually improved and one month later he was discharged from hospital with muscle power returning to 3 of 5 in all extremities.

Discussion

The precise cause of Guillain-Barré syndrome is not yet known but it has been reported to be associated with viral infections, lupus erythematosus, lymphoma, Hodgkin's disease and other situations (1) and the underlying mechanism

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Table 1. Reports of Guillain Barré Syndrome after Streptokinase Therapy

Paper	Number of Patients	Cause for thrombolytic Therapy	Year
Eden KV (2)	1	Axillary vein thrombosis	1983
Leaf DA et al. (3)	1	MI	1984
Cicale MJ (4)	1	Pulmonary embolism	1987
Roquer J et al. (5)	1	MI	1990
Barnes D et al. (6)	1	MI	1992
Ancillo P et al. (7)	1	MI	1994
Taylor VT et al. (8)	2	MI/ Pulmonary embolism	1995

is probably immunological.

Here, we report a case of anterior MI that developed Guillain Barré syndrome seven days after receiving streptokinase. Although this is not the first case, streptokinase and other thrombolytic agents are rarely reported in the literature as precipitants of Guillain-Barré syndrome (Table 1). There are also reports of Guillain-Barré syndrome after MI in patients without thrombolytic therapy (9, 10).

Therefore, it is not well determined whether the process of MI itself is the promoter of poly neuropathy or thrombolytic agents are initiators of this condition.

Streptokinase is a single chain polypeptide extracted from beta hemolytic streptococci. The protein nature of this drug makes it antigenic in the body and thus can stimulate immunologic reactions (11). This is probably the pathophysiologic basis in the development of Guillain-Barré syndrome after streptokinase therapy. Kaiser et al found that serum titers of immunoglobulin were elevated 64-fold for IgG, 16-fold for IgM, and 4-fold for IgA compared to controls after streptokinase therapy (12). Others have argued against an association of Guillain-Barré syndrome and streptokinase therapy based on its low incidence (13).

Although streptokinase has been abrogated in a considerable number of countries, it is still routinely used in many developing countries. Therefore this rare condition should be kept in mind as one of the complications of streptokinase.

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