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Acute Transverse Myelitis Complicated in Korean Hemorrhagic Fever: A Case Report and Review of the Literature

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Involvement of the central nervous system in Korean hemorrhagic fever is expected. When such involvement does occur, it is usually in the form of cerebral hemorrhage or pituitary necrosis.

Paralytic disease due to Korean hemorrhagic fever is exceptional. A case of transverse myelitis in an adult female, in which a serologic test of immunofluorescent antibodies to Hantaan virus was positive with clinical pictures of Korean hemorrhagic fever, is reported here.

Key Words: *Acute transverse myelitis, Korean hemorrhagic fever*

INTRODUCTION

Korean hemorrhagic fever is an acute febrile illness characterized by renal failure and hemorrhagic diathesis. This disease is caused by an agent, designated Hantaan virus, and intraspecific transmission of the virus was experimentally demonstrated in the striped field mouse, Apodemus Agrarius Coreae.[1,2] The hemorrhagic diathesis occurs on skin, bulbar conjunctiva, soft palate, pharynx, heart, kidney, adrenal gland, gastrointestinal tract and central nervous system.

The purpose of this paper is to report on an unusual neurologic complication of Korean hemorrhagic fever with illustrations of recently observed findings of acute transverse myelitis associated with Korean hemorrhagic fever.

REPORT OF A CASE

A 31-year-old housewife experienced an abrupt development of chills, high fever and generalized myalgia for three days. For following two days she complained of frequent nausea and vomiting, abdominal pain and generalized malaise. On the fourth day, she was unable to void. Catheterization released 900cc of urine from the bladder and a catheter was maintained in the bladder. Over this period she also suffered from severe weakness on both legs with paresthesias of numbness and stiffness of neck, and she was hospitalized. On the following day there was a rapidly progressive decrease in urine volume and she was transferred to us from the neurosurgical department. The past and family histories were not remarkable.

At hospitalization, she was irritable but clear conscious with a pulse of 84/min; blood pressure, 120/90 mmHg; and oral temperature, 36.5C. There was intense reddening with petechial hemorrhages in the pharynx and palate. Diffuse abdominal and costovertebral-angle tenderness was elicitated. Neurologic examination revealed marked symmetric motor weakness of both lower limbs and decreased deep tendon reflexes without pathologic reflexes. Sensation was diminished below the level of L_2, but pain and temperature senses were present. Cranial nerve function and motor and sensory findings in the upper limbs were normal. By the following day the weakness had progressed to a flaccid paraparesis of both lower limbs and she was unable to support herself in a standing position. At that time she con-

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tinued to complain of nausea, vomiting and abdominal pain. Laboratory values included hemoglobin 12gm/dl; hematocrit, 38%; leukocyte count, 13,450/mm³ (55% polymorphonuclear cells, 11% band forms, 21% lymphocytes, 11% monocytes, 1% eosinophils and 1% basophils); ESR, 58mm/hr; platelet count, 222,000/mm³; bleeding time, 1'00"; coagulation time, 9'00"; and prothrombin time, 12.5 sec(100%). The urea nitrogen was 31.4mg/dl; creatinine, 1.4mg/dl; GOT, 44IU and GPT, 30IU. The urine gave a + + test for protein; the sediment contained many red cells and 5 to 12 white cells. Radiographs of spine and lumbar myelogram, performed on the day of admission revealed no abnormality. A serologic test for immunofluorescent antibodies to Hantaan virus of Korean hemorrhagic fever, done on the seventh day, gave + + +, a titer of approximately 100 and was significantly elevated.

A gastrofiberscopy,[3] performed on the seventh day, showed mucosal hemorrhage spread densely on the entire gastric mucosa (Fig. 1). On the twelfth day, a follow up study showed almost complete disappearance of mucosal hemorrhage.

The nerve conduction study and electromyogram were performed on the fifth day; The motor conduction in peroneal and tibial nerve of both legs showed diminished amplitude, prolonged distal latency of action potential and normal velocity. The sensory conduction showed no abnormalities. The electromyography showed increased insertional ac-

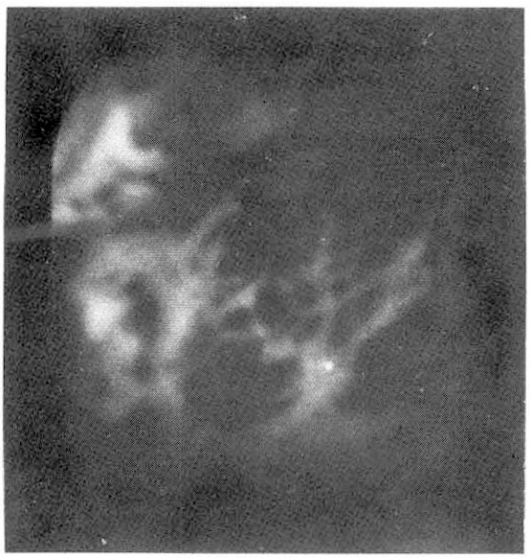

Fig. 1. *Hemorrhagic gastritis with bleeding, gastroscopic finding.*

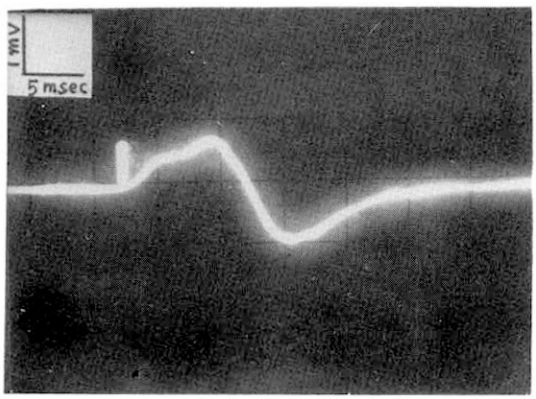

Fig. 2. *Distal motor action potential, left peroneal nerve.*

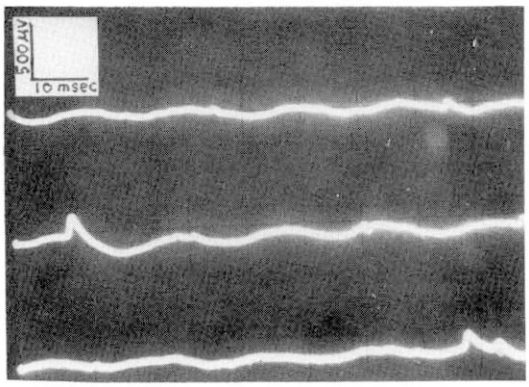

Fig. 3. *Positive sharp wave, right Tibialis anterior.*

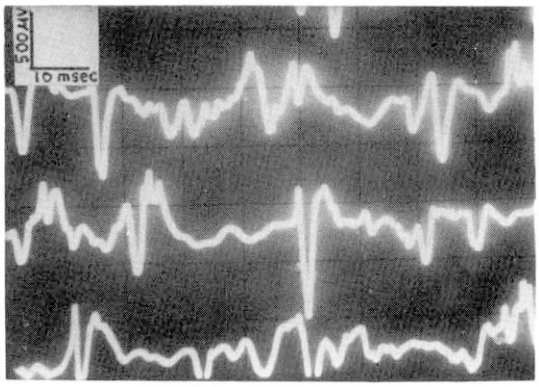

Fig. 4. *Polyphasic potential and motor unit, lateral head of left Gastrocnemius.*

tivities, on volition positive sharp wave at rest in right tibialis anterior and left biceps femoris; polyphasic potentials (30%) with slight decreased number of motor units in most muscles of both legs (Fig. 2-4).

She had no specific treatment except conservative management. On the tenth hospital day, her condition began to improve gradually. She required a urinary catheter for relief of urinary retention (1,000 to 1,500 cc) for two weeks, but at the end of this time she regained control of her bladder. She was able to return to home three weeks after admission. At that time, examination revealed slight weakness and wasting of all muscle groups in the legs, but she was able to walk with support.

When seen four months after discharge, her recovery was virtually complete.

She walked in a normal fashion and the bladder function was normal.

COMMENT

Acute transverse myelitis refers to a syndrome of acutely developing intramedullary spinal cord dysfunction, either ascending or static, usually transversely involving function of both halves of the cord, appearing at any age, without any prior history of neurologic symptoms and includes paralysis and a variable sensory loss.

Present day this syndrome is the subject of controversy in the literatures and confusion still exists on many points, among etiology, pathogenesis, clinical course and prognosis.[4,5]

Transverse myelitis has been described in association with many etiologic factors.[4,5] The possible relationship of acute transverse myelitis to many viral or bacterial diseases as primarily, an infectious process has been suggested by several authors. Demyelinating process, neoplasm and metabolic disease have all been implicated. Acute vascular or ischemic lesions of the spinal cord may cause these symptoms. Spinal artery embolism is probably a common cause. Toxic reaction to drugs such as arsenicals and sulfonamides, and reaction to contrast media have also reported. Other allergic or autoimmune predisposing factors have also been suggested. But majority of myelitis have idiopathic etiology.

In many of infectious myelitis, it has been observed that major pathologic change occurred in mesodermal structures (blood vessels and meninges) but rarely the cord proper.[6] Infectious microorganisms may produce lesions of the cord indirectly by invading the vessel walls and producing vascular obstruction and circulatory interference with ultimate destruction of the substance of the cord. It is partly, of course, a philosophical question whether it is, in fact, due to the action of an infectious agent on the spinal cord itself, via an undefined hypersensitivity reaction, or by induced vascular lesions, possibly in turn also associated with sensitivity. It has been examined pathologically that nonspecific necrosis affects the gray and white matter indiscriminatly, and is not confined to the area of blood supply of any particular artery and destroying axones and cell bodies as well as myelin in most of idiopathic acute transverse myelitis.

It is well known that nerve conduction studies and electromyographic findings can be of great value in confirming the diagnosis and identifying the segment of diseased nerve with damaged myelin sheath or axonal degeneration.[7] It is possible that severe involvement of the legs and bladder in this case with characteristic findings in the nerve conduction and electromyographic studies were evidence of acute transverse myelitis.

Epidemic hemorrhagic fever was recognized for the first time in Korea in 1951 during the Korean War among United Nations troops and since that time it has been known as Korean hemorrhagic fever. In 1976 Lee and Lee sucessfully demonstrated an antigen in the lungs of Apodemus Agrarius Coreae and very recently, have perfected a serologic test[8] for diagnosis of Korean hemorrhagic fever. The major pathologic change is wide spread capillary and endothelial damage which result in dilatation of all small vessels in tissue, congestion, plasma transudation, and multiple small hemorrhage especially in the renal medulla, right atrium and gastrointestinal submucosa. A peculiar type of necrosis of the renal pyramids, anterior lobe of the pituitary body, and adrenal gland and a mononuclear cellular infiltration of the myocardium, spleen and liver.

The history and follow-up of this case was consistent with acute transverse myelitis. Compressive spinal cord lesions were ruled out by myelography and manometric studies of the spinal fluid. Based on the clinical data in this case, Korean hemorrhagic fever is thought to play a causative role. The onset of paralysis and sensory loss was associated with Korean hemorrhagic fever and eventually recovered. Without direct proof, it is once again suggested on clinical grounds that either direct invasion of the spinal cord or some form of allergic response to a coexisted Korean hemorrhagic fever may play an etiologic role in this case.

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