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Reversible Dysfunction of the Sinus Node during Acute Phase of Aseptic Meningitis

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ABSTRACT : A fifty-two-year-old man who had reversible, severe dysfunction of the sinus node during acute phase of aseptic meningitis is reported. A diagnosis of aseptic meningitis was made on the basis of following clinical findings ; moderately high fever, meningeal signs and abnormalities of cerebrospinal fluid such as high pressure, an increase in white cell count, high protein content and negative culture of the fluid for bacteria, mycobacteria and fungi. During the acute phase of aseptic meningitis the patient developed marked sinus brabycardia with sinoatrial block and sinus arrest causing dizziness. The sinus rate began to increase gradually and sinoatrial block and sinus arrest disappeared as the clinical manifestations of aseptic meningitis subsided. The sinus rate was significantly raised by intravenous administration of atropine (1.0 mg). During the recovery phase of aseptic meningitis with normal pressure of cerebrospinal fluid, overdrive suppression test revealed normal values of the sinus node recovery time and the patient was in normal sinus rhythm at a rate of 60 to 70 beats/min. It is assumed that increase in the vagal tone resulted from a high intracranial pressure during the acute phase of aseptic meningitis may play an important role in the genesis of severe sinus node dysfunction in this patient.

INTRODUCTION

Many electrocardiographic (ECG) abnormalities such as abnormal Q waves, abnormalities of ST segments and T waves, prominent U waves and prolonged QT intervals have been described in patients with central nervous system diseases including cerebrovascular accident, intracranial space occupying lesion, head trauma, and meningitis, etc[1~5]. Atrial fibrillation and sinus bradycardia have also been reported in such patients[7]. However, severe dysfunction of the sinus node has not been reported in patients with meningitis. This report describes a man who had reversible, severe dysfunction of the sinus node with frequent emergences of long sinus pause accompanied by dizziness during acute phase of aseptic meningitis.

CASE REPORT

A fifty-two-year-old man was admitted to the hospital because of moderately high fever, severe headache and nausea. A routine examination three years before admission showed no abnormalities including cardiac arrhythmias. He was well until two weeks earlier, when he began to notice sore throat, nasal discharge and mild headache. During the one week before admission he experienced moderately high fever with the temperature ranged from 37.5 to 38.2 ℃, increasing headache and nausea.

On admission, the patient appeared moderately ill and sweaty. Pulse was regular but slow

at a rate of 44 beats/min. Blood pressure was 152/100 mmHg. The temperature was 37.8°C. The heart was not enlarged. A grade 2 systolic ejection murmur was heard along the lower left sternal border. The lungs were clear. Liver and spleen was not felt. There was no peripheral edema. The meningeal signs such as nuchal rigidity and Kernig's sign were observed. Other neurological examinations did not disclose significantly abnormal findings.

The urine was normal. The hematocrit was 44.7 per cent; the white-cell count was 10600, with 69 per cent neutrophils, 22 per cent lymphocytes, and 9 per cent monocytes. The urea nitrogen was 13 mg, the creatinine 1.1 mg, the bilirubin 1.0 mg, the calcium 4.7 mg, and the protein 7.1 mg per 100 ml. The sodium was 145 mEq, the potassium 3.7 mEq, the chloride 101 mEq per liter. The serum GOT was 10 U, the lactic dehydrogenase (LDH) 279 U, and the alkaline phosphatase 4.9 U per liter. A lumber puncture yielded clear, slightly yellowish cerebrospinal fluid under a high pressure of 230 mm of water; the fluid contained 347 white cells, mainly lymphocytes, per cubic millimeter; the protein was 75 mg, and the glucose 55 mg per 100 ml; a serologic test for syphilis and microscopical examination of a stained smears and cultures of the fluid for bacteria, mycobacteria and fungi were negative. The computed tomographic scan of the brain showed no significant abnormalities. X ray films of the chest

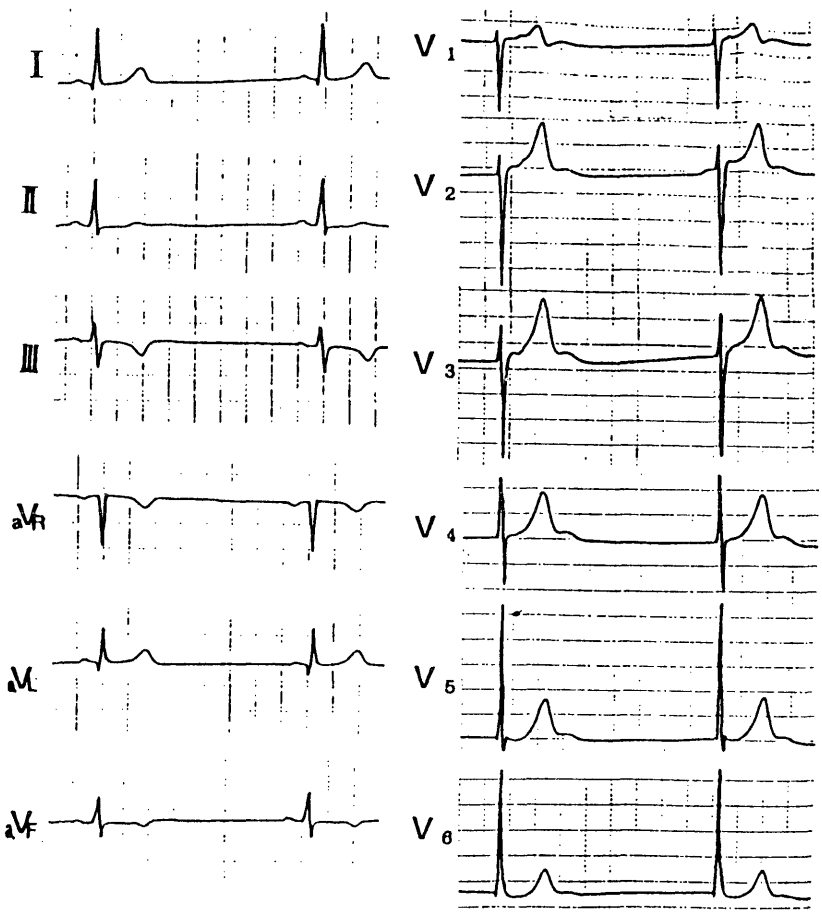

Fig. 1. A twelve lead electrocardiogram recorded on admission showing a sinus bradycardia with A-V junctional escape beats at a rate of 35 beats/min and T wave inversion in the leads III and aVF and tall U waves in the chest leads. QTc is 0.38 sec.

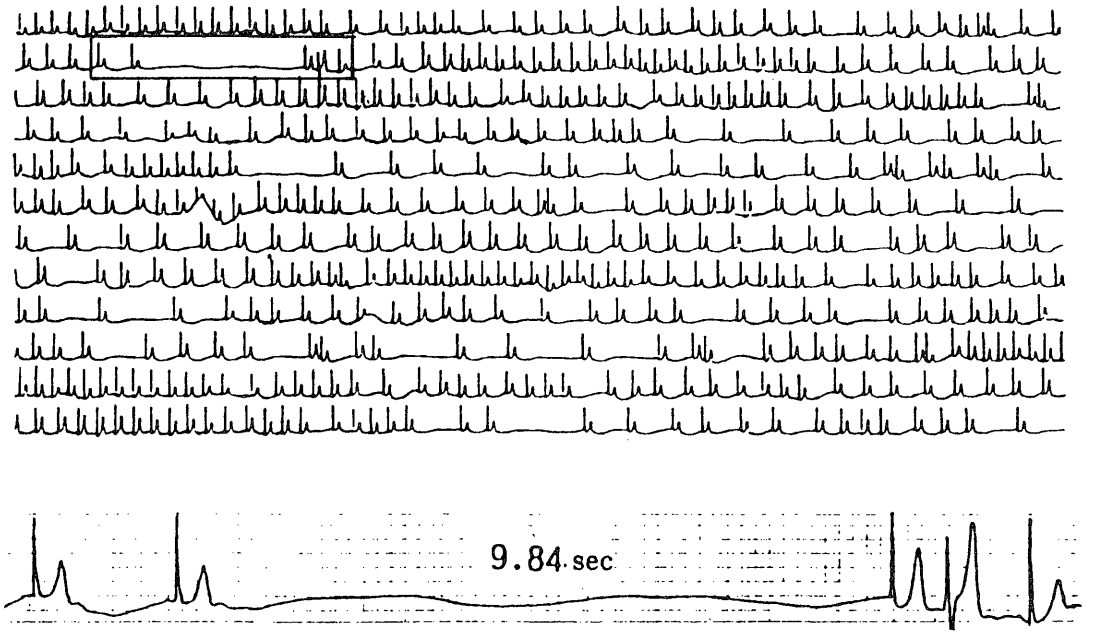

Fig. 2. Top: A twelve minute tracing (0:40 pm to 0:52 pm) from a Holter electrocardiographic monitoring over 24 hour period, performed on the 8th hospital day, showing frequent emergences of long sinus pause. Bottom: This tracing is identical with a boxed part of the twelve minute tracing and shows a long sinus pause of 9.84 sec.

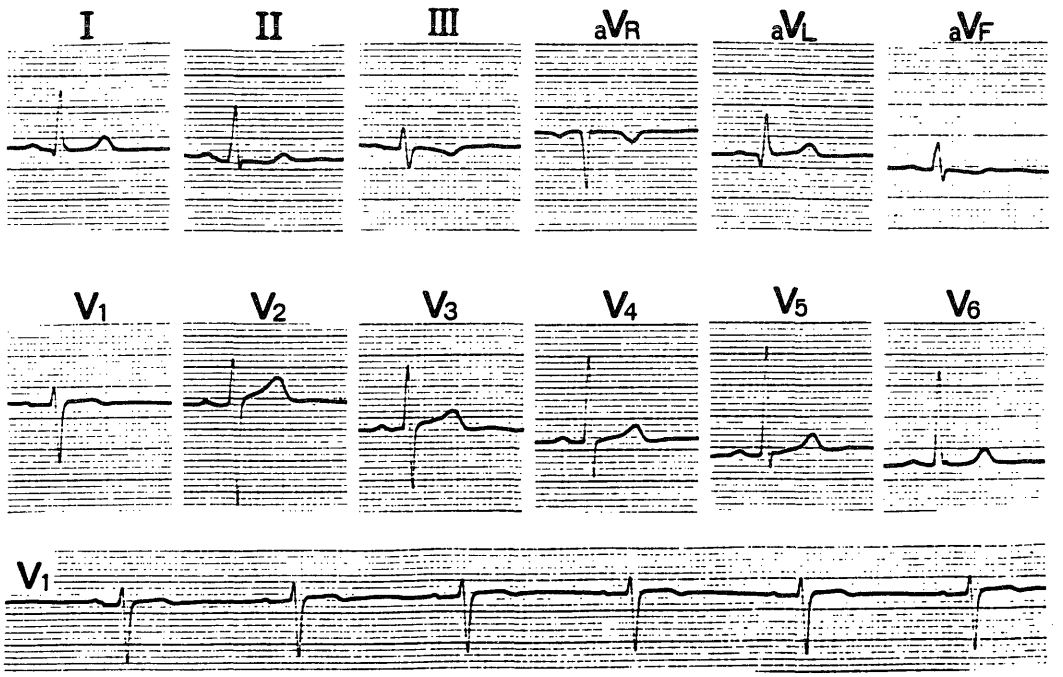

Fig. 3. A twelve lead electrocardiogram recorded on the 50th hospital day showing a normal sinus rhythm at a rate of 60 beats/min. T wave inversion in the leads III and aVF is still observed, but tall U waves are not seen. QTc is 0.36 sec.

showed no abnormalities with a cardiothoracic ratio of 48 %. Echocardiogram disclosed no apparent structural heart disease. On the basis of these clinical findings a diagnosis of aseptic meningitis was made. A conventional therapy for aseptic meningitis was started. As shown in **Figure 1**, a twelve lead electrocardiogram, recorded on admission, showed a sinus bradycardia with frequent emergences of the A-V junctional escape beat at a rate of 35 beats/min. T wave inversion in the leads III and aVF and tall U waves in the chest leads were seen. QTc was 0.38 sec. During the next 10 days following admission the heart rate remained slow and all the electrocardiograms, recorded every day, showed marked sinus bradycardia with frequent emergences of the A-V junctional escape beat. During this period heart rate ranged from 35 to 40 beats/min. A Holter ECG monitoring over 24 hour period, performed on the 8th hospital day, demonstrated marked sinus bradycardia with sinoatrial block and sinus arrest resulting in emergence of the A-V junctional escape beat during the entire period of monitoring. As shown in **Figure 2**, long sinus pause occurred frequently during the entire period of monitoring. The longest sinus pause of 9.84 seconds was accompanied by dizziness. Thereafter, the heart rate began to increase gradually and sinus rhythm with slightly slow rate ranging from 50 to 55 beats/min resumed by the 19th hospital day, when the fever, meningeal signs and leukocytosis had already subsided. At this time, intravenous injection of atropine (1.0 mg) raised sinus rate from 54 to 86 beats/min. A Holter ECG monitoring over 24-hour period, performed on the 35th hospital day, revealed no apparent sinus bradycardia and no emergence of the sinus pause. Overdrive suppression test, performed on the 36th hospital day, revealed normal values of the sinus node recovery time with the longest one of 1.72 seconds. A lumbar puncture, performed on the 50th hospital day, yielded clear, colorless cerebrospinal fluid under a normal pressure of 110 mm of water that contained 40 white cells, mainly lymphocytes, per cubic millimeter ; the protein was 53 mg and the glucose 50 mg. As shown in **Figure 3**, a twelve lead electrocardiogram, recorded on the same day, showed normal sinus rhythm at a rate of 60 beats/min. T waves in the leads III and aVF remained inverted, but tall U waves in the chest leads were not seen. The QTc was 0.36 sec. The patient was discharged on the 64th hospital day, when he was well and in normal sinus rhythm.

DISCUSSION

In the present patient a diagnosis of aseptic meningitis was made from the clinical findings of moderately high fever, meningeal signs such as severe headache, nausea, nuchal rigidity and Kernig's sign and abnormal findings of the cerebrospinal fluid such as a high pressure, an increase in white cell count, high protein content and negative cultures of the fluid for bacteria, mycobacteria and fungi. During the acute phase of this aseptic meningitis, the patient developed severe sinus node dysfunction with long sinus pauses which recovered gradually to normal sinus rhythm as the clinical manifestations of the aseptic meningitis subsided.

It has been well known that many electrocardiographic abnormalities occur in a variety of central nervous system diseases such as cerebrovascular accidents, intracranial space occupying lesions, head trauma, meningitis, etc[1~5]. These electrocardiographic abnormalities include abnormalities of ST segments and T waves, prominent U waves, abnormal Q waves and prolonged QT intervals. Sinus bradycardia and atrial fibrillation during the course of the central nervous system diseases have also been reported[7]. Chandra *et al* reported a case of tuberculous meningitis with reversible sick sinus syndrome with junctional and ventricular escape and fusion beats. However, they did not find long sinus pauses causing dizziness in their case as observed in our case[6]. Furthermore, Bhatnager *et al* have reported a case of sick sinus syndrome in association with cerebrovascular accidents[7]. To our best knowledge, the present patient is the first case of aseptic meningitis presenting reversible, severe dysfunction of the sinus node with frequent emergences of long sinus pause, the longest sinus pause of 9.84 seconds, accompanied by dizziness.

The underlying mechanism responsible for

the sinus node dysfunction in patients with the central nervous system disease has been discussed in relation to the high intracranial pressure[2~5]. Abildoskov *et al* concluded that electrocardiographic abnormalities are related to changing intracranial pressure causing abnormalities of the sympathetic tone to the heart resulting into vagal dominance which suppresses the sinus node function[4]. It has been reported that the cerebrovascular accidents such as intracranial and subarachnoid hemorrhages showing rapid increase in the pressure of cerebrospinal fluid have a high incidence of electrocardiographic abnormalities[3]. The present patient had no apparent myocardial and pericardial disease being able to cause sinus node dysfunction both before and after the onset of aseptic meningitis. Sinus node function in the present patient became almost normal as clinical manifestations of aseptic meningitis subsided. Therefore, sinus node dysfunction in the present patient is thought to be directly related to concurrent aseptic meningitis. High pressure of cerebrospinal fluid implicating high intracranial pressure was observed when the present patient developed severe dysfunction of the sinus node during acute phase of aseptic meningitis. It is assumed from this finding that severe dysfunction of the sinus node in the present patient is related to high intracranial pressure. Furthermore, in the present patient slow sinus rate was considerably increased by intravenous administration of atropine during the convalescent phase of aseptic meningitis. It is thought from these findings that increase in the vagal tone resulted from a high intracranial pressure during acute phase of aseptic meningitis may play an important role in the genesis of sinus node dysfunction in the present patient.

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