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<td>Author(s)</td>
<td>Yano, Katsusuke; Mitsuoka, Takao</td>
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Reversible Dysfunction of the Sinus Node during Acute Phase of Aseptic Meningitis

Katsusuke YANO, M.D., and Takao MITSUOKA, M.D.

The Third Department of Internal Medicine,
Nagasaki University School of Medicine

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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 bradycardia 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. Atrial fibrillation and sinus bradycardia have also been reported in such patients. 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°C, 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 lactate dehydrogenase (LDH) 279 U, and the alkaline phosphatase 4.9 U per liter. A lumbar 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 microscopic 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 cardiothora-
cic 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 brady-
cardia 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 not seen. The QTc
was 0.38 sec. During the next 10 days follow-
ing 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 moni-
toring. 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
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 electrocar-
diographic abnormalities occur in a variety of
central nervous system diseases such as
cerebrovascular accidents, intracranial space
occupying lesions, head trauma, meningitis, etc1-5.
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
reported7. 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 case6. Furthermore,
Bhatnager et al have reported a case of sick
sinus syndrome in association with cerebro-
vascular accidents7. To our best knowledge,
the present patient is the first case of aseptic
meningitis presenting reversible, severe dys-
function of the sinus node with frequent emer-
gences of long sinus pause, the longest sinus
pause of 9.84 seconds, accompanied by dizzi-
ness.

The underlying mechanism responsible for

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,
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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\textsuperscript{3}-\textsuperscript{5}). Abildoskov \textit{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\textsuperscript{1}). 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\textsuperscript{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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