

CASE REPORT

MORBITZ TYPE I SECOND DEGREE AV BLOCK DURING RECOVERY FROM DENGUE HEMORRHAGIC FEVER

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Abstract. Two patients with serologically-proven dengue virus infection and Morbitz type I second degree atrioventricular (AV) block are described. A 7 years old boy (patient 1) with grade 2 and a 7 years old girl (patient 2) with grade 3 illness were admitted to the hospital on the 3rd and the 5th day of the illness, respectively. Both had typical resentation for dengue hemorrhagic fever including fever, hepatomegaly, thrombocytopenia and signs of extravascular leakage. The 7 year old girl also had epistaxis and anemia (Hct 24%). Morbitz type I second degree and 2:1 AV block developed on day 7 (patient 1) and day 8 (patient 2) of the illness, both during recovery periods. Patient 1 also had occasional monomorphic premature ventricular contraction (PVC). There was no other abnormality in the 12-lead EKGs and echocardiograms showed normal ventricular systolic function in both. Other than mild hypokalemia (3.3 and 3.4 mgq/l), serum electrolytes were normal. Neither patients had elevation of serum creatine phosphokinase (CPK). In patient 1, exercise (on day 10) normalized AV conduction and abolished the PVC. Follow up EKG and physical examination at 10 months after the illness was normal. The rhythm in patient 2 resolved to 1st degree AV block (with occasional morbitz type I second degree at night) on day 12. In this patient, exercise resulted in shortening of the PR interval and Valsalva maneuver resulted in further PR prolongation. The patient was well at 1-month follow up with a normal EKG. Morbitz type I second degree AV block during recovery from dengue hemorrhagic fever may be a transient functional impairment of the AV node, in which altered autonomic tone may play a role.

Dengue hemorrhagic fever is one of the most common diagnoses for children admitted to pediatric wards in Thailand. Various electrocardiographic abnormalities were reported previously (Boon, 1976; Pongpanich *et al*, 1973; Smyth and Powell 1954; Lim *et al*, 1970; Chuah, 1987). These are ST segment abnormalites, low QRS voltage sinus bradycardia, first degree AV block, premautre atrial contraction (PAC), and premature ventricular contraction (PVC). We report two patients who developed Morbitz type I second degree atrioventricular (AV) block during recovery from dengue hemorrhagic fever (DHF).

Case 1

VV was a previously healthy, 7-year-old boy who came to the hospital with 2-day history of fever associated with loose stool and poor appetite.

Physical examination revealed an alert, obese boy in no acute distress. Vital signs: BT 38.2°C, PR 100/minute, RR 28/minute and BP 120/60 mmHg. Except for injected pharynx and buccal mucosa, the rest of the physical examination was within normal limits. The patient was admitted to the hospital with the diagnosis of acute gastroenteritis and possible dengue hemorrhagic fever. Laboratory investigations were as follows: CBC showed total WBC of 2,300/mm³ (PMN = 73%, lymphocytes = 21%, monocytes = 6%), hemoglobin = 12.1 g/dl, hematocrit = 38% and platelet count = 207,000/mm³. Urinalysis showed specific gravity 1.020, pH 6.5, protein trace, sugar negative. Serum electrolytes showed Na⁺ 139, K⁺ 3.1, Cl⁻ 104 and total CO₂ 20 meq/l. Treatment consisted of maintenance intravenous fluid and supportive care. Fever subsided on the 5th day of the illness with the patient developing a confluent petechial rash on the skin. Repeated CBC showed total WBC of 2,600/mm³ (PMN 28%, lymphocytes 57%, monocytes 2%), hemoglobin 12.8 g/dl, hematocrit 41%, platelet count 52,000/mm³. Clinical diagnosis of dengue

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hemorrhagic fever was made and the patient was continued on intravenous fluid with no clinical signs of shock. On the 7th day after the illness, irregular pulses were detected without any symptoms or hemodynamic instability. Except for the irregular pulse, cardiovascular examination was normal. A twelve-lead electrocardiogram (EKG) was obtained, which showed sinus rhythm with Mobitz type I, second-degree atrioventricular (AV) block and occasional monomorphic premature ventricular contraction (PVC) (Fig 1). CBC showed WBC 5,700/mm³ (PMN 37%, lymphocytes 32%, monocytes 14%), hemoglobin 11.1 g/dl, hematocrit 36%, platelet count 95,000/mm³. Serum electrolyte showed Na⁺ 140, K⁺ 3.3, Cl⁻ 107, total CO₂ 22 meq/l, serum CPK 44 μ/l, SGOT 262 μ/l, SGPT 233 μ/l, LDH 554 μ/l. Echocardiogram was normal. Dengue virus titer (IgM) was positive. The patient was asymptomatic with stable vital signs so he was monitored only without additional treatment given. The rhythm (Mobitz type I, second-degree AV block and occasional PVC) persisted until day 10 of the illness. Sub-maximal exercise test (peak heart rate = 70% of maximal heart rate) at this time showed normalization of the AV conduction and disappearance of the PVC. The patient was then discharged home. He did not come to follow up until 10 months after the illness, at which time a repeat 12-lead EKG and physical examination were completely normal.

Case 2

KT was a 7 years old girl with asymptomatic homozygous HbE disease. She was admitted to a community hospital on the 5th day of the illness characterized by fever, decreased appetite and slight cough. She also had epistaxis 1 day prior to admission. Initial physical examination showed a low blood pressure (90/60 mmHg), hepatomegaly (1.5 cm below right costal margin) and positive tourniquet test. Initial laboratory findings were WBC = 6,300/mm³ (PMN 62%, lymphocytes 38%), hematocrit 24%, platelet count 1000,000/mm³. The diagnosis of dengue hemorrhagic fever was made, fluid 20 ml/kg was given for hypotension and the patient was transferred to Surin Hospital. At Surin Hospital, 10 ml/kg of fresh whole blood was given for anemia and low blood pressure. Repeated CBC showed WBC 5,860/mm³ (PMN 54%, lymphocytes 32%), hematocrit (before blood transfusion) 26.5%,

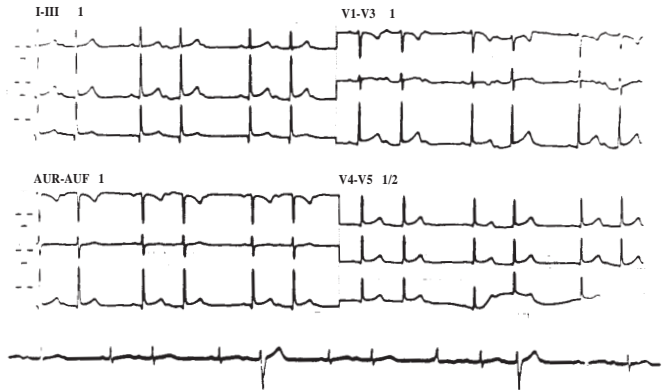


Fig 1—12-lead electrocardiogram of the patient 1 showing Mobitz type I atrioventricular block and occasional monomorphic premature ventricular contraction (PVC) on day 7th after the onset of fever.

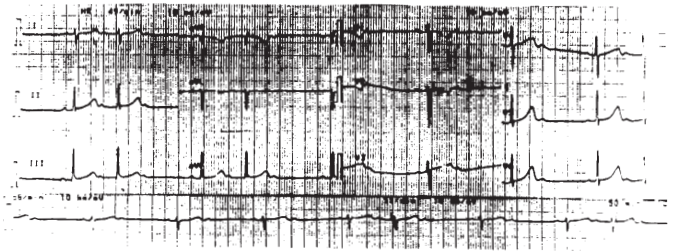


Fig 2—12-lead electrocardiogram of the patient 2 showing Wenckebach and 2:1 AV block on the 9th day after the onset of fever.

platelet count 69,000/mm³. A chest radiograph showed normal cardiac shadow and right pleural effusion. On the next day (7th day after onset of fever) the patient's clinical status improved with no fever, stable vital signs (BP 90/50-110/70) and good urine output (3.1 ml/kg/hour). Laboratory investigations showed hematocrit 33%, platelet count 63,000/mm³. A dose of furosemide (Lasix) was given for the pleural effusion. On the 8th day of the illness, irregular pulse was noted without any hemodynamic instability. Twelve-lead EKG showed 1st and 2nd degree AV block. Laboratory investigations at this time showed hematocrit 32%, platelet count 106,000/mm³, serum Na⁺ 145, K⁺ 3.4, Cl⁻ 110 and total CO₂ 30 meq/l, serum CPK 18 μ/l, SGOT 111 μ/l, and LDH = 989 μ/l. The patient was then referred to Chulalongkorn hospital. There, Wenckebach AV block and 2:1 block was seen without any symptoms or hemodynamic instability (Fig 2). Echocardiogram was normal except for trace aortic and mitral regurgitation. Antibody to dengue virus was positive for IgM and > 1:20,480 for IgG (for dengue virus type 2, no paired serum

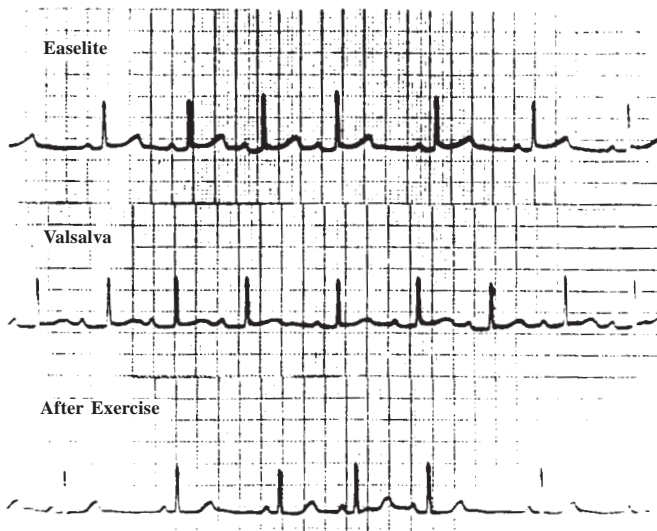


Fig 3—Response of atrioventricular (AV) conduction to Valsalva maneuver and exercise during the period of first degree AV block in patient 2 (day 12 after onset of fever).

was available). No treatment other than close observation was required. On the 12th day of the illness, the AV block resolved to first degree. Autonomic maneuvers were done at this time which showed improved AV conduction with exercise and further PR segment prolongation with Valsalva maneuvers (Fig 3). Twenty-four hour Holter monitoring at this time showed sinus rhythm with first degree AV block in the daytime and occasional Wenckebach AV block and 2:1 block at night. The patient was discharged home on the 14th day of the illness. A repeated physical examination and 12-lead EKG at 1-month follow up was normal.

Electrocardiographic abnormalities have been observed in as many as 44-75% of patients with viral hemorrhagic fever (Boon, 1967; Lim *et al*, 1970). Although sinus bradycardia and prolongation of the PR interval were commonly observed, atrioventricular block beyond first degree appeared to be rare in these reports. In one review (George and Lum, 1997), varying degrees of nodal block during convalescence was said to be frequently seen, although descriptions of the patients were not given in this review.

Many similarities existed in these 2 cases that are worth consideration. First, the AV blocks were confined to first degree and Mobitz type I second degree. Although, by definition, the type of 2:1 block cannot be specified from a surface EKG, in

these cases they most likely represented Mobitz type I in the presence of a Wenckebach phenomenon without a higher degree of AV block. Second, the AV block responded to autonomic alteration. During the period of the AV block, sympathetic stimulation (exercise) resulted in improved conduction and vagal stimulation (Valsalva maneuver) resulted in worsening of the AV block. These observations led us to conclude that the site of heart block in these 2 cases was at the atrioventricular node (rather than the His-Purkinje system). In general, blocks at the AV node (compared to infra-nodal blocks) carry a better prognosis and in many cases, represent functional rather than permanent pathology. Third, AV block in these patients were transient and occurred during the recovery period, in which transient sinus bradycardia is also commonly seen (George and Lum, 1997; Nimmarnit, 1996). Because of similarities in the cellular

electrophysiologic properties of the sinus (SA) and the atrioventricular (AV) node, these transient abnormalities may represent a transient functional (rather than anatomical) impairments resulting from the same mechanism. Among them are abnormalities in the autonomic tone, adenosine metabolism or other abnormalities in the cells that use predominantly calcium current for depolarization. Alternatively, localized pathology such as minute bleedings in the areas of SA and AV nodes may be responsible. Subendocardial hemorrhage, mostly in the interventricular septum has been reported in 47% of autopsy cases of patients who died from dengue hemorrhagic fever (Bhamarapravati *et al*, 1968). It is possible that hemorrhages in the vicinity of the AV node may result in transient AV block although it is difficult to explain why a higher degree of AV block is hardly seen.

While the mechanism for this phenomenon is still unclear and needs further investigation, we believe that a clinical implication exists from this observation. Heart block in the presence of acute viral infection often signifies serious pathology such as acute myocarditis. Although whether myocarditis exists in patients with dengue hemorrhagic fever is still a matter of debate (Chuah 1987; Bhamarapravati *et al*, 1968; Wali *et al*, 1998), we believe that the transient, low grade AV block in these 2 cases without myocardial dysfunc-

tion or signs of myocardial injury (EKG changes or elevated CPK) made diagnosis of acute myocarditis unlikely. Thus, Mobitz type I AV block in asymptomatic patient during the recovery phase of dengue hemorrhagic fever may be benign and careful observation alone in such a patient may be justified. Further study of the incidence and clinical courses of this phenomenon may prevent unnecessary transferring of these patients to tertiary centers. It must be noted that the clinical distinction between dengue hemorrhagic fever and acute myocarditis can be difficult in certain patients, and heart block in patients who present in shock (or recovering from shock) must always be evaluated carefully. If the presentation is atypical, or one is simply uncertain about the diagnosis, referral to a center capable of taking care of patients with acute myocarditis is still recommended. Finally, although this phenomenon has been previously observed (George and Lum, 1997), there is still paucity of data which makes further follow up and reports of cardiac conduction defects in dengue hemorrhagic fever a necessity.

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