

TRANSIENT VENTRICULAR ARRHYTHMIA AS A CARDIAC MANIFESTATION IN DENGUE HAEMORRHAGIC FEVER — A CASE REPORT

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SYNOPSIS

Ventricular arrhythmias occurring during the acute stage of dengue haemorrhagic fever have not been frequently reported. In this case report, a young adult presenting with transient ventricular arrhythmia as a cardiac complication of dengue haemorrhagic fever is described. A myocarditis due to an Arbovirus B infection is thought to be the probable cause.

INTRODUCTION

The pathogenesis of Dengue Haemorrhagic Fever/Dengue Shock Syndrome (DHF/DSS) remains a perplexing problem. Several research workers in this intriguing field have described many interesting findings in DHF/DSS such as leukopenia (Simon 1931) (1), eye ground changes (Gill 1923) (2), varied neurological syndromes (Siler et al 1926 (3); Goldsmid and Crosse 1916 (4)) and cardiac abnormalities (Hyman 1943 (5); Smyth and Powell 1954 (6); Wong and Tan 1967 (7)). Commonly cited cardiac manifestations include conduction defects and non-specific ST and T wave changes (6, 7). In this case report transient ventricular ectopy occurring as a manifestation of DHF is highlighted.

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CASE REPORT

A 31 year old, previously healthy man working as a public health overseer presented to District Hospital, Kuala Lipis, Pahang in October 1985 with a three days history of fever associated with chills but no rigors. Other accompanying symptoms were a mild non-productive cough, polyarthralgia, malaise, vomiting and anorexia. There were no urinary or bowel symptoms. He did not complain of chest pain or palpitations. There was no family history of coronary artery disease. He is a non-smoker and a non-alcoholic.

Physical examination showed he was comfortable. He was febrile with a temperature of 38.5°C. An erythematous rash was noted over his abdominal wall and limbs. BP was 100/70 mm Hg and he had an irregular pulse rate of about 80 per minute. Tourniquet test was positive. Apart from a soft short systolic praecordial murmur, no abnormalities were detected in his heart. His liver was palpable 3 cm. below the costal margin. Systemic examination was otherwise

normal.

His haemoglobin concentration was 11.8 gm./dl, white cell count $3.1 \times 10^9/l$. (neutrophils 39%, lymphocytes 61%), platelet count $80 \times 10^9/l$, packed cell volume 0.49, bleeding time 3 mins., clotting time 4 mins., 45 secs. ESR 8 mm. 1st. Hr, serum electrolyte concentrations were sodium 140 m Eq/l, chloride 105 m Eq/l, potassium 4.1 m Eq/l, urea 4.2 m mol/l (25 mg/dl.) Urinalysis showed albumin +++ associated with cellular and granular casts. 24 hours urine protein was 0.1 gm. Chest X-ray was normal. Cardiac enzymes were elevated. Serum lactic dehydrogenase (reversed) 725 U/l, (N 230—461 U/l), serum creatine kinase 194 U/l, (N 24—170 U/l) serum hydroxy-butyric dehydrogenase 294 U/l, (N 7—182 U/l) aspartate transaminase (AST) 159 U/l (N up to 37 U/l). Throat swab and blood cultures were negative. Blood uric acid was 6.5 mg/dl. Electrocardiographic abnormalities recorded during the first few days in hospital are shown in figures 1 and 2.

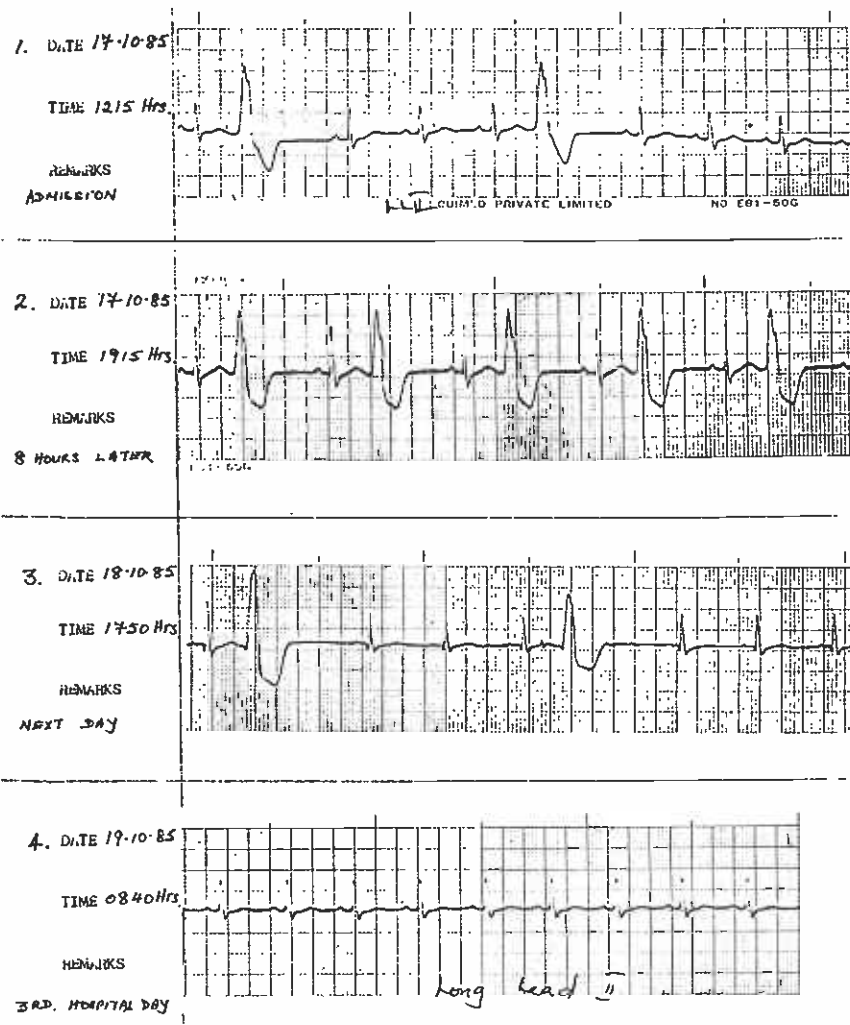


Figure 1. Electrocardiographic tracings recorded during the acute stage of dengue haemorrhagic fever. On admission uniform, unifocal ventricular ectopis were detected at a frequency of about 1:3. Few hours later (2nd. tracing) this progressed to ventricular bigeminy. The following day the ectopics were less frequent and subsequently sinus rhythm was restored by the third hospital day (i.e. 6th day of illness).

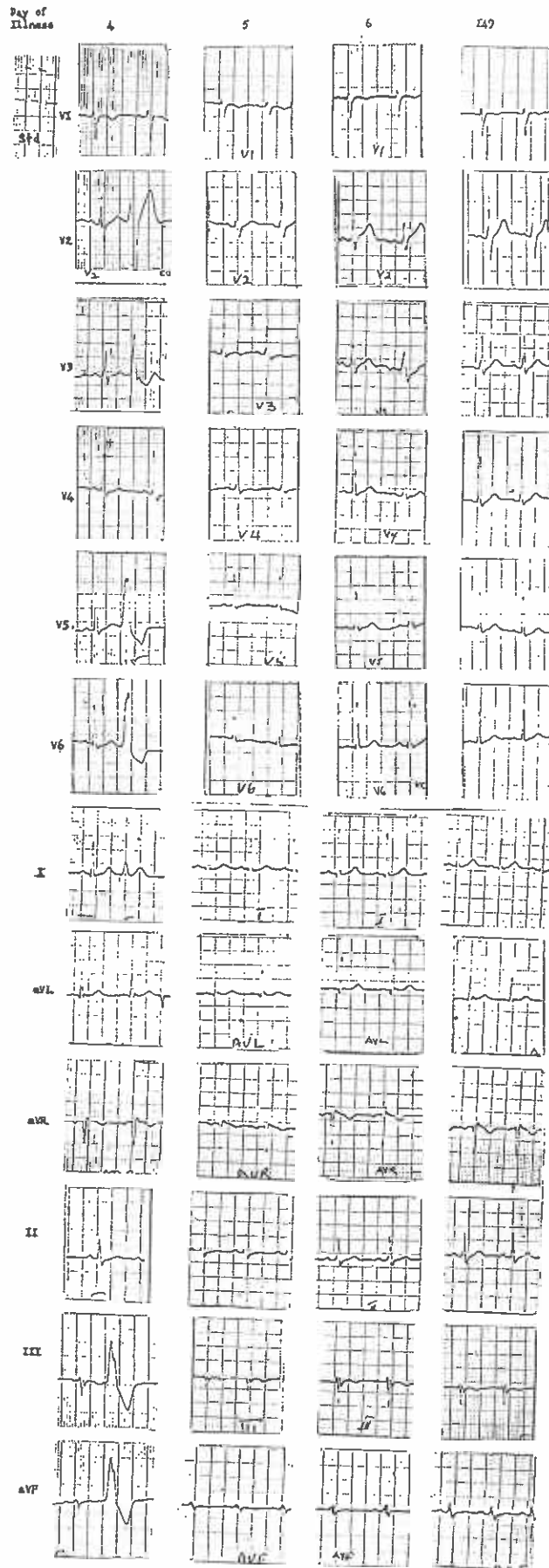


Figure 2. Serial electrocardiograms showing non-specific negative T-wave changes in leads III, aVF, V1, V3, V4 and flattened T waves in leads II, V5, V6 on the fifth day of illness; the tracing on the fourth day of illness showed upright T waves in most of the leads. By the sixth day of illness there were reversion of the T waves to normal. By five months (149th day) the T waves were upright in all leads except aVR and III.

Throughout his whole hospital stay, he remained relatively well. No anti-arrhythmic agents were given. He was discharged well after a week, during which time his albuminuria cleared and his platelet counts returned to normal. Follow-up till five months was unremarkable.

COMMENT

Many viruses have been incriminated in the causation of myocarditis; well known examples are the influenza, ECHO and Coxsackie B viruses. Cardiac abnormalities in the form of conduction defects and non-specific ST-T wave changes (6,7), attributable to viral haemorrhagic fever (which includes Thailand and Philippine viral haemorrhagic fever, also known as dengue haemorrhagic fever, Korean haemorrhagic fever and Argentina viral haemorrhagic fever) have been well documented. But ventricular arrhythmias occurring during the course of DHF are less frequently reported.

In an attempt to explain the possible mechanism underlying the onset of the ventricular arrhythmia (Figure 1), predisposing factors were looked into. This patient did not show any biochemical evidence of electrolyte imbalance, in particular the serum potassium concentration. It would seem more likely that there is some degree of myocardial damage as suggested by the elevated cardiac enzymes. Thus, a myocarditis could be the cause for the transient ventricular arrhythmia and negative T wave changes (Figure 1, 2). The haemagglutination — inhibition test (Table 1) supports the diagnosis of an Arbovirus B infection and the clinical picture of fever with a positive tourniquet test and thrombocytopenia suggests the dengue virus as the probable causative agent in his patient.

TABLE 1
ARBOVIRUS STUDIES USING
HAEMAGGLUTINATION — INHIBITION TEST.
(PROCEDURE OF CLARKE AND CASALS (1958)
USING THE MICROTITRE TECHNIQUE).
(IMR B 8531/8532)

Virus Antigen	4th Day of Disease	9th Day of Disease
Group A Sindbis	10	10
Group B Dengue 1	160	320
Dengue 2	640	1280
Dengue 3	320	320
Dengue 4	1280	1280
Jap Encephalitis	1280	1280

Report: Presumptive positive to group B infection.

Although Hyman (5) did not believe that dengue virus produces a myocarditis, other recent workers like Milei et al (8) in Argentina, in a study of 106 necropsy

cases of viral haemorrhagic fever (VHF) comprising 17 Argentina VHF, 5 Thailand VHF, 67 Philippine VHF, 8 Bolivian VHF and 10 Korean VHF found that myocardial damage is common in VHF. These cardiac alterations consisted of congestion, oedema, haemorrhagic and necrotic phenomena, interstitial inflammatory reactive changes (IRC) and interstitial myocarditis. It is postulated that the congestion, oedema and necrotic phenomena may be related to the shocked state of the patient. However, the haemorrhagic phenomena, IRC and the interstitial myocarditis may be directly associated with the causative viruses. IRC is a distinctive picture which may suggest that an immunologic phenomenon may play a role in the pathogenesis of myocardial damage in VHF (9). These workers (8) concluded that cardiac damage exists in a great proportion of VHF. These extensive haemorrhages and interstitial myocarditis may elicit mechanical or electrical disturbances. However, it must be borne in mind that these phenomenal histological features were studied in autopsy cases; in less severe cases of VHF these changes may be expected to be proportionately less extensive and perhaps reversible.

In outbreaks of DHF/DSS, the haemorrhagic manifestations and circulatory collapse are alarming features and nearly always appropriately anticipated. To be alerted to this fatal complication is paramount to the management of DHF/DSS. As clinicians we must be attentive also to the cardiac complications that may occur during the evolution of dengue haemorrhagic fever even in the milder forms of the DHF spectrum as shown in the case presented. In this way we may be able to detect other cardiac abnormalities either of a transient nature or otherwise which may have been missed in the past.

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