# LETTER TO THE EDITOR

# *CHROMOBACTERIUM VIOLACEUM* SEPTICAEMIA IN MALAYSIA.

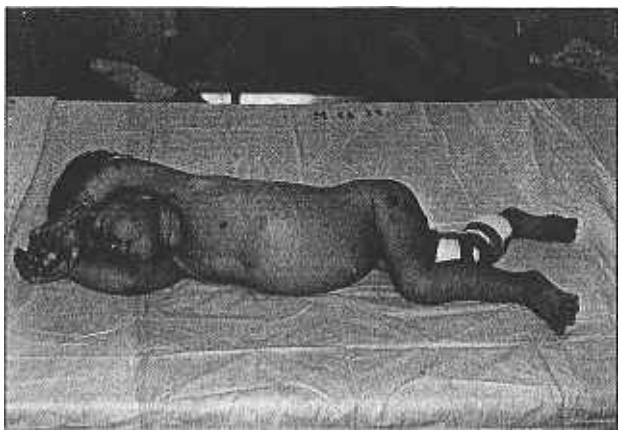
SINGAPORE MED J 1994: Vol 35: 426

Dear Sir,

We read with keen interest the paper by Hassan et al[1] regarding a fatal case of *Chromobacterium violaceum* septicaemia in a 19-year-old man. We note that this is the first case reported in Malaysia since JE Lessler's report in 1927. We recently reported two paediatric cases: a 10-month-old girl who survived, and a 9-year-old boy who died[2]. Since then, one new fatal case was reported to us involving a 4-year-old boy admitted to one of our district hospitals. We present here a summary of our findings. Incidentally, our first case was also reported in June and the second case was reported in July. The most recent case was reported in March 1993, and we hope to publish a full report soon.

CASE 1. (F, 10 months). Admitted in June, 1991 for fever following accidental cut and grazes on her right leg. She was treated by a general practitioner. Three days later she developed bilateral leg swelling with refusal to straighten the right knee. Furuncles were noted over the right buttock and left thigh. The right toe, right first, second, third fingers, and left middle fingers became gangrenous necessitating amputation (Fig 1). Gangrene also affected most of the soft tissues of the nose from the columella to the root, with erosion of the nasal cartilage necessitating debridement. The left eye later developed chemosis, pussy discharge, proptosis and corneal ulceration, with eventual destruction. The left elbow developed septic arthritis, and pathological dislocation requiring open reduction and internal fixation (Fig 1). Three sets of blood culture and pus swab from the abscess grew *Chr. violaceum* two days after admission. The patient was discharged after 4 months of hospitalisation.

**Fig 1. – Physical manifestations of *Chromobacterium violaceum* infection in a surviving 9-month-old girl. The right toe, left fingers and nose were gangrenous. Multiple furuncles affected the buttock, right thigh, arm, face and abdomen.**



CASE 2. (M, 9). Admitted in July 1991 with high temperature, toxaemia and delirium of one day duration. Multiple small ulcers were noted on the lower limbs. He sustained a right ankle injury at school 9 days prior to admission. Three days after the injury the patient had high fever and was given an injection by a traditional Chinese medical practitioner. Six hours after admission the patient collapsed and died. *Chr. violaceum* was grown from the blood culture.

CASE 3. (M, 4). Admitted in March 1993 with fever and bronchopneumonia. He was pale and highly agitated. Chronic ulcer were noted on his right foot. He collapsed and died seven hours after admission, due to septicaemic shock. *Chromobacterium violaceum* was isolated from blood culture taken on admission. Further clinical details will be published soon.

All our cases involved paediatric patients. As in the Hassan et al case, our cases had history of skin injuries. *Chr. violaceum* was the invasive primary pathogen, and was isolated from aerobic and anaerobic blood culture, taken on admission.

The main clinical features seen in our cases were skin ulcer, septicaemia and toxaemia. Antibiogram were similar to those reported by Hassan et al. As noted by Hassan et al *Chr. violaceum* infection is frequently fatal and two of our three cases were fatal. Prognosis may be improved with early definitive laboratory identification, and susceptibility testing together with appropriate surgical intervention.

Contributory factors leading to the fatal outcome of the infection were late referral, inappropriate antibiotic treatment and a general lack of awareness of *chromobacterium septicaemia*. In contrast, the surviving 10-month-old girl was given aggressive antibiotic therapy together with surgical interventions. We hope the report by Hassan et al and this letter will help increase awareness amongst microbiologists and clinicians regarding this rare but frequently fatal soil transmitted infection.

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BOOK REVIEW

# A REVIEW OF DISEASES IN MALAYSIA

ed. KG Lim

Malaysia. Pelanduk Publications. 1993

This is not a book which offers material for light reading and should not be read from cover to cover but to be used as a useful reference one to have in one's office room or clinic. It deals with a wide range of diseases commonly encountered in Malaysia. It is divided into three sections. Section 1 deals with a variety of conditions, namely, genetic diseases, nutritional disorders, physical and chemical injuries and cancers. Section 2 deals with various types of microbial infections. Section 3 deals with a variety of conditions affecting different systems of the body. On the main, it takes a demographic approach to these diseases. Clinical signs and symptoms have been reduced to the minimum. The book, as the author points out, is targeted at four groups of people: the practising doctor, the scientific investigator, the administrator and the medical student. It thus seeks to provide: 1) the practising doctor with a knowledge of the pattern of disease in his locality, 2) the investigator with epidemiological information about diseases, 3) the administrator with the necessary information about the medical and health resources, and 4) the medical student with useful information for his future career. The style of writing is simple and readable although the deluge of numerical data may sometimes appearing boring - but this method of quantitative presentation is unavoidable if one wants to get the message through. The author has put in a tremendous amount of effort to collate all the data into one reasonably sized book. It is not erudite in terms of academic knowledge but it is certainly a laudable effort and should be recommended as an important resource for this type of information.

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## ANSWER TO ELECTROCARDIOGRAPHIC CASE

Diagnosis: myocardial ischaemia

## DISCUSSION

This patient's electrocardiogram shows typical as well as more subtle signs of myocardial ischaemia. The obvious abnormality is downsloping ST segment depressions in leads I, V2 to V6. A closer look reveals inverted U waves (best seen in leads V4 to V6) and prolonged QT interval (0.48 seconds after correction for rate).

The first signs of coronary insufficiency are usually evident in the repolarisation process ie abnormalities of the ST segment, the T waves, the U waves and the QT interval[1]. Horizontal or downsloping depression of the ST segment is one of the more typical signs of myocardial ischaemia or injury. It should be distinguished from the junctional type of ST segment depression found in normal persons. The latter is characterised by depression of the proximal part of the ST segment with an upward slope of the distal part and a duration less than 0.08 second. It should also be distinguished from digitalis effect which produces a straight downward slope of the ST segment beginning from the isoelectric base without depression of the proximal part of the segment[2]. In addition, digitalis produces shortened QT interval as opposed to ischaemia which is associated with prolonged QT.

Another differential diagnosis of ST segment depression is ventricular hypertrophy with strain but this is usually accompanied by other criteria of ventricular hypertrophy.

Interestingly, this electrocardiogram shows inverted U waves which are often unnoticed. The U wave is a small wave of low voltage. It is sometimes seen following the T waves and has been postulated to represent repolarisation of the papillary muscle or the Purkinje system. Its normal polarity is the same as that of the T wave[3].

An inverted U wave is abnormal and associated with heart disease. It is seen in patients with myocardial infarction as well as ischaemia without infarction. It may be seen at rest or only during exercise and may be the only abnormality in patients with coronary artery disease[4]. Inverted U waves in the resting electrocardiogram are claimed to be a faithful index of significant stenosis of the left main or left anterior descending artery[5]. Even the mere absence of a U wave has been reported to predict a significant increase in the incidence of myocardial infarction[6]. If the U wave becomes inverted in the beat following an extrasystole, this is indicative of myocardial disease, most commonly coronary insufficiency. Inverted U wave can also be seen in the left precordial leads in left ventricular hypertrophy, especially with volume overload such as aortic insufficiency. Similarly, the U wave may also be inverted in the right precordial leads in right ventricular hypertrophy[4].

Another feature of ischaemic heart disease seen in this electrocardiogram is prolonged QT interval. A prolonged QT means there is delayed repolarisation of the ventricular myocardium and is associated with an increased predisposition to reentry and hence serious ventricular tachyarrhythmias, syncope and sudden death. Myocardial ischaemia is an important cause of prolonged QT[7]. Others include congestive heart failure, rheumatic fever, myocarditis, cerebrovascular accidents, electrolyte imbalance, drugs and congenital prolonged QT syndrome.

Thus this lady who presented with syncope has multiple features of myocardial ischaemia in her resting electrocardiogram. Syncope is uncommonly the presentation of ischaemic heart disease. Her symptoms were due to episodes of ischaemia-induced ventricular tachyarrhythmias which were subsequently documented on continuous electrocardiographic monitoring in the ward. A coronary angiogram done showed critical stenosis of the left main coronary artery and the patient underwent emergency coronary artery bypass surgery.

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