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Serum Transaminase in Diphtheritic Myocarditis

by

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Abstract

Ninety-one cases out of 102 diphtheritic children admitted to the University of Gadjah Mada Hospital were studied, correlating enzyme levels (SGOT and SGPT) with ECG findings. The results showed that there was a correlation between the SGOT value and the severity of the ECG abnormalities. Beside ECG, the determination of serum transaminase should be considered as an adjunct laboratory examination in evaluating the diagnosis and prognosis of diphtheritic myocarditis.

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Introduction

Serum enzyme determinations for the diagnosis of heart diseases are widely used (La Due et al., 1955; MacDonald et al., 1957; La Due, 1957; Friedberg, 1966). Glutamic oxalacetic transaminase (GOT) is found in the heart, muscle, liver, brain, and kidney. In the absence of disease in the other organ systems, elevation of SGOT is assumed to be indicative of acute myocardial damage (La Due, 1957; MacDonald et al., 1957). It is well known that the most serious complication of diphtheria is cardiac damage which is due to exotoxin liberated by diphtheria bacillus. Post-mortem examinations reveal evidence of varying degeneration, including necrosis of myocardial fibres, myolysis, and interstitial inflammation (Fahr, 1966).

ECG abnormalities are the earliest evidence of acute diphtheritic myocarditis. The mortality rate of patients suffering from diphtheria with ECG abnormalities is higher than of those without any ECG abnormality (Kwari et al., 1965; Morgan, 1963). Serum glutamic pyruvic transaminase (SGPT) is not a very sensitive index for cardiac damage, but in the heart with massive damage or in congestion of the liver caused by cardiac failure, an elevation of SGPT may be found. The purpose of this study is to investigate any correlation between the elevation of SGOT and

SGPT with the finding of ECG abnormalities in diphtheritic myocarditis.

Material and methods

All children suffering from diphtheria admitted to the Department of Child Health, University of Gajah Mada Hospital, during the period from January 1973 until January 1974 were studied. Twelve lead ECG examinations were performed, with the frequency depending upon the course of the disease. The ECG abnormalities were classified according to Tahernia's classification (1969) into 2 groups:

- minor variations consisting of S-T shift, T wave changes, prolonged QT interval, and low voltage of QRS in the precordial leads; and
- major variations consisting of disturbances of impulse formation such as supraventricular tachycardia, ventricular fibrillation, and disturbances of impulse conduction such as bundle branch block and all degrees of atrioventricular block.

The examination of SGOT and SGPT were carried out at the Department of Clinical Pathology, University of Gajah Mada, spectrophotometrically using the Reitman-Frankel method, the frequency depending upon the finding of ECG abnormalities and the course of the disease.

Results

Of 102 diphtheritic children admitted to the hospital, only 91 children were included in this study and the other 11 were excluded due to incomplete examinations. The range in age was from 7 months to 12 years. There were 57 children in whom either clinical or ECG examinations showed no abnormalities. These were included in group A and considered as controls. Twenty-four children who showed myocarditis with minor abnormalities were included in Group B, and 10 children with major abnormalities in Group C. Two children of Group A, 2 of Group B, and 4 of Group C died.

Although several determinations of the serum level of enzymes were done, only the highest levels of serum transaminase activity encountered during the peak of disease were taken for analysis. The results of SGOT and SGPT determinations are shown on Table 1. The elevation of SGOT lasted for 10-20 days and afterwards returned to normal. The ECG abnormalities usually disappeared with convalescence, but in 2 children who remained under observation, the abnormalities were found to persist for 8 and 12 months respectively. The results show that there is a correlation between the value of GOT in the serum and the ECG finding (Fig. 1).

TABLE 1: *Results of SGOT and SGPT determinations in 3 groups.*

	SGOT unit/ml.		SGPT unit/ml.	
	Range	Mean \pm S.E.M.	Range	Mean \pm S.E.M.
Group A	5.3 — 62.8	34.71 \pm 1.93	2.2 — 26.4	10.32 \pm 0.81
Group B	31.7 — 100.6	68.40 \pm 3.01	5.8 — 41.7	15.25 \pm 1.68
Group C	47.2 — 158.2	105.35 \pm 11.73	6.4 — 66.7	27.97 \pm 4.17

Discussion

ECG abnormalities in patients with diphtheria usually occur at the end of the first week or during the second week. This is generally the earliest evidence of myocarditis. Therefore, a routine ECG examination is very important for the early

detection of cardiac involvement. The intensity of electrocardiographic abnormalities is generally parallel to the clinical severity of the disease (Ledbetter et al., 1964). Kwari et al. (1965) reported a mortality rate of 40% in patients with ECG abnormalities which is 10 times that of patients with normal ECG.

Our study showed an elevation of SGOT roughly coinciding with the onset of ECG changes. This indicates that either ECG abnormalities or elevation of SGOT activity are manifestations of cardiac cell damage. The elevation of SGOT lasted for 10 — 20 days, which was longer than generally seen in myocardial infarction where the elevation of SGOT returns to normal at the 4th — 7th day of the disease (Cantarow and Trumper, 1962; Friedberg, 1966). This can be explained rationally, since the causative factor of myocardial infarction usually is of a coronary artery occlusion followed by the abrupt release of enzyme from damaged myocardial cells (Choremis

and Leonidas, 1962). Usually this phenomenon does not last more than a few days, unless a new process occurs (La Due, 1957). In diphtheria, the influence of toxin on cardiac muscle lasts longer and therefore the release of the enzymes is more prolonged.

The determination of SGPT in this study showed no clear elevation. The obvious explanation is that myocardial cells are rich in GOT but do not contain significant quantities of GPT, which is more abundant in liver cells. Choremis and Leonidas (1962) and Tahernia (1969) found the elevation of SGPT in severe cases of diphtheritic myocarditis, probably related to gross cardiac failure.

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Fig. 1. : SGOT and SGPT values \pm sem.

