SILENT MYOCARDIAL ISCHEMIA IN HEMODIALYSIS PATIENTS — FACTOR FICTION?

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ABSTRACT

Thirty eight patients underwent Holter ECG monitoring for a 48 hour period covering dialysis and intermediate period to detect incidence of myocardial ischemia manifesting as ST segment changes. Seventeen patients (44.7%) had 165 episodes of dynamic ST segment changes lasting from 1 to 177 minutes, with maximum ST depression of 4 mm. The mean age of patients was 45 ± 14 years and 14 (82.6%) of them were males. Ten (58.8%) patients had hypertension, and 5 (29.4%) patients each had diabetes mellitus and pre-existing coronary artery disease. Six (35.3%) patients with dynamic ST segment changes had ventricular ectopics ranging from isolated ventricular premature contractions to episodes of ventricular tachycardia. No significant hypotension or angina was documented during these episodes of ST segment deviation. We concluded that hemodialysis plays an important role in the genesis of the above ECG changes.

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KEY WORDS: Ischemic heart disease; Holter monitoring; Renal dialysis.

Introduction

Silent myocardial ischemia has been thoroughly investigated in coronary artery disease and related diseases [1]. However, there is little information concerning its existence and frequency in hemodialyzed patients, even though cardiovascular complications are very common in uremic patients on regular hemodialysis (HD) and are often the cause of death [2].

The purpose of this study was to investigate painless myocardial ischemia in hemodialyzed patients, to identify predictors if any, and to identify correlation between episodes of myocardial ischemia and clinical events like hypotension, angina and ventricular arrhythmias.

Material and Methods

Thirty eight patients of chronic renal failure on maintenance HD were studied. All patients were hemodialyzed twice a week for 4 hours at a time

using a Terumo 1.2 M hollow fibre kidney. Blood flow rates were maintained at 225-250 mL/min. A standard acetate dialysate solution was used.

Body weights and blood pressures were recorded before and after dialysis. In addition, blood pressures were recorded every thirty minutes during the dialysis. A pre-dialysis laboratory profile consisting of hematocrit, blood urea, serum creatinine, sodium, calcium, potassium and inorganic phosphorous was done in each patient.

All patients were subjected to a standard 12-lead surface electrocardiography (ECG), chest radiography, and M-mode and 2-dimensional echocardiography. Continuous Holter monitoring was done starting 24 hours before HD and continued during the dialysis for a further 24 hours. Thus a post-dialysis record for 20 hours was also obtained. Two bipolar precordial leads were recorded simultaneously. The tapes were analyzed using a computer assisted analyzer for rapid analysis and

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Lown's system of grading was used to classify the ventricular arrhythmias.

An episode of myocardial ischemia was taken as a ST depression of more than 0.1 mV, 80 ms after J point, persisting for period of 1 minute or more [3]. Left ventricular hypertrophy (LVH) was diagnosed on an ECG if the voltage of R wave in aVL was more than 13 mm, or if in the chest leads the sum of voltages of S wave in V1 and R in V5 was more than 35 mm [4]. On echocardiography the diagnosis of LVH was made when the inter-ventricular septum (IVS) or the posterior left ventricular wall thickness was more than 12 mm. Left ventricular dysfunction was diagnosed when left ventricular ejection fraction (LVEF) was less than 50 per cent [5].

The results were analyzed statistically using non-parametric tests, Student's unpaired 't' test and chi square test with Yate's correction. P values less than 0.05 were considered significant.

Results

The patients were divided into two groups according to the presence or absence of S-T segment deviation. Seventeen patients (44.7%) had dynamic S-T changes (Group 1) while 21 patients did not show any dynamic S-T segment change (Group 2). The pre-dialysis physical and laboratory profile did not differ significantly in the two groups.

Group 1 : Mean age of patients was 45 ± 14 years and 14 (82.4%) were males. 165 episodes of dynamic ST changes were seen lasting from 1-177 minutes with a maximum ST depression of 4 mm. In 7 patients, ST-T wave changes were seen only during hemodialysis while in other patients changes persisted for 10 hours after dialysis. Less than 5 episodes of ST-T changes were seen in 5 patients, 6-15 episodes were documented in 3 patients, while 16 episodes of ST-T changes were seen in 6 patients.

Hypertension was present in 10 (58.8%) patients while diabetes mellitus and pre-existing coronary artery disease was seen in another 5 (29.4%) patients each. The mean LVEF was 52 ± 10 per cent while the IVS thickness was 1.2 ± 0.08 cm. Two patients had LVH and one patient had left ventricular dysfunction respectively (Table 1).

None of the patients had hypotension or angina during the index hemodialysis. The relationship to ventricular ectopics is shown in Table 2.

Group 2 : The mean age of patients was 35 ± 8 years and 17 (80.9%) were males. Hypertension and pre-existing coronary artery disease were seen in 5 (23.8%) and 1 (4.8%) patients respectively. None had diabetes mellitus. The mean LVEF was 51 ± 11 per cent and IVS thickness was 1.2 ± 0.06 cm. Two patients had LVH and left ventricular dysfunction (Table 1).

TABLE 1
Cardiac status of patients with dynamic ST segment changes during haemodialysis

	Group 1 (n=17)	Group 2 (n=21)	p
Left ventricular ejection fraction%	52 ± 10	51 ± 11	> 0.05
Ischaemic heart disease (No. of patients)	5 (29.4%)	1 (4.8%)	> 0.05
Left ventricular hypertrophy	2 (11.8%)	2 (9.5%)	> 0.05
Left ventricular dysfunction	1 (5.9%)	2 (9.5%)	> 0.05

TABLE 2
Dynamic ST changes and ventricular ectopics

Arrhythmia	Dynamic ST changes present	Dynamic ST changes absent
Ventricular premature beats, < 1 hr	6	2
Couplets	3	2
Bigeminy	6	3
Ventricular tachycardia	3	3

Discussion

Silent myocardial ischemia has been recognized since early this century as 'angina sine dolore' [6]. It occurs in the presence of coronary artery disease where there is objective evidence of myocardial ischemia without symptoms. Such evidence usually includes the presence of S-T segment deviation detected during either exercise or ambulatory electrocardiographic monitoring. In this study, we found silent myocardial ischemia in 44.5 per cent

of our patients. Patients with and without episodes of SMI had comparable degree of predialysis hematocrit, serum sodium, potassium, calcium, phosphorous, urea and creatinine values.

There was no significant difference in the degree of LVH or left ventricular dysfunction between the two groups. However, a significantly greater number of patients with SMI had underlying hypertension, diabetes mellitus and coronary artery disease. This suggests that in predisposed patients HD precipitates SMI. But the factors which induce SMI could not be identified in our study. Apart from myocardial ischemia the ST segment deviations have been attributed to repolarization abnormalities in patients with LVH [7], and to changes in the plasma concentrations of potassium [8] or even to the dialysis itself [9,10].

Kremastinos et al [3] showed that their patients on chronic hemodialysis with Holter evidence of SMI had insignificant coronary artery lesions on angiography and suggested the term 'equivalent SMI' to describe these changes, since none of their patients had proven coronary artery disease. Recently hypomagnesemia has been implicated in dialysis induced SMI [11]. We, however, did not measure serum magnesium levels before or after dialysis.

In our study, we could not attach any clinical significance to these episodes of SMI. No patient had any evidence of angina or hypotension during HD. We however did not record the blood pressures continuously and may have missed transient episodes of hypotension. Ventricular arrhythmias occurred with equal frequency in patients with and without ST segment changes.

In conclusion, the results of this study indicate that ECG changes compatible with silent myocardial ischemia are known to occur in patients with chronic renal failure on HD. These changes have

no clinical significance. The probable pathogenetic mechanism postulated is the production of asymptomatic coronary artery spasm leading to regional myocardial dysfunction, the nature and long term consequences of which are unknown.

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