Serum myeloperoxidase, CPK, CK-MB, and cTnI levels in early diagnosis of myocardial ischemia during ERCP; Once or repeated once?

To the Editor,

In the 2014 (25) issue of Turkish Journal of Gastroenterology, Yıldız et al. (1) reported that the increase in troponin levels during ERCP may show cardiac damage. Patients undergoing ERCP, who were diagnosed in advance of having ischemic cardiac problems (n=48) and without cardiac problems (n=76), were recruited in this study. Before and after ERCP, ECG, blood pressure, heart rate, oxygen saturation levels, MPO, CPK, CK-MB, and cTnI levels were evaluated in both groups. The effectiveness of these parameters in demonstrating myocardial ischemia during ERCP was investigated. While there were no ECG changes in patients without coronary history, 11 patients (22.9%) had ECG changes that may indicate myocardial ischemia in the ischemic group during ERCP.

This is a well designed and an observational study. There are contradictory results in silent myocardial ischemia during ERCP. While some publications indicate that cardiac damage is rare during ERCP (2%) (2,3), some studies indicate otherwise (10%-18%) (4,5).

In ERCP patients who were diagnosed in advance of having ischemic cardiac problems, post-ERCP ECG findings have been mentioned, but the pre-existing or newly emerged ECG findings were not specified in this study. In addition, how long the ST changes continued was not specified. Therefore, the recorded ECG changes are not clear, depending on the post-ERCP cardiac injury.

Johnston et al. (4) reported that in 9 patients (n: 41) who had ECG changes during ERCP, significant ECG changes were observed in 4 patients. However, there were significant ECG changes in only 1 of 9 patients who had cardiac history. In 3 patients, despite the absence of cardiac history, significant ECG changes were observed (4). In another study involving 71 patients, Lee et al. reported that 9 patients (5.2%) without cardiac history developed ischemic ECG changes (5). In this study, in patients without cardiac history, lack of ECG changes is inconsistent with the literature cited above.

These criticisms in the last two paragraphs could be prevented in the following way, like Lee et al. did (4): 30 minutes before ERCP and post-ERCP for 24 hours, Holter ECG application could be made.

In a study, along 8 hours after ischemia, followed by measurements at least 3 times with 3-hour intervals, cTnI was seen as a highly effective test (6). Cullen et al. reported that highly sensitive cTnI measurements were found to be effective in 2 hours after ischemia (7). Another point is that the Siemens cTnI kit used in this study, in cardiac injury except for MI, does not rise significantly (8). Finally, if we will detect the presence of cardiac damage that occurred during the ERCP, cTnI changes that occurred pre- and post-ERCP must be demonstrated. According to the literature, cTnI is anticipated to increase 30-42% in cardiac injury (9,10). However, the authors measured cTnI levels only once and with standard methodology.

In summary, if we evaluate cardiac damage during ERCP, we need more detailed cardiological data.

Conflict of Interest: No conflict of interest was declared by the authors.

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Author's Reply

To the Editor,

First of all, we are grateful to Alper and Arabul for their critical reading of our study. Early detection of myocardial ischemia with ECG changes is, indeed, not feasible, because some changes may happen in ERCP without myocardial ischemia evidenced by highly accurate methods as serum cTnI measurement which was the case in our study. On the other hand myocaridal ischemia may occur without early significant changes in ECG. Besides, ECG interpretation in the critically ill patients for the presence of myocardial ischemia or infarction showed moderate reliability at best; however, knowledge of the patient's troponin values increased the reliability for all studied ECG changes and resulted in a statistically significant increase in the interrater reliability for diagnosing myocardial ischemia or infarction (1). Furhtermore, a study for evaluation of reliability of the exercise ECG in detecting silent ischemia in patients with prior myocardial infarction (n:116) was also supported this reality; 3 (43%) of 7 patients who showed chest pain without ECG change had abnormal redistribution score (2). Pre and post ERCP ECG changes were being specified in the Table 1. Duration of ST changes was not mentioned in the manuscript because of none of our study patient progressed to myocardial infaction, and those changes were mostly temporary. We, actually, measured serum cTnI levels, before and after 2 hours of procedure and we believe that two hours is a reasonable time after procedure to detect myocardial injury. Correct timing of cTnI test is still a controversial issue and it seems that it would continue to be so until an excellent generation of test is found (3,4).

In our hospital, sedation for all the ERCP procedures are managed by an anesthesia team and pre-procedural evaluation is done by them as well. In some patients we prefer general anesthesia. This approach may have a positive impact on untoward events like myocardial injury, although it seems to be uncommon according to the results of our study.

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