DOES SHOCK WAVE LITHOTRIPSY OF RENAL STONES CAUSE CARDIAC MUSCLE INJURY?
A TROTONIN I-BASED STUDY
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ABSTRACT

Objectives. To investigate whether shock wave lithotripsy (SWL) causes cardiac muscle injury that alters the levels of troponin I plasma, a cardio-specific enzyme shown to be useful in diagnosing cardiac muscle injury because of its high specificity.

Methods. Patients treated by SWL for renal stones participated in the study. They had undergone a baseline 12-lead electrocardiogram (ECG) a few days earlier. One day after SWL, they were queried about any chest discomfort, blood was drawn for evaluation of troponin I and creatine kinase with isoenzymes (CK-MB), and an ECG was carried out.

Results. Thirty-two patients (21 men and 11 women, mean age ± SD 51.0 ± 10.6 years) comprised the study group. Fourteen SWL treatments were on the right side and 18 on the left. The mean number of shock waves was 2859 ± 202. The mean time to evaluation after SWL was 22.3 ± 1.3 hours. None of the patients reported chest discomfort. The mean value was 6.6 ± 9.2 mU/mL for CK-MB and 0.02 ± 0.04 ng/mL for troponin I. No ECG changes suggestive of myocardial injury were evident. None of the 5 patients who experienced ventricular extrasystoles during SWL had any evidence of cardiac muscle injury.

Conclusions. We evaluated the symptoms, perioperative ECG changes, and cardiac troponin I and CK-MB plasma levels in patients who underwent SWL for renal stones and did not identify any myocardial damage. Troponin I plasma levels were not elevated after this procedure and, therefore, remain suitable for evaluation of patients complaining of chest pain after SWL. 


After shock wave lithotripsy (SWL), patients may present to the emergency department with chest discomfort suggestive of acute myocardial infarct that is not necessarily related to the SWL procedure. A substantial number of patients with an acute myocardial infarct may not have recognizable symptoms or specific changes on their electrocardiograms (ECGs). For them, plasma biochemical markers of myocardial damage are essential for establishing the diagnosis of myocardial infarction. Plasma troponin I, a biochemical marker of myocardial damage, was shown to be valuable in diagnosing cardiac muscle injury because of its high specificity. During SWL of renal stones, the shock wave may pass over the myocardium and, as a consequence, may cause ventricular premature contractions when applied in a non-heart rate-synchronized mode. Therefore, the question arises as to whether subclinical cardiac muscle injury that alters the patient's troponin I plasma level might occur during SWL. The clinical merit of this information is apparent when patients who have undergone SWL procedures are evaluated afterward in the emergency department for chest discomfort or pain suggestive of an acute coronary event. Our study was designed to address the question of whether troponin I plasma levels are elevated after SWL of renal stones as the result of heart muscle injury inflicted by the procedure.

MATERIAL AND METHODS

PATIENT POPULATION
Consecutive patients with radiopaque renal stones who underwent SWL in our department were eligible for the study. Those with impaired liver (ie, conjugated bilirubin concentration greater than 60 mg/dL and unconjugated bilirubin greater
than 20 mg/dL) or renal function (ie, plasma level of creatinine greater than 1.4 mg/dL or blood urea nitrogen greater than 25 mg/dL) were excluded, as were patients with stones in their ureters. Six of the study patients had a history of ischemic heart disease. All suitable candidates signed an informed consent form before participating in this study, which was approved by the Tel Aviv Medical Center Helsinki committee.

**SWL Procedure**

The SWL treatments were conducted using the Econolith 2000 (Medispec, Israel) under fluoroscopic guidance for stone location. The tubeless lithotriptor, which was used for all the treatments, had an electrohydraulic (spark plug) energy source. The ellipsoid aperture was 17.6 cm in diameter, with a focal distance of 13.5 cm and a peak pressure at the focal point of 360 to 1000 bar. The focal zone size of the lithotriptor we used was 13 × 58 mm. Patients were anesthetized with fentanyl and propofol infusion, and a laryngeal mask with spontaneous breathing was kept in place. Each patient was connected to an ECG monitor throughout the procedure. Shock waves were discharged at a fixed rate of 90 per minute without synchronization to the heart rate. For patients in whom cardiac arrhythmias were observed, the treatment mode was switched to synchronization of triggering the release of the shock waves with the refractory phase of the heart cycle. All study participants were treated with at least 2500 shock waves and were discharged home within a few hours after the procedure.

**Study Protocol**

All patients underwent a baseline 12-lead ECG 1 to 7 days before undergoing SWL. One day after SWL, each patient was examined and queried about any discomfort in the chest area. Blood was drawn to determine the levels of troponin I and creatine kinase (CK) with isoenzymes (CK-MB). A follow-up 12-lead ECG was obtained and compared with the baseline ECG. Cardiac troponin I and CK-MB plasma levels were measured 5 minutes before the SWL treatment in the first 14 patients.

**CK-MB Analysis**

The immuno-inhibition assay for the quantitative in vitro determination of the MB isoenzyme of CK in human plasma was carried out on automated clinical chemistry analyzers (Roche Diagnostics, Indianapolis, Ind). The upper value of normal was set at 16 mU/mL. CK-MB myocardial type. Human CK-MB is composed of two subunits, CK-M and CK-B, both of which have an active site. Using polyclonal antibody to CK-M, the activity of the CK-M subunits is almost totally inhibited without affecting the

**Troponin I Analysis**

The AxSYM system (Abbott Laboratories, Diagnostics Division, Abbott Park, Ill), a microparticle enzyme immunoassay for the quantitative determination of cardiac troponin I in human plasma, was used. The upper value of normal was considered as 2 ng/mL.

With a sample size of 32 patients, the rate of pathologic findings was 10% or more (ie, 3 patients with a troponin I level greater than 2 ng/mL), we could have detected it with an alpha of 0.05 and a power of 87.5%.

**RESULTS**

Thirty-two patients (21 men and 11 women, mean age ± SD 51.0 ± 10.6 years, range 28 to 82) comprised the study group. Fourteen treatments were on the right side and 18 on the left. The mean number of shock waves was 2859 ± 202 (range 2500 to 3000). The mean number of hours that passed after SWL until the evaluation was 22.3 ± 1.3 (range 20 to 24). None of the patients complained of chest discomfort. As expected, these plasma levels were within normal limits in all 14 patients in whom cardiac troponin I and CK-MB levels were measured 5 minutes before SWL treatment. After SWL, the mean CK-MB level for the whole group was 6.6 ± 9.2 mU/mL (range 0.0 to 120.0), and the mean troponin I level was 0.02 ± 0.04 ng/mL (range 0.0 to 0.2). In 3 patients, the total CK was elevated to greater than 200 mU/mL but the CK-MB level for each of them was less than 3%.

No ECG changes suggestive of myocardial injury were detected after any of the SWL procedures. Five patients had ventricular extrasystoles during SWL, and the mode of delivery for them was switched to that of synchronization, triggering the release of the shock waves with the refractory phase of the heart cycle. None of these patients had any evidence indicative of cardiac muscle injury on their ECGs or blood tests. None of the 6 study patients with a history of ischemic heart disease had any evidence indicative of cardiac muscle injury on their ECGs or blood tests.

**COMMENT**

A precise diagnosis of acute myocardial infarction in patients presenting with chest discomfort after SWL has clinical consequence. Administration of anticoagulation agents to these patients may cause severe complications, such as massive bleeding. Katz et al.\(^5\) reported a patient who had had an acute myocardial infarction after SWL and was treated with anticoagulation and emergency coronary angioplasty; this patient subsequently developed life-threatening retroperitoneal hemorrhage. Patients evaluated in the emergency department with a suspicion of an acute coronary syndrome constitute a diagnostic, therapeutic, and prognostic challenge for the treating physician. As noted, a substantial number of these patients may have no recognizable symptoms\(^1\) nor any specific changes on their ECGs.\(^2\) Biochemical markers of myocardial damage, such as CK-MB, are essential for diagnosis, and troponin T and troponin I measurements have been shown to be especially valuable for early risk stratification and selection of treatment in these patients.\(^6\)

CK is a dimeric enzyme occurring in four different forms: a mitochondrial isoenzyme and the cytosolic isoenzyme muscle type, the brain type, and the CK-MB myocardial type. Human CK-MB is composed of two subunits, CK-M and CK-B, both of which have an active site. Using polyclonal antibody to CK-M, the activity of the CK-M subunits is almost totally inhibited without affecting the
CK-B subunits. The remaining CK-B activity, corresponding to one half of the CK-MB activity, is determined by the CK NAG method. From the measured CK-B activity, the catalytic activity of CK-MB is calculated by multiplying the results by two.\(^7\) CK-MB begins to rise 4 to 6 hours after myocardial injury; it reaches peak activity at 18 to 24 hours and then returns to normal levels by 36 to 48 hours after the event.\(^8\)

Troponin I is the regulatory subunit of the troponin complex associated with the actin thin filament within muscle cells. In conjunction with other troponins (C and T), it plays an integral role in the regulation of muscle contraction.\(^9\) Troponin I rises at 4 to 6 hours after myocardial injury and remains elevated for up to 10 days afterward.\(^10\)

Troponin I has become the standard tool in the evaluation of patients with cardiac muscle injury, because it is a sensitive marker of this type of injury and because it may rise and still be measured up to 10 days after the occurrence of such an event even after apparently minor insults.

Measuring the plasma level of troponin I is a well-accepted method for evaluating patients with nonischemic cardiac muscle injury, such as myocardial trauma. Because myocardial trauma has been described during gastroesophageal reflux laparoscopic surgery owing to the associated proximity of cardiac structures, Boccara et al.\(^11\) used ECG, cardiac troponin I, and CK-MB enzyme changes to evaluate the influence of this surgical procedure on the myocardium. Bertinchant et al.\(^12\) investigated stable patients after blunt chest trauma using cardiac troponin I and cardiac troponin T. The percentage of patients with elevated circulating cardiac troponin I and cardiac troponin T (0.1 \( \mu \)g/L or greater) was significantly greater in patients with myocardial contusion. To assess the troponin plasma level as an expression of myocardial injury, we used the parameters of physical symptoms, ECG, and plasma troponin I and CK-MB levels in a similar fashion to that of Boccara et al.\(^11\) It should be noted, however, that troponin I plasma levels may be elevated without clinical myocardial damage (ie, after ultra-endurance exercise), as was demonstrated in well-trained cyclists.\(^13\)

In our study, none of the parameters we used to assess cardiac muscle injury demonstrated any pathologic findings suggestive of myocardial injury subsequent to SWL. Our results are similar to those reported by Parr et al.\(^14\) Because shock waves during SWL may pass throughout the heart, those investigators explored the issue of cardiac muscle injury during the procedure in the early days of SWL. They evaluated 114 consecutive patients undergoing extracorporeal SWL by testing serial CK with isoenzymes and serial ECGs. Despite elevations in CK levels, no CK-MB isoenzyme fraction was detected in any of their patients. Although there were some random ECG changes before and after SWL, none appeared to be directly related to the procedure. In our current study, we found no changes between the pre and postprocedure ECGs. Given the reported results of Parr et al.,\(^14\) our findings are not surprising, but rather confirmatory.

It should be noted that our findings are specific to the lithotripter and treatment protocol we used. Additional studies on other types and models may be needed to ensure that no differences exist among lithotriptors regarding cardiac muscle injury.

**CONCLUSIONS**

We evaluated the symptoms, perioperative ECG changes, and specific cardiac troponin I and CK-MB levels and did not detect any clinical or subclinical myocardial damage among our study patients who underwent SWL for renal stones. We conclude that the troponin I plasma level is not elevated after this procedure and that it is, therefore, suitable for use in the evaluation of patients who complain of chest pain after SWL.

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**REFERENCES**


