

## Increased platelet serotonin levels in hypertensive patients already taking antihypertensive drugs

### *Antihipertansif tedavi alan hastalarda artmış trombosit serotonin seviyeleri*

Serotonin (5'-hydroxy tryptamine; 5-HT) is a bioamine, which is synthesized from L-tryptophan mainly in enterochromaffin cells of the gastrointestinal tract and central nervous system. It has been implicated in various disorders including pulmonary hypertension and essential hypertension (1). However, there is paucity of data about the effect of antihypertensive therapy on platelet serotonin levels.

Ninety-one patients with previous diagnosis of hypertension and already taking antihypertensive drugs were included in the study. We further subdivided these patients into "uncontrolled" (Group A, n=44, mean age: 48±10 years, male/female ratio: 26/18) and controlled groups (Group B, n=47, mean age: 51±13 years, male/female ratio: 29/18) according to whether their average 24-hour blood pressure value is higher (uncontrolled group) or lower (controlled) than 135/85 mmHg. Thirty-three healthy volunteers were also included in the study as Group C (mean age: 46±12 years, male/female ratio: 19/14). The platelet serotonin levels were determined in venous blood samples of the participants by using a high performance liquid chromatography system (Agilent 1100 Series; CA, USA). Continuous variables were expressed as mean ± 1 SD. The statistical comparisons among groups were done by one-way ANOVA test. Chi-square test was used for comparison of categorical data. The significance of p value was set at 0.05.

There were no statistically significant differences among groups in terms of age, gender distribution, weight and height. Antihypertensive drug use in Group A and B patients was as following: diuretics (23% vs. 19%, p=0.186), calcium channel blocker (20% vs. 28%, p=0.142), angiotensin converting enzyme inhibitors (30% vs. 34%, p=0.161), angiotensin II receptor blockers (20% vs. 17%, p=0.194) and beta-blockers (6% vs. 4%, p=0.308). Our results showed that although mean platelet 5-HT concentrations did not differ significantly between Group A (5.9±4 nmol/10<sup>9</sup>) and group B (6.2±2.9 nmol/10<sup>9</sup>), control patients had significantly lower mean platelet 5-HT concentration (1.4±0.3 nmol/10<sup>9</sup>) than patients in Group A and B (p<0.001 for both). Contrary to our results, platelet serotonin uptake and consequent platelet serotonin levels have been found to be decreased in essential hypertension (2, 3). We thought that the presence of antihypertensive therapy may be the underlying cause for high platelet serotonin levels in our study. Indeed, antihypertensive with isradipine increased platelet serotonin content (4). Such an effect can consequently decrease plasma serotonin levels as previously shown by Huang et al. in 68% of their hypertensive patients treated with quinapril (5).

In conclusion, antihypertensive therapy was associated with higher platelet serotonin levels in hypertensive patients than in control subjects. However, further controlled trials are needed for both confirmation and evaluation of our results.

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## Acute stress-induced late drug-eluting stent thrombosis leading to hyperacute myocardial infarction

### *Hiperakut miyokard infarktüsüne yol açan akut stres ilişkili geç ilaç kaplı stent trombozu*

Drug-eluting stents (DES) have been proven to reduce restenosis and reintervention compared with bare-metal stents (BMS). However, a number of analyses have recently shown increased rates of late stent thrombosis in patients with DES. The exact mechanisms leading to stent thrombosis remain unclear. It can occur at any point during the follow-up period, not necessarily triggered by interruption of dual antiplatelet therapy.

A 55-year-old man presented with acute onset of central chest pain. He had undergone percutaneous coronary intervention (PCI) with the DES deployed in the proximal left anterior descending (LAD) and right coronary artery in August 2007, and had not had any health problems or cardiac complaint since then. He was on the following medications: aspirin 150 mg/day, clopidogrel 75 mg/day, simvastatin 40 mg/day, lisinopril 10 mg/day, and metoprolol 50 mg/day. He was readmitted with acute chest pain in February, 2008. The chest pain was preceded by severe emotional distress following a car accident, where the patient ran into an elderly pedestrian and thought that he killed the man. This event caused extreme anxiety in the patient. Immediately afterwards, the central chest patient started. An electrocardiogram obtained at the ambulance revealed extensive anterior ST-segment elevation. Emergency coronary angiography revealed the complete stent occlusion

of the proximal LAD and PCI with implantation of one DES was successfully performed. The patient was discharged in stable condition and prescribed the same medical regimen as described above.

Significant adverse effects of acute emotional stress on the heart can be divided broadly into 3 areas: left ventricular contractile dysfunction (stress cardiomyopathy or Takotsubo cardiomyopathy), acute myocardial ischemia/infarction (AMI), or disturbances of cardiac rhythm (1). AMI can be triggered by a number of exogenous as well as endogenous triggers. Physical exertion and episodes of anger occurring within 1-2 h of onset of symptoms have been identified as triggers of AMI. The wake-up time, Mondays, winter season, physical exertion, emotional upset, overeating, lack of sleep, cocaine, marijuana, anger, sexual activity, stressful soccer matches, blizzards, earthquakes and terrorist attack are some of the previously documented triggers in vulnerable patients (2). Strike et al. reported that when anger was the trigger the patients were more likely to present with ST-elevation myocardial infarction rather than non-ST-elevation myocardial infarction or unstable angina (3).

The cardiovascular effects of stress are executed through the brain's neurochemical pathways associated with fear and anxiety. Several regional central nervous system centers are neuroanatomically and functionally interconnected to form a network that initiates and shapes sympathoadrenal responses. The physical or emotional stress can trigger the onset of cardiac events, perhaps by stimulating the release of stress hormones (catecholamines and corticosteroids) and hypercoagulability factors, and precipitating sympathetic activity leading to a number of hemodynamic changes, including an increase in heart rate, blood pressure, vascular resistance, and ventricular contractility. These factors can increase shear stress of blood against a vulnerable atherosclerotic plaque, contributing to rupture of the plaque and subsequent myocardial infarction (4). In the presented case, we considered that the triggering factor was acute mental stress related to intense emotional reaction to a near-fatal car accident. It could be that gradual restenosis preceded acute thrombotic events; however, it can not be determined if the in-stent restenosis occurred gradually, or at the time of the event (5). The future multidisciplinary evaluation of patients with stress-induced cardiovascular events might clarify the mechanisms of emotional triggering.

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## Harvesting internal mammarian artery by using ultrasound harmonic scalpel: experience of 154 cases

*Harmonik skalpel kullanılarak internal mammariyan arter hazırlanması: 154 vakalık deneyim*

Early and late outcomes of eligible trials with large number of patients clearly expose that internal mammarian artery (IMA) is the most ideal coronary artery graft due to patency rates and effects on the cardiac functions. This valuable artery must be harvested by using minimal invasive approach. An injury to the IMA endothelium can activate a coagulation cascade, which can result in thrombus formation thus leading to early graft failure. Moreover, the damage to the endothelium can also promote the atherosclerotic process, thus eventually leading to the long-term development of graft stenosis or occlusion (1). There are lots of publications and articles about harvesting IMA by using different approaches, techniques, devices (2-5).

We aimed to share our experience of harvesting IMA by using ultrasound harmonic scalpel (Harmonic Scalpel, Ethicon, Cincinnati, USA) in 158 patients for a time period between September 2005 and September 2006. Overall, 58 of these patients were female and mean age was 63 years (36-83 years). We logged all the peroperative and postoperative data of our patients. Mean number of sponges that we used during harvesting IMA was 1.4, mean time for graft preparation was 8 minutes (3-12 min). All the harvested IMA's were patent and used for left anterior descending (LAD) anastomoses. Mean number of hemoclips that were expended for the branches of IMA was 1.2 (0-5). Mean number of coronary bypass grafts was 2.3 and also mean postoperative drainage volume for these patients was 654 ml per day (375-1350 ml). We had two postoperative revisions for bleeding. One was for focal bleeding point on the residual thymic tissue, and the other we could not determine the reason for bleeding in other case of revision. We lost only one patient because of acute renal failure on his 15<sup>th</sup> postoperative day. All the other patients were discharged with full recovery.

Ultrasonic scalpel can cut and coagulate tissues by making denaturation. Unlike electrocautery Harmonic Scalpel can let surgeons to work closer than 1 mm to IMA for 3-4 seconds without giving any harm or causing dissection (1-3). In fact; no endothelial damage was recorded even if the device touched directly to the artery (2-5).

As a conclusion; we recommend to use harmonic scalpel for all the cardiac surgery training centers, during the preparation of the arterial graft because of its advantages over electrocautery such as less injury, causing less bleeding, shorter time for harvesting, using fewer hemoclips and allowing to work closely to the artery.