

TRAVMATİK OLMAYAN TALAR OSTEOKONDRAL DEFECTDE İSKEMİK ETYOLOJİNİN DEĞERLENDİRİLMESİ

Evaluation of Ischemic Etiology in Non Traumatic Talar Osteochondral Defect

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ÖZET

Amaç: Bu çalışmada travmatik olmayan osteokondral defektler ile serum ortalama trombosit hacmi arasındaki ilişkiyi incelemeyi amaçladık

Gereç ve yöntemler: Ayak bileği MR çekimi yapılmış hastaları retrospektif olarak taradık. Talar osteokondral defekti (OKD) olan 15 hasta ile OKD'si olmayan 11 hasta çalışmaya dahil edildi. OKD'si olan hastalardan travma anamnezis olanlar çalışmaya dahil edilmedi. Total trombosit sayısı (TS), ortalama trombosit hacmi (OTH) ve trombosit dağılım genişliğini (TDG) içeren laboratuvar testleri hasta kayıt sisteminden elde edildi. Bu değerler hasta ve kontrol grup arasında karşılaştırıldı.

Bulgular: TS, OTH ve TDG değerlerini hepsi iki grupta da normal sınırlarda olup iki grup arasında anlamlı farklılık saptanmadı.

Sonuç: Travmatik olmayan talar OKD'lerde değişik etyolojik sebepler tanımlanmıştır. Bunlardan biri olan iskemi travmatik olmayan OKD'nin nedenlerinden biri olmayabilir.

Anahtar kelimeler: Osteokondral defekt; Ortalama trombosit hacmi; Trombosit dağılım genişliği, Trombosit sayısı

ABSTRACT

Aim: We aimed in this study to evaluate the relationship between serum mean platelet volume and non traumatic talar osteochondral defects .

Materials and methods: We retrospectively analyzed the patients who underwent ankle MRI. 15 patients with talar osteochondral defect, 11 patients with no talar osteochondral defects were included in the study. The patients with osteochondral defect who had history of trauma were excluded from the study. Laboratory tests including serum mean platelet volume (MPV), platelet distribution width (PDW) and total platelet (PLT) count compared between the patients and control group.

Results: MPV, PDW and total platelet count were all in normal range both in patients and control group. We revealed no significant difference between the patients and control group

Conclusion: Different etiologic factors are described in the non traumatic talar OCDs. Ischemia, which is the one of the estimated factors, may be less likely to predispose to non traumatic talar OCD.

Keywords: Osteochondral defect; Mean platelet volume; Platelet width distribution, Platelet count

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INTRODUCTION

Increased mean platelet volume (MPV) has been defined as an independent risk factor in the development of thromboembolism (1). There are many studies investigating the role of MPV in occlusive diseases like stroke (2-4) and coronary vascular disease (5-7). Several possible etiological factors for talar osteochondral defect (OCD) have been described including traumatic, embolic, hereditary, endocrine, developmental and idiopathic (8,9). We aimed in this study to evaluate the role of MPV in non traumatic talar OCDs which can be ischemic in etiology.

MATERIALS AND METHODS

The study was approved by the local ethics committee. Between september 2014 and November 2015 , the patients who underwent ankle and hip MRI were analyzed retrospectively. 15 patients (5 men, 10 women, mean age $42,40 \pm 13,05$) with talar OCD were included in the study. The patients with OCD who had history of trauma were excluded from the study. Who had no laboratory tests including serum platelet count, MPV and PDW levels within 2 weeks earlier or later from the MR examination were excluded from the study. A control grup for OCD that included 11 patients (1 men, 10 women, mean age $52,09 \pm 13,78$) was selected from patients who underwent ankle MRI for any reason which had no talar OCD. Figure 1 shows the T1W and figure 2 shows the T2W sagittal images of a patient with stage 4 talar OCD.



Fig. 1 T1W sagittal image shows the hypointense stage 4 osteochondral defect and surrounding hypointense line.

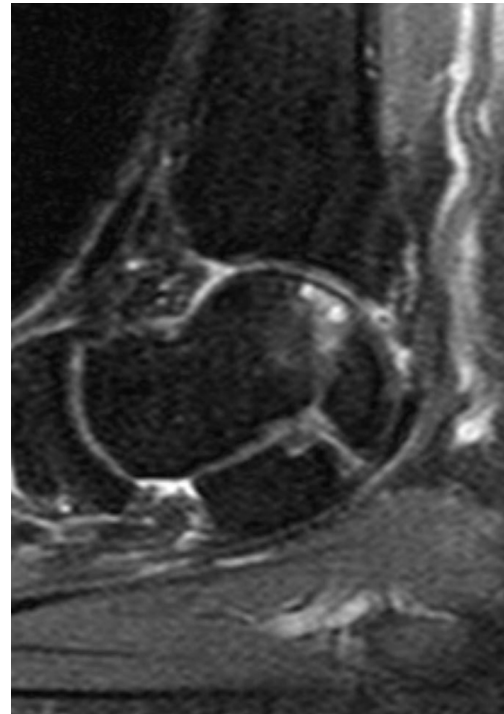


Fig. 2 T2W sagittal image shows the hyperintense stage 4 osteochondral defect of talus.

MR examinations were re-analyzed by two radiologists who were experienced in musculoskeletal imaging and who were blinded to clinical and laboratory findings of the patients. Laboratory findings including MPV, PDW and total PLT count were obtained from the hospital information system. All of the MR images were obtained using a 1,5-Tesla MRI scanner (Philips Achieveva Intera Release, Einthoven, Netherlands). Sagittal T1 (TR:340, TE: 9, FOV:200x200, Matrix: 256x128), axial T1 (TR: 428, TE: 10, FOV:110x160, Matrix:256x86), sagittal T2 (TR: 2470, TE: 84, FOV:200x200, Matrix:256x128), axial T2 (TR: 661, TE: 23, FOV:143x180, Matrix:256x102) and coronal PD (TR: 1520, TE: 28, FOV:125x200, Matrix:256x110) sequences were acquired for each patient. Serum total PLT count, MPV and PDW values were compared between the patients and control groups. Statistical analyses were performed using the SPSS software version 20. The variables were investigated using analytic methods (Kolmogorov-Smirnov test) to determine whether or not they are normally distributed. Descriptive analyses were presented using means and standard deviations for normally distributed variables. Since the MPV, PDW and PLT count values were normally distributed; the Student's t-test was used to compare these parameters between the patient and control groups. A p-value of less than 0.05 was considered to show a statistically significant result.

RESULTS

The total PLT count, MPV values and PDW values were $256,87 \pm 65,35$ cells/ μ L, $9,04 \pm 1,19$ (fL) and $16,39 \pm 1,03$ (fL) respectively in patients with OCD and $281,18 \pm 106,131$ cells/ μ L, $9,55 \pm 1,08$ (fL) and $15,85 \pm 2,32$ (fL) respectively in control group. There was no significant difference between the patients and control group. (p for MPV=0,279, p for PDW= 0,423, p for PLT = 0,477)

DISCUSSION

Plug formation following the vessel wall injury is the main function of platelets (10).MPV which is the most commonly used measure of platelet size (11), is an indicator for platelet reactivity and function (12). Small platelets have lower functional capabilities. Large

platelets produce more thromboxane A2 (13,14) and have more fibrinogen receptors (15). Elevated MPV is associated with increased thromboxane synthesis, platelet aggregation, and increased expression of adhesion molecules (11). All of these information shows that mean platelet volume (MPV) is an important risk factor for the development of atherothrombosis and embolism (16). There are a lot of studies investigating the role of MPV in vascular occlusive diseases (2-7). Several possible aetiological factors for talar OCD have been described including traumatic, embolic, hereditary, endocrine, developmental and idiopathic (8,9). The majority of the cases are associated with previous trauma including involves repetitive and prolonged joint overloading or a sudden impact from high compressive stress (17,18). Ischaemia, subsequent necrosis and possibly genetics are aetiologic factors in non-traumatic OCDs (19). A detailed anamnesis was obtained from all patients whether or not they had any trauma including ankle. To evaluate the ischemia as an etiologic factor in non traumatic OCDs, we excluded all of the patients with history trauma. We expected MPV values to be higher in patients compared with the control group. However, we found no significant difference in total PLT count, MPV values and PDW values between the patients and control group.

In conclusion, we think that ischemia is less likely an etiologic factor in idiopathic OCDs. Patients probably do not remember the trauma, especially the repetitive microtrauma. In the other hand ischemia may not be related with thrombus formation. We believe that larger studies are needed to investigate the role of ischemia in the etiology of non traumatic OCDs.

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