

showed statistical differences between patient with UA and those with MI ($P=0.042$ and $P=0.031$) respectively unlike other parameters (platelets count or PDW) ($P=0.703$ and $P=0.094$) (Table 2).

It is found also that MPV will exceed 11.6 fl and 12.10 fl at percentile 95 in case of UA and MI respectively and similarly P-LCR will exceed 37.66 and 41.20 at percentile 95 in the above two groups respectively which may indicate a higher level of activity. (Table 3)

There were no correlation found between MPV and other platelets indices with existing past history of stable angina as well as other risk factors for acute coronary syndrome ($P=0.811$) i.e. these PVI did not altered significantly with these risk factors and their difference is related directly to acute events.

Discussion

The findings indicate that increased platelet volume is associated with a higher risk of suffering an acute coronary event independent of the extent of a previous coronary artery disease (CAD). Percentile 95 value will indicate a higher risk of getting acute coronary event with being increased platelet volume and a higher percentage of large size cells independent of existence of other risk factors. Thus MPV and P-LCR above these percentile values may represent an independent risk factors for MI similar to other studies^(1, 2, 3), but there were no practical application of platelet count which had been demonstrated by Kilici-Cmur N. et al⁽²⁾.

The mechanism for an increased platelet volume are not well fully understood, possibly cytokines may trigger the production of larger more reactive platelet following platelet destruction in peripheral blood including interleukin-6 (IL-6)⁽¹⁴⁾,

although, it is not settled completely⁽¹⁾.

In this study we neglected the drugs used by patients as there are limited data about the effect of pharmacological therapy on platelet count and size. It has been proved previously that standard medical treatment for coronary diseases did not significantly change platelet markers⁽³⁾. In previous studies, an increased MPV was found to be associated with coronary artery disease^(10, 15, 16), UA^(9, 10), AMI^(1, 9) and even congestive heart failure⁽¹⁸⁾ as well as in cerebrovascular diseases (18) and this can be explained on base of increased platelet hyperactivity after erosion or rupture of atherosclerotic plaque leading to potentiated prothrombotic complication like MI or cerebrovascular events^(1, 6).

Large platelets that contain more dense granules are metabolically and enzymatically more active than small platelet with a higher thrombotic capacity (1) as they express higher levels of prothrombotic substances, thromboxane A₂, serotonin b, B-thromboglobulin and procoagulation surface protein such as P-selectin and glycoprotein IIIa⁽¹¹⁾. An increased MPV decreases the inhibitory effectiveness of PG I₂ on both platelet aggregation and the release reaction⁽¹⁹⁾. Higher levels of P-selectin was previously reported to associate with acute MI and its measurement was promising as predictors of vascular risk due to platelet aggregation⁽²⁰⁾.

The size of platelet has been found to associate with an increased number of megakaryocyte⁽³⁾. In agreement with Kilicli-Camur observation, we did not report a significant correlation between MPV and history of stable angina, and this is in contrast to others findings like Endler G. et al and Erne P. et al^(1, 17).