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ORIGINAL ARTICLE

Mean Platelet Volume: Is It a Predictive Parameter in The Diagnosis of Sudden Sensorineural Hearing Loss?

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Abstract The mean platelet volume (MPV) is associated with increased platelet reactivity and increased atherothrombosis. High MPV values are a risk factor for thrombotic vascular diseases. Our aim was to investigate whether a relationship exists between sudden sensorineural hearing loss (SSHL) and a high MPV value. The records of 46 patients who were admitted to the ENT (ear nose throat) ward for SSHL and received medical treatment and 46 patients in a control group were retrospectively screened. The correlation among the levels of the MPV, the number of platelets, and SSHL were evaluated in the two groups. The ages, genders, and the platelet count values of the patients showed a normal distribution in both groups. No significant difference was found for the MPV values between the groups. The MPV is not a predictive parameter in the diagnosis of SSHL.

Keywords Mean platelet volume · Sudden sensorineural hearing loss · Thrombotic vascular diseases

Introduction

Sudden sensorineural hearing loss (SSHL) is a clinical condition characterized by the acute onset of unilateral or bilateral hearing loss. It is commonly defined as sudden SSHL at least 30 dB or more across three contiguous frequencies in <3 days [1].

The incidence of SSHL has been reported to range from 5 to 20 cases per 100,000 persons. [2, 3] However, the exact incidence of SSHL is higher because a large portion of patients heal spontaneously without medical and surgical treatment [4–6].

One reason the vast majority of cases cannot be found of SSHL, and they are considered to be idiopathic. Different theories have been proposed to explain the etiology. These include, for example, bacterial, viral, and protozoan infections, vascular occlusion, mechanisms associated with the immune system, ototoxicity drugs (salicylates, aminoglycosides) and traumatic, vascular, neoplastic, and metabolic conditions [7–12].

Blood platelets, which are involved in hemostasis, are the smallest cells of the peripheral blood. They play a very significant role in the formation of thrombosis in the vessel. Platelets release mediators and substances, which are crucial for coagulation, inflammation, thrombosis, and atherosclerosis [13, 14]. Platelets aggregate with mediators that they release, and they form a hemostatic obturator and lead to aggregation. Platelets are heterogeneous in volume and density. The volume of platelets provides an objective parameter for the evaluation of the size of thrombocytes. The mean platelet volume (MPV) is a commonly used parameter to indicate the volume of platelets. This parameter is also a potential marker showing the function and activity of platelets. Large platelets are both more active metabolically and enzymatically and have more thrombotic potential [15, 16].

Large platelets also have a higher tendency for aggregation than small platelets. In light of this tendency, many studies have investigated the correlation between vascular ischemic events and the MPV and considered whether changes in platelet volume markers might be important

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prophylactically and diagnostically in thrombotic and prothrombotic cases. Those studies detected a correlation between clinical cases such as myocardial infarction (MI), stroke, and venous thromboembolism [17, 18]. and increased MPV measurements, and an increased MPV was stated to be an independent risk factor in MI and stroke [19–21].

Based on the idea that vascular ischemic events are associated with an increased MPV, we investigated whether there was a relationship between SSHL and MPV values.

Materials and Methods

This was a retrospective study involving two groups: an SSHL group and a control group. The SSHL group comprised 46 patients (21 females and 25 males between the ages of 16 and 73 years, with a mean age of 45.4 years) who were admitted to the ENT ward for SSHL and received medical treatment between January 2006 and December 2011.

Those with a history of otological surgery or previous ear problems or the head region a history of trauma, hematological, or endocrine disease were excluded from the study.

The control group comprised 46 healthy patients (22 females and 24 males aged between 8 and 79 years, with a mean age of 41.4 years) who had been admitted to the ENT polyclinic for a routine check-up examination and had no active symptoms, and their audiological findings were normal. The patients' files are analyzed retrospectively. Age, gender, MPV values, femtolitre (fL), and platelet counts ($\times 10^9$/L) were recorded for evaluation. The correlations among the levels of the MPV and the number of platelets were evaluated in the SSHL group and the control group.

Statistical Analysis

Data analyses were performed with the statistical package for the social sciences (SPSS for Windows, version 15.0) software. The distributions of the data were analyzed with the Shapiro–Wilk test. The independent sample T test comparison was used to analyze the (age, MPV levels, platelet count). In a normal distribution, the Mann–Whitney U test was used to compare the groups (gender). A level of <0.05 was considered significant.

Results

The SSHL group and the control group did not differ significantly in terms of age and gender. (Table 1). The platelet levels of the SSHL group and the control group did not differ significantly ($p = 0.270$) (Table 2). When the MPV levels in the SSHL and the control groups were compared, there was no statistically significant difference ($p = 0.113$) (Table 3).

Discussion

SSHL is a clinical condition that frequently occurs in ENT practice, and its etiology is still an enigma. Some have proposed that infarction may be an etiological source of sudden hearing loss [22], in addition to vascular ischemic events such as thromboembolic stroke and MI. The blood flow to the cochlea is usually through the labyrinthine artery, which is a terminal division of the anterioinferior cerebellar artery (AICA). Cochlear injury occurs when there is a reduction in blood flow arising from a vascular problem resulting from the small diameters and a lack of collateral blood supply. SSHL has also been reported to be associated with clinical conditions such as ischemic vascular disease, transient ischemic attacks, and amaurosis fugax [23]. For example, in a microscopic examination of the temporal bones of 12 ears with idiopathic SSHL, Vasama and Linthicum [12] found that SSHL appeared to have a viral origin rather than a vascular origin or a ruptured inner ear membrane origin for SSHL. Risk factors for ischemic vascular diseases such as smoking, hypertension (HT), and hyperlipoidemia have also been suggested to be involved in the development of SSHL in some studies [24, 25]. One study reported that the risk of stroke is 1.6 times more in patients with SSHL and that a significant number of strokes in these patients originated from the AICA [26].

The blood flow to the cochlea remains important in the etiology of SSHL. Poor vascular nutrition of the inner ear and an absence of collaterals predispose patients to ischemic events. Thrombosis, hemorrhage, and vasospasm are possible mechanisms leading to disruption of inner ear arterial flow.

On the other hand, our clinical observations and some clinical and experimental studies do not support a vascular etiology for the development of SSHL. For instance, many ENT physicians consider that thromboembolic events commonly occur in the elderly. However, this not the case

Table 1 Intergroup comparison of age and gender

	Study group	Control group	p
Age	45.39 ± 15.70	41.38 ± 16.70	0.229
Gender			0.708
Female	21 (46 %)	25 (54 %)	
Male	22 (48 %)	24 (52 %)	

Table 2 Intergroup comparison of platelet results, showing the median, minimum, and maximum values

	Study group	Control group	*p*
Platelet	243,000 (144,000–470,000)	275,000 (170,000–500,000)	0.270

Table 3 Intergroup comparison of the MPV results expressed as mean ± standard deviation, showing no significant difference between the groups

	Study group	Control group	*p*
MPV	8.25 ± 0.86	7.98 ± 0.87	0.113

with SSHL, where the condition occurs in a relatively younger healthy population. The most common mechanism of AICA infarction is either thrombotic occlusion or enhancement of the basilar artery plaque in the AICA and occlusion of the AICA orifice [27]. SSHL can develop in such cases, but other symptoms of cerebellar and brain involvement are expected, for example, ataxia, hypalgesia and, peripheral facial palsy. SSHL associated with the brainstem or cerebellar exposure is very rare [28, 29]. In normal physiological conditions, the blood flow to the inner ear (cochlea and vestibule) is through the labyrinthine artery, which is the end division of the AICA. An embolisation in blood flow here is expected to result in both cochlear and vestibular symptoms. However, contrary to expectations, vestibular symptoms accompany SSHL in only a very small number of patients [30].

In an experimental study, irreversible hearing loss developed one hour after occlusion of the blood flow [31]. In fact, a large number of patients with SSHL recover from hearing loss with treatment, and a significant number recover even without treatment. Indicators of a vascular response such as connective tissue deposits in the cochlea and new bone formation have been observed in some studies [32–34]. Fayez conducted a histopathological examination of the temporal bone of patients with experience of SSHL 22 years ago [35]. The analysis revealed no signs of bone neoformation anywhere in the inner ear and no signs of cochlear damage by vascular occlusion. In addition, the arterioles and the venules responsible for blood flow seemed intact. Studies have shown that higher MPV levels of platelet activity, including increased platelet aggregation, increased thromboxane synthesis and the release of b-thromboglobulin, as well as the expression of adhesion molecules [36–38].

MPV is a parameter that is routinely examined in complete blood cell. When compared with other platelet activity markers, MPV is a simple, cheap, and practical biomarker not requiring special equipment and laboratory conditions; it is also accepted as prognostically important for cardiovascular diseases [19].

There is no consensus on the treatment of SSHL because of the lack of a clear etiology. Tested in various treatment methods from the past to the present day. SSHL treatment can be effective once the pathogenesis is determined.

Conclusion

In our study, no significant difference was observed in MPV values between the control group and the patient group treated for SSHL. This supports the theory that there is no microvascular response in the etiology of SSHL.

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