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Platelet Membrane Proteins as Pain Biomarkers in Severe Dementia Patients

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Abstract

Pain is one of the most common health issues, and its assessment and treatment are heavily reliant on patient self-report. When obtaining a self-report is not possible, the therapeutic decision becomes more difficult and limited. The purpose of this study is to see if some membrane platelet proteins can help with pain characterization. We used 53 blood samples from palliative patients, 44 with non-oncological pain and nine without pain, to achieve this goal. When comparing patients with and without pain, we found a decrease in the percentage of platelets expressing and the levels of expression. Furthermore, an increase in the percentage of platelets expressing was observed in pain patients.

Keywords: Chronic pain • Pharmacology • Platelets • Biomarkers

Introduction

The International Association for the Study of Pain (IASP) defines pain as a "unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage". Because pain is multifactorial and involves multiple pathways, it is a multidimensional experience that can significantly impair an individual's quality of life. Pain is classified into three types: tissue damage, nerve damage, and altered pain modulation. Paincharacterization is critical for the correct approach for patients with chronic pain, beginning with pain intensity assessment. Pain multidimensional hetero-assessment scales can be used as an alternative, especially for dementia patients with and/or patients who cannot characterise their pain.

Preclinical and clinical studies have looked into the possibility that biomarkers could be used to identify and quantify pain. A preclinical study found that inflammatory and neuropathic pain have different biomarkers, but most studies found no correlation between pain duration or intensity. More research into pain biomarkers is needed to improve pain management practises and patient care, particularly for those with severe cognitive decline or dementia who are unable to express themselves. Platelet heterogeneity and subpopulations may indicate distinct biological roles for different platelet subpopulations and may be useful in assessing inherited or acquired platelet disorders as well as platelet function in health and disease [1].

Literature Review

Individual and clinical data, as well as peripheral blood samples, were collected from 53 palliative patients suffering from non-oncological diseases for this study. This is a non-interventional, observational, analytic, transversal study of chronic pain patients using medical and nursing records. The ethics committees of the University of Porto's Faculty of Medicine and the North Regional Health Administration of Portugal approved the research procedures, and the study was carried out in accordance with the Helsinki Declaration. Participants or their legal representatives gave informed consent for participation prior to enrollment. The international ethical guidelines of confidentiality, anonymity of personal data, and the abandonment option were followed.

We consulted the records in the individual clinical files to collect individual and clinical data, which was then recorded in a protected Microsoft Excel sheet. An alphanumeric code was used to identify each patient, keeping the identity private because only the researcher knows it. These electronic files will be deleted in accordance with the European General Data Protection Regulation following the completion of the study and publication of the results. The following variables were collected: age, gender, type and intensity of pain, opioid and other analgesics, such as nonsteroidal anti-inflammatory drugs and acetaminophen, and doses used. We also noted whether the patient was in pain at the time of blood collection.

Discussion

We cannot rely on self-report in patients with severe dementia, and characterising pain is difficult. Several preclinical and clinical studies were conducted to investigate the hypothesis that biomarkers could be used to identify and quantify pain. A preclinical study discovered that biomarkers for inflammatory and neuropathic pain differ. Further research yielded conflicting results. Cystatin C levels in cerebrospinal fluid, for example, appear to be a predictive marker for postherpetic neuralgia in varicella-zoster virus patients and a pain marker in women experiencing labour pain. It is not, however, related to pain duration or intensity. According to research into potential biomarkers for chest pain, cardiac markers used to aid in the diagnosis and prognosis of cardiac disease correlate with tissue damage rather than pain [2-4].

Platelet receptors are essential for platelet function because they either activate platelets or act as adhesion molecules interacting with damaged endothelium, other platelets, and leukocytes. Platelets play a role in inflammation, antimicrobial activity, angiogenesis, tumour growth, and metastasis, in addition to hemostasis. Platelets cannot perform these functions in the absence of their receptors. Integrins, leucine-rich repeats receptors, selectins, tetraspanins, transmembrane receptors, prostaglandin receptors, lipid receptors, immunoglobulin superfamily receptors, tyrosine kinase receptors, and other platelet receptors are examples of well-known platelet receptors. Glycoprotein is a cell surface protein that belongs to the class B scavenger receptor family. It is a multiligand pattern recognition receptor that interacts with a wide range of structurally dissimilar ligands, including long chain fatty acids [5].

Platelet surface receptors have also been measured as markers of platelet activation, and platelet activation is increased in dementia. Platelet expression and complex activation are both significantly increased in Alzheimer's disease

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patients. Platelet activation was found to be a marker of dementia, and an increase in complex expression in platelets was linked to a faster cognitive decline in AD. However, there is a severe scarcity of additional clinical studies. CD62p, another platelet receptor found on activated platelet membranes, promotes platelet adhesion and thrombin formation. CD62p levels in the blood increased in Alzheimer's disease patients, but there was no significant change in membrane-bound levels. The soluble form of CD62p was also significantly higher in HIV patients who were not on cART compared to those who were on cART and healthy control groups, implying a role in combination monitoring.

Conclusion

Furthermore, aside from the involvement of platelets in several diseases, we had no patients with autoimmune diseases such as inflammatory bowel disease, multiple sclerosis, psoriasis, or other autoimmune diseases. According to the literature, other diseases in these patients' known medical histories had no effect on platelet markers. This is one of the largest pain biomarker studies we are aware of, and it is the only one that compares patients with non-oncological pain to specific platelet biomarkers. However, the sample size is small, and more research into these markers is needed to confirm their viability as pain markers and as a marker of moderate-to-severe pain. Several preclinical and clinical studies have been conducted to investigate the hypothesis that biomarkers can be used to identify and quantify pain. A preclinical study discovered that inflammatory and neuropathic pain have distinct biomarkers. Further investigation yielded conflicting results. Cystatin C levels in cerebrospinal fluid, for example, appear to be a predictive marker for postherpetic neuralgia in patients with varicella-zoster virus and a pain marker in women experiencing labour pain. It is, however, unrelated to pain duration or intensity. Investigations into potential biomarkers for chest pain revealed that cardiac markers used to aid in the diagnosis and prognosis of cardiac disease correlate with tissue damage rather than pain. More research into pain biomarkers is needed to improve pain management practises.

Acknowledgement

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Conflict of Interest

There are no conflicts of interest by author.

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