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The influence of Tramadol on platelets´ function

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Background Tramadol is one of the major opioids and a ligand with weak μ-opioid receptor affinity. In addition there is also an analgesic mechanism via serotonin reuptake inhibition. Serotonin affects blood clotting via several mechanisms: first, platelets carry serotonin receptors, activation of which leads to enhancement of platelet aggregation triggered by other neurotransmitters such as adenosine diphosphate (ADP) and thrombin; and second, serotonin-mediated vasoconstriction reduces blood flow in smaller blood vessels and facilitates wound healing.

In recent years, various analgesics have been investigated for their potential to affect the coagulation cascade or platelet function. Alterations have been demonstrated for NSAIDs such as metamizole as well as acetaminophen and co-analgesics such as SSRIs. On the question of the influence of tramadol on platelet function, interestingly, only a few papers with opposite results can be found.

Methods The aim of our study is to quantify the effect of tramadol on platelet aggregation. To this end, the effect is demonstrated in an ex vivo study using optical aggregometry (LTA) on whole blood.

In addition, two further questions will be investigated: -A titration series will be used to attempt to establish a dose-response curve for the effect of tramadol on platelet function. - Tramadol is often co-administered with other drugs such as ibuprofen, novalgin, or SSRIs. A number of studies are being conducted to determine if these combinations alter the effect of tramadol on platelet function.

Due to the lack of studies to date, there is no indication of the magnitude of the effect, therefore, based on studies with comparable methodology, as well as the statistical limitations of the study methodology (standard deviation LTA, interindividual differences in platelet function), we plan to study 15 subjects.

Conclusion Our study is currently ongoing, we expect first results by approximately Q1 2023.