Multiplate can be used to detect increased platelet reactivity in dogs with diseases known to predispose for hypercoagulability and thrombosis

Marschner, Clara Büchner; Kristensen, Annemarie Thuri; Spodsberg, Eva; Jessen, Lisbeth Rem; Wiinberg, Bo

Published in:
22nd ECVIM-CA Congress

Publication date:
2012

Document version
Early version, also known as pre-print

Citation for published version (APA):
Marschner, C. B., Kristensen, A. T., Spodsberg, E., Jessen, L. R., & Wiinberg, B. (2012). Multiplate can be used to detect increased platelet reactivity in dogs with diseases known to predispose for hypercoagulability and thrombosis. In 22nd ECVIM-CA Congress
MULTIPLATE CAN BE USED TO DETECT INCREASED PLATELET REACTIVITY IN DOGS WITH DISEASES KNOWN TO PREDISPOSE FOR HYPERCOAGULABILITY AND THROMBOSIS.

CB Marschner, AT Kristensen, EH Spodsberg, LR Jessen, B Wiinberg, Faculty of Health and Medical Sciences, University of Copenhagen, Denmark, Novo Nordisk A/S, Maaloev, Denmark.

There are few diagnostic laboratory methods available for evaluation of platelet function and contribution to thrombotic events in the clinical setting. The impedance whole blood platelet aggregometer Multiplate® has recently become available. Although it is being marketed for monitoring effect of antiplatelet therapy, it can also be used for assessment of platelet aggregation in response to various agonists, reflecting platelet function, activity and reactivity in response to disease. The purpose of this study was therefore to investigate Multiplate® as a diagnostic tool for detection of variations in platelet aggregation in dogs with diseases known to predispose to hypercoagulability and thrombosis and to evaluate whether there is a correlation between Multiplate aggregation response and the maximal amplitude (MA) measured by thromboelastography (TEG).

Twenty clinically healthy dogs and eighteen diseased dogs with neoplasia, generalized inflammation or protein losing enteropathy or -nephropathy admitted to the University Hospital for Companion Animals, University of Copenhagen, were included in the study. Citrated and heparinised blood samples were collected. Multiplate® aggregations were performed on diluted heparinised whole blood for 12 minutes using ADP, Collagen (COL) and Arachidonic Acid (AA) as agonists and NaCl as buffer control. Results were recorded as area under the curve (AUC). Dilute (1:50000) Tissue Factor TEG analyses were performed on citrated whole blood.

Diseased dogs had significantly increased AUC compared to healthy dogs for NaCl buffer control (p=0.0005), ADP (p<0.0001) and COL (p=0.0048) whereas no significant difference was obtained for AA as agonist (p=0.3116). TEG-MA was significantly higher (p=0.0114) in diseased dogs compared to healthy dogs. A significant correlation was not found between TEG-MA and Multiplate AUC using ADP (p=0.1720, r=-0.3366), COL (p=0.2274, r=-0.2994) or AA (p=0.4304, r=-0.1982).

These results demonstrate that Multiplate® aggregation responses are significantly increased in a population of diseased dogs with diseases known to predispose to hypercoagulability and thrombosis, but results are not significantly correlated to TEG-MA. This suggests that the Multiplate method can be used to detect increased platelet reactivity in dogs with diseases known to predispose for hypercoagulability and thrombosis and that Multiplate provides additional information on platelet function than TEG alone in this patient group. Further studies are needed to determine how Multiplate and TEG-MA results correlate to thrombosis and whether there may be an added benefit of using them in combination.