COMMENTARY

Simple and accessible screening method for congenital thrombopathies using an impedance haematology counter – reply: The differences between impedance aggregometry in whole blood versus aggregometry in PRP. Is there a need for caution?

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Abstract  Brahimi et al. in this journal formed a hypothesis that “the platelet count is underestimated, by an automated cell counter, each time platelet aggregates are present in the sample tube.” The addition of a platelet agonist to a stimulated sample tube will lead to the formation of platelet aggregates and hence to a drop in the platelet count. In the case of a hereditary platelet dysfunction, platelet aggregates cannot be formed upon addition of a platelet agonist and the platelet count will remain unchanged. The authors propose a hypothesis to develop “a more accessible screening technique for these hereditary platelet dysfunctions.” In our reply, we critically evaluate this screening method and focus on the importance of the differences between impedance aggregometry in whole blood versus aggregometry in platelet-rich plasma.

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Dear sir,

We read with great interest the article by Brahimi et al. that was recently published in this journal [1]. Brahimi et al. hypothesise that “the platelet count is underestimated, by an automated cell counter, each time platelet aggregates are present in the sample tube.” The addition of a platelet agonist to a stimulated sample tube will lead to the formation of platelet aggregates and hence to a drop of the platelet count. In the case of a hereditary platelet dysfunction, platelet aggregates cannot be formed upon addition of a platelet agonist and the platelet count will remain unchanged. The authors propose a hypothesis to develop “a more accessible screening technique for these hereditary platelet dysfunctions” [1].

The mentioned method was tested at our haematology laboratory in the blood samples taken from patients without thrombopathy. For analysis, adenosine diphosphate (ADP) and ristocetin were used as inducers. The samples were collected into tubes containing ethylenediaminetetraacetic acid (EDTA) or citrate, because it is important that for aggregometry, the blood samples must be drawn into evacuated citrate tubes, while the haematology cell counters are calibrated for blood to be drawn to EDTA. Each sample was stirred and analysed on the haematology cell counter DxH 800 (Beckman Coulter, Pasadena, CA, USA). After adding the inducer into citrated blood samples, the results showed an abnormal drop in the platelet count from a normal range to minimal values of approximately 2–10 × 10^9/l. This result could be consistent with the hypothesis of Brahimi et al. [1]. However, because of the low number of tested samples, we were not able to standardise the method. The sample of a patient with thrombopathy was not provided; therefore, no quality control was carried out. Moreover, it is necessary to stress that the aggregation is accelerated at 37°C, upon rapid stirring, upon high concentrations of ADP and at a high platelet concentration; a decrease in any of these conditions slows down the process. It is also important to consider the interference of the cells.

Finally, the hypothesis requires a difficult standardisation of methodology by fulfilling the conditions necessary for platelet aggregation. At present, it is not possible to use it in practice.

Overview Box

First Question: What do we already know about the subject?
A summary of our knowledge about congenital thrombopathies and possibilities of their diagnostics is concisely described in paper by Brahimi et al., published in this journal (J Med Hypotheses and Ideas 2013;7:11–4).

Second Question: What does your proposed theory add to the current knowledge available, and what benefits does it have?
The authors suggest a simple and accessible screening method for the detection of congenital thrombopathies using only a haematology counter and some reagents.

Third question: Among numerous available studies, what special further study is proposed for testing the idea?
Similarly, as was stressed in the article by Brahimi et al., there is a lot of work to be done in comparative clinical studies with normal subjects and patients with thrombopathies for the standardisation of the proposed method and before its use in routine clinical practice.

Conflict of interest

We have no conflict of interest to declare.

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Reference