Vertebral canal haematoma and coagulopathy

Editor—Vertebral canal haematoma (VCH) is a rare but potentially devastating complication of epidural analgesia requiring rapid confirmation and urgent neurosurgical decompression.\(^1\) \(^2\) We witnessed such a case after a Whipple’s procedure in a 73-yr-old man with pancreatic carcinoma. Our case was associated with an unanticipated perioperative coagulopathy, which delayed definitive neurosurgery and contributed to a poor neurological recovery.

The patient’s liver function was abnormal before operation [bilirubin 50 mmol litre\(^{-1}\), alkaline phosphatase 645 IU litre\(^{-1}\), aspartate transaminase 207 IU litre\(^{-1}\), gamma GT 1030, international normalized ratio (INR) 1.36] but all other results were normal. Epidural insertion required attempts at more than one level due to contact with bone but was otherwise atraumatic and there were few other risk factors for VCH (Table 1).\(^3\) Surgery was uneventful and recognized intraoperative causes for coagulopathy such as acidosis, hypothermia, and hypocalcaemia were absent.\(^4\) However, after 2 litre of Hartmann’s, 2.5 litre of Gelofusine, and 2 units of packed cells, haemodilution cannot be excluded.

Two hours into recovery, the patient complained of weakness and decreased sensation in his legs. Repeat clotting tests showed: INR 2.5, activated partial thromboplastin time (APTT) ratio 1.6, and platelets 288 \(\times\) 10\(^9\) litre\(^{-1}\). Over 12 h and several rounds of concentrated clotting factors (in total 3 \(\times\) 500 IU Beriplex, 15 ml kg\(^{-1}\) fresh frozen plasma, 2 pools of cryoprecipitate, and 10 mg vitamin K) were required to reduce the INR below 1.5 and permit decompressive laminectomy. In spite of ‘correcting’ standard laboratory coagulation values, the neurosurgeons reported heavy blood loss consistent with an ongoing coagulopathy.

Haemostasis maintains blood flow and, in the event of vessel injury, rapidly arrests blood loss and promotes repair of the damaged vessel. Cholestatic liver disease impairs absorption of fat-soluble vitamins including vitamin K, leading to the reduced synthesis of vitamin K-dependent factors (II, V, VII, IX, and X) and may prolong the INR. However, INR, APTT, and fibrinogen results only provide information on coagulation and delineate neither formation of the primary platelet plug nor fibrinolysis. INR and APTT were developed to assess the therapeutic effects of warfarin and unfractionated heparin, respectively, and were not designed to answer the question ‘will the patient bleed?’\(^5\) They do not provide a surrogate to guarantee normal haemostatic function and yet these values are still used as endpoints to guide haemostatic therapy, perhaps driven by the perception of medico-legal safety.\(^6\) So how might intraoperative bleeding risk be best predicted?

The use of a screening questionnaire and, if indicated, further platelet function analysis has been shown to be more efficacious and cost-effective at detecting patients at increased risk of intraoperative bleeding.\(^5\) Family history of bleeding and previous bleeding after minor trauma should be recorded in addition to a full drug history.
Thromboelastography (‘TEG’) and rotational thromboelastometry (‘ROTEM’) are used increasingly in cardiac and liver surgery to evaluate clotting factor deficiencies and platelet dysfunction. Both monitor viscoelastic properties of whole blood as it is induced to clot in a low-shear environment (resembling sluggish venous flow) and provide valuable information on haemostasis from initiation to fibrinolysis.

Our case illustrates the limitations that routine clotting tests have in predicting the risk of haemorrhage and we postulate that had results of TEG or ROTEM been available the extent of the coagulation defect might have been determined more expediently. We believe that there may be an increasing role for such point of care testing in this setting.

Conflict of interest
None declared.

M. J. E. Peck*
A. Retter
P. Karuppasamy
M. F. Dunisire
London, UK
*E-mail: marcus.peck@nhs.net

Comparison between RapidTEG® and conventional thromboelastography in cardiac surgery patients

Editor—Coagulopathy after cardiopulmonary bypass (CPB) remains a complex issue in terms of monitoring and treatment. Point-of-care testing with thromboelastography (TEG) has shown to have a positive predictive value of 87–89% and a negative predictive value of 92% for postoperative haemorrhage. Since it takes 15–20 min to obtain complete conventional TEG results, a more rapid modification, Rapid-TEG®, was developed, which incorporates tissue factor to the kaolin-activated thromboelastogram. This double activation accelerates both the intrinsic and extrinsic coagulation pathways. We designed a study to assess if the data obtained from RapidTEG® concerning clot strength correlate with similar data from conventional TEG.

In 24 adult cardiac surgery patients, three samples were obtained from each patient, one before (baseline) and two after CPB (one immediately after protamine and one 10 min later). All samples underwent simultaneous analysis with RapidTEG® and conventional TEG. R- and K-times, \( \alpha \)-angles, and MA amplitudes were compared. Pre- and post-CPB platelet counts were recorded as well. Data are reported as mean (sd) or median (inter-quartile range). The Wilcoxon rank-sum test was used for intergroup comparisons. The Spearman correlation (\( \rho \)) and locally weighted regression analysis were used to examine the association between different TEG measurement methods. Bias and limits of agreement were investigated by the Bland–Altman analysis. A P-value of <0.05 was considered statistically significant.

MA amplitudes decreased significantly with both TEG methods during CPB. With conventional TEG, from baseline median 70.0 (66.4–72.5) to 59.4 (57.4–66.5) mm (P<0.0002) in sample 1 and to 59.3 (56.9–65.1) mm (P<0.00001) in sample 2. With RapidTEG®, from 67.2 (62.4–69.6) to 60.8 (57.3–63.8) mm (P=0.0001) in sample 1 and to 62.0 (57.2–66.1) mm (P<0.01) in sample 2.

Of all the TEG variables studied, only MA amplitudes demonstrated a significant correlation between conventional and RapidTEG®. The Bland–Altman analysis showed minimal bias for baseline values (4.5%) and Protamine-2 (–1.6%), with small 95% limits of agreements (within 20%). More variations in MA values were noted at the time point of Protamine-1 (Fig.1) Median platelet count decreased significantly from 238 (190–277) \times 10^9 \text{ litre}^{-1} \text{ pre-CPB} to 142 (108–170) \times 10^9 \text{ litre}^{-1} \text{ post-CPB} (P<0.0001).

The results show that there is a significant correlation for MA magnitude data obtained with RapidTEG® compared with conventional TEG, providing a reliable indication of maximal clot strength. The correlation was better 10 min after heparin reversal. MA values are reported to be mainly dependent on platelet numbers and on fibrinogen levels. The relationship between platelet numbers and MA values was confirmed in our study. In contrast, there was no correlation for \( \alpha \)-angles, representing the speed of clot formation, including platelet activity. Earlier studies have shown that these values are strongly depending on concentrations of fibrinogen and FXIII. They are apparently significantly affected by RapidTEG®.

These findings are important, since impaired haemostasis after CPB is considered to be partly due to platelet dysfunc-

dion, which has a multifactorial underlying mechanism including preoperative medication, fibrinolysis, receptor defect, contact activation, and hypothermia.

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