Laboratory QA

Interpretation of Coagulation Test Results Using a Web-Based Reporting System

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ABSTRACT

Background: Web-based synoptic reporting has been successfully integrated into diverse fields of pathology, improving efficiency and reducing typographic errors. Coagulation is a challenging field for practicing pathologists and pathologists-in-training alike.

Objective: To develop a Web-based program that can expedite the generation of a individualized interpretive report for a variety of coagulation tests.

Methods: We developed a Web-based synoptic reporting system composed of 119 coagulation report templates and 38 thromboelastography (TEG) report templates covering a wide range of findings.

Results: Our institution implemented this reporting system in July 2011; it is currently used by pathology residents and attending pathologists. Feedback from the users of these reports have been overwhelmingly positive. Surveys note the time saved and reduced errors.

Conclusion: Our easily accessible, user-friendly, Web-based synoptic reporting system for coagulation is a valuable asset to our laboratory services.

Keywords: Coagulation, Web-based, synoptic, reporting, interpretation, hemepathreview

Interpretation and reporting of coagulation tests is a time consuming task for practicing pathologists and pathology residents.[1] Creating a report that is concise and complete can be difficult. Learning to formulate reports is a fundamental aspect of pathology residency; however, it can be challenging without suitable guidance. The reports created by pathology residents may require extensive editing by the attending pathologist, thus delaying the final report. Once a resident learns the skills involved in report creation, implementing an efficient system to generate similar reports can expedite the sign-out process and reduce turnaround time. However, attempts to reduce the time spent generating reports often increase the frequency of typographic errors.

Abbreviations
TEG, thromboelastography; PT, prothrombin time; PTT, partial thromboplastin time; MA, Maximum Amplitude; Ly30, Lysis after 30 minutes; HTML, Hypertext Markup Language; LIS, laboratory information system

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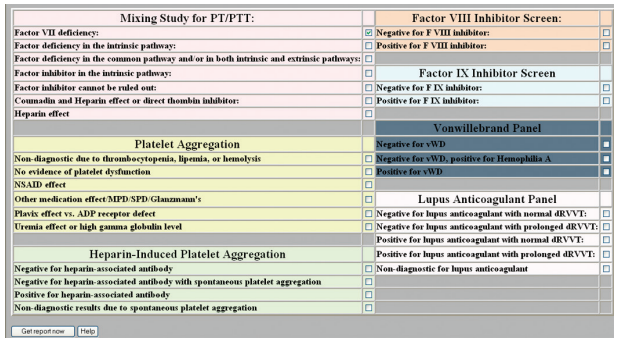
The College of American Pathologists has provided a series of checklists to assist pathologists in producing surgical pathology reports.[2] Web-based synoptic reporting systems have been successfully integrated into some specialties, including surgical pathology and hematopathology.[3-5] These systems have several advantages, including greater efficiency, reduced turnaround time, and fewer reporting errors. Our institution previously developed and described[6] a system to generate peripheral blood smear reports. Due to its practical and educational success, a system was subsequently created for the interpretation and generation of coagulation analyses.

Thromboelastography (TEG) is used to assess hypo- and hypercoagulable states and can guide transfusion with fresh-frozen plasma, platelets, or coagulation factor concentrates.[7] At our institution, TEG is primarily used for patients in the emergency room, trauma unit, or critical-care departments. We report our TEG results separately, rather than integrating them with other conventional coagulation studies.

The interactive reporting program that we describe herein assists in formulating the best possible report in a short amount of time; it serves as an educational tool for residents and a time-management resource for practicing pathologists.

Figure 1

User interface for selection of coagulation tests (www.hemepathreview.com).



Materials and Methods

We developed a Web-based reporting system composed of 119 coagulation report templates (**Appendix 1**) and 38 TEG report templates (**Appendix 2**) covering a wide range of clinical and laboratory findings. The knowledgebase is comprised of reports previously issued for patient care that members of the clinical pathology faculty selected as typical examples for a given result.

The interactive coagulation panels consist of 29 findings that can be selected by themselves or in any combination. These findings are subdivided by test and include mixing study for prothrombin time (PT)/partial thromboplastin time (PTT), platelet aggregation, heparin-induced platelet aggregation, factor VIII inhibitor screen, factor IX inhibitor screen, von Willebrand panel, and lupus anticoagulant panel (**Figure 1**). The TEG template generator allows for the selection of normal, low, or high values for reaction time (R), angle alpha (α), maximum amplitude (MA), and lysis after 30 minutes (Ly30) (**Figure 2**). Once the selections are made, the report templates are displayed in a text window for editing. Both programs can be accessed freely on the Internet at www.hemepathreview.com, by selecting the heading labeled "9. INTERPRETATION REPORT TEMPLATES USED FOR TRAINING," followed by selecting "Coagulation Report Templates" or "Thromboelastograph Templates." The system is designed to provide users with a set of concise yet comprehensive templates for a broad range of diagnoses related to coagulation disorders.

Our Web-based reporting system was implemented in Hypertext Markup Language (HTML).[8] Our system achieved interactivity with users by means of JavaScript, a scripting language that adds dynamic features to Web pages. The dynamic features allow users to interact with the graphical interface components displayed on the computer screen, such as buttons, lists, and check boxes, to retrieve the desired information. These dynamic features can be coded in HTML files in the form of JavaScript functions or subroutines. We also used JavaScript to create functions and subroutines used in the search engine for displaying the report templates.[8] The website is installed on a Microsoft Window 7 server running the Microsoft Internet Information Server (Microsoft Corporation, Redmond, WA) software in the Department of Pathology and Laboratory Medicine, University of Texas–Houston Medical School. The hospital laboratory information system (LIS) at our institution is Cerner Millennium (Cerner Corporation, North Kansas City, MO), which is integrated with other Cerner clinical systems, including electronic medical records, radiology, and pharmacy.

The Web-based reporting system made available to residents on the hematopathology service beginning in July 2011. All reports generated by this system are reviewed by the attending pathologist for accuracy and typographical errors before release.

To demonstrate how the system works, we offer the following example. The user interprets the results of a mixing study as indicating factor VII deficiency. He or she checks the corresponding box under the correct test and then left-clicks the "get report now" button (**Figure 1**). Two sample reports are generated in the text box under the heading "DRAFT FOR COAGULATION REPORT" (**Figure 2**). The user can now copy and paste the report that most closely matches the patient data into the "final draft" text box, after which the report can be edited with necessary changes or additions. The final draft will then be copied into the report in the LIS.

Figure 2

Sample program generated reports appear under the heading "DRAFT FOR COAGULATION REPORT." Users can freely edit within the same window or copy and paste onto the "FINAL DRAFT" section (www.hemepathreview.com).

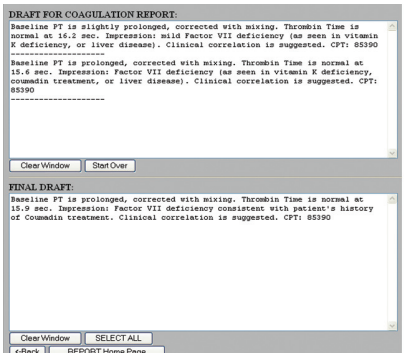
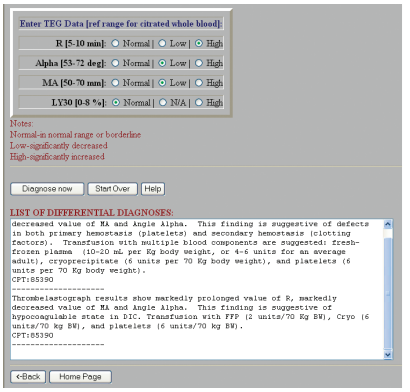


Figure 3

User interface for thromboelastography (TEG) report generation with sample reports (www.hemepathreview.com).



Results

We evaluated our Web-based coagulation reporting system via survey of 21 pathology residents who rotated through the hematopathology service from July 2011 through July 2012. The recruited residents used the system for drafting preliminary coagulation reports during their month-long rotation in hematopathology. None of the residents had previous experience with the system. The attending pathologist evaluated the draft reports prepared by residents for accuracy, comprehensiveness, and the presence of typographic errors. At the end of the rotation, an interviewer (A.N.N.) asked each resident the following survey questions, regarding whether the synoptic system:

(a) Decreases the amount of time preparing the report
(b) Decreases typographical errors and grammatical errors
(c) Includes all critical information in the report

The responses from residents to this survey were uniformly positive. All participants stated that this Web-based reporting system greatly improved turnaround time (estimated 30% to 40% decrease in the amount of time required to prepare reports). All residents observed that typographic errors, grammatical errors, and omission of important information in the drafts were significantly decreased. Similar benefits were noted by the attending pathologists who signed out the final reports.

Discussion

Web-based education has been shown[9-12] to be an effective teaching tool. Interactive Web-based tools[13] stimulate higher-order thinking and foster the learning of concepts rather than rules. This methodology has also proven effective in teaching pathology.[14-16] At our institution, we have promoted and implemented this valuable teaching method.[17-20]

We have created online interactive templates in hematopathology to assist in the generation of clinical reports, to reduce turnaround time and to aid in resident education. Two of us reported previously[6] on the use of

such templates for peripheral blood smears; we have now expanded on this information by adding coagulation and TEG templates.

The online format of the templates conveys numerous benefits. Unrestricted free access via www.hemepathreview.com allows users convenient, broad access to this Internet-based tool. JavaScript allows us to edit the website with relative ease and ensures that its content stays up to date.[8] The free access eliminates the need to purchase and install software. Also, the website is designed to be intuitive and user-friendly. Further, the ability to copy and paste directly from the website onto the reporting software of each institution bypasses compatibility issues. The text can be freely edited within the website or in the reporting software, depending on the personal preference of the user. Because our templates and the LIS interface are opened in separate windows (different threads in the operating system), security is not considered an issue. Our intention is for this website and the templates it contains to be a tool for pathologists everywhere. The success of our endeavor will ultimately be gauged by the number of people who regularly use it.

The reports serve as templates; thus, they require editing because each patient is unique. However, editing is minimal for most standard reports, and according to our residents and faculty members, processing feedback on certain sections of a report is preferable to typing an entire report.

The use of a template also eliminates the need for transcription. There are multiple daily coagulation studies and TEG studies that require timely reporting. In the case of so many relatively brief reports, removing the transcription step from the sign-out process reduces turnaround time and cost.

Resident education can also be enhanced by the use of the online templates. Use of our templates does not eliminate the need for critical thinking. Residents must still generate their diagnoses and conclusions based on the available data. They can use the website to generate a report by selecting their diagnosis from the displayed options. The website merely provides a tool for generating reports that require minimal editing. Because the focus of this program is on the reporting aspect and not on producing the diagnosis, the resident can concentrate on making the diagnosis rather than the report. For an experienced pathologist, the reduction in the amount of time spent on sign-out accrues from having a convenient method for generating a multitude of different coagulation reports.

However, this reporting method has certain limitations. Any updating of the website requires that the user or administrator edit files on the Web server. Although JavaScript programming language is relatively simple to use, it still requires a certain level of technical knowledge. Further, if an institution blocks all outside websites, our site would not be accessible.

An easily accessible, user-friendly, Web-based synoptic reporting system for coagulation can be an asset to pathologists. Survey data indicate that the program improves efficiency by reducing typographic errors and decreasing turnaround time. This system is currently being used by residents and faculty members at our institution, and is the preferred method for generating coagulation reports. LM

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Appendix 1: Coagulation Templates

1. Baseline prothrombin time (PT) is slightly prolonged, corrected with mixing. Thrombin time is normal, at 16.2 seconds. Impression: mild Factor VII deficiency (as observed in vitamin K deficiency or liver disease). Clinical correlation is suggested. CPT: 85390.
2. Baseline prothrombin time (PT) is prolonged, corrected with mixing. Thrombin time is normal, at 15.6 seconds. Impression: factor VII deficiency (as observed in vitamin K deficiency, warfarin sodium treatment, or liver disease). Clinical correlation is suggested. CPT: 85390.
3. Baseline partial thromboplastin time (PTT) is prolonged, corrected with mixing. Thrombin time is normal, at 18.8 seconds. Impression: results are suggestive of factor deficiency in the intrinsic pathway (factors VIII, IX, XI, or XII). CPT: 85390.
4. Hemophilia A

   Baseline partial thromboplastin time (PTT) is prolonged, corrected with mixing. Thrombin time is normal, at 17.3 seconds. Impression: results are consistent with factor deficiency in the intrinsic pathway. Factor VIII is low (21%), with normal von Willebrand factor level, consistent with hemophilia A. CPT: 85390.
5. Baseline partial thromboplastin time (PTT) is prolonged, partially corrected with mixing. Thrombin time is normal, at 19.2 seconds. Impression: findings are suggestive of factor deficiency in the intrinsic pathway. However, an inhibitor cannot be ruled out (such as lupus anticoagulant or Factor VIII inhibitor). CPT: 85390.
6. Baseline prothrombin time (PT) is slightly prolonged, corrected with mixing.

   Baseline partial thromboplastin time (PTT) is prolonged, partially corrected with mixing. Thrombin time is normal, at 17.1 seconds. Impression: findings are suggestive of factor deficiency in the common and/or in intrinsic and extrinsic pathways. However, an inhibitor cannot be ruled out (such as lupus anticoagulant). CPT: 85390.
7. Baseline prothrombin time (PT) and partial thromboplastin time (PTT) are prolonged, corrected with mixing. Thrombin time is normal, at 18.8 seconds. Impression: findings are suggestive of factor deficiency in the common and/or in intrinsic and extrinsic pathways. CPT: 85390.
8. Baseline prothrombin time (PT) and partial thromboplastin time (PTT) are markedly prolonged, corrected with mixing. Thrombin time is prolonged, at 28.8 seconds. Impression: findings are suggestive of factor deficiency in the common and/or in intrinsic and extrinsic pathways. CPT: 85390.
9. Normal von Willebrand panel results: no decrease in any components

   von Willebrand panel results show no decrease in any components (factor VIII, von Willebrand factor [vWF]: antigen and functional). Impression: no evidence of von Willebrand disease (except for postinfusion level in patients with known disease). CPT: 85390.
10. von Willebrand panel results: decrease in all components

    The von Willebrand panel results show decrease in all components (factor VIII, von Willebrand factor [vWF]: antigen and functional). Impression: results are consistent with von Willebrand disease. CPT: 85390.
11. Factor VIII deficiency, no evidence of von Willebrand disease

    The von Willebrand panel results show a decrease in factor VIII level (7%), no decrease in von Willebrand factor (vWF) (antigen and functional). Impression: results are consistent with factor VIII deficiency, no evidence of von Willebrand disease. CPT: 85390.
12. Normal platelet function

    Platelet aggregation study shows adequate aggregation with all reagents tested (arachidonic acid, adenosine diphosphate [ADP], collagen, epinephrine, and ristocetin). We observed no loss of secondary aggregation. Impression: no evidence of platelet dysfunction. CPT: 85576 x5.
13. Normal aggregation with collagen/ristocetin

    Platelet aggregation study results show decreased aggregation with arachidonic acid, adenosine diphosphate (ADP), and epinephrine. Loss of secondary aggregation is observed with ADP and epinephrine. Aggregation with collagen and ristocetin is adequate. Impression: findings are consistent with platelet dysfunction. This pattern is suggestive of medication effect (most likely, nonsteroidal anti-inflammatory drugs [NSAIDs]). Clinical correlation is suggested. CPT: 85576 x5.
14. Normal aggregation with ristocetin

    Platelet aggregation study results show markedly decreased aggregation with arachidonic acid and moderately decreased aggregation with adenosine diphosphate (ADP), collagen, and epinephrine. Loss of secondary aggregation is observed with ADP and epinephrine. Aggregation with ristocetin is adequate. Impression: findings are consistent with platelet dysfunction. This pattern is suggestive of medication effect (most likely, nonsteroidal anti-inflammatory drugs [NSAIDs]). Clinical correlation is suggested. CPT: 85576 x5.
15. Abnormal aggregation with adenosine diphosphate (ADP)

    Platelet aggregation study shows adequate aggregation with arachidonic acid, collagen, epinephrine, and ristocetin. Aggregation with ADP is markedly decreased, with loss of secondary aggregation. Impression: findings are consistent with platelet dysfunction. This pattern is suggestive of medication effect (such as of clopidogrel bisulfate) or platelet ADP-receptor defect. Clinical correlation is suggested. CPT: 85576 x5.

16. Abnormal aggregation with adenosine diphosphate (ADP) at low concentration

    Platelet aggregation study results show adequate aggregation with arachidonic acid, collagen, epinephrine, and ristocetin. Aggregation is markedly decreased, with loss of secondary aggregation at a low concentration of ADP. However, aggregation is adequate at a high ADP concentration. Impression: findings are consistent with mild platelet dysfunction. This pattern is suggestive of medication effect (such as clopidogrel bisulfate) or platelet ADP-receptor defect. Clinical correlation is suggested. CPT: 85576 x5.

17. Negative heparin-induced thrombocytopenia (HIT) test results

    Heparin-induced platelet aggregation study results show no significant increase in aggregation with heparin added (as high as 4% above the value with normal saline used as baseline). Impression: negative for heparin-associated antibody via the heparin-induced platelet aggregation method. CPT: 85576.

18. Negative heparin-induced thrombocytopenia (HIT) test results/ spontaneous aggregation of platelets

    Heparin-induced platelet aggregation study results show spontaneous aggregation with normal saline (20%) and no significant change in aggregation with heparin added. Impressions: a) Negative for heparin-associated antibody via the heparin-induced platelet aggregation method. b) Spontaneous aggregation of platelets, suggestive of thrombogenic plasma causing activation of platelets. CPT: 85576.

19. Baseline prothrombin time (PT) and partial thromboplastin time (PTT) are markedly prolonged, essentially corrected with mixing.

    Thrombin time is normal, at 18.7 seconds. Impression: findings are suggestive of factor deficiency in the common and/or in intrinsic and extrinsic pathways. CPT: 85390.

20. Baseline prothrombin time (PT) and partial thromboplastin time (PTT) are prolonged, only partially corrected with mixing.

    Thrombin time is normal, at 16.8 seconds. Impression: findings are suggestive of an inhibitor (such as lupus anticoagulant). CPT: 85390.

21. Baseline prothrombin time (PT) is slightly prolonged; this is corrected with mixing. Baseline partial thromboplastin time (PTT) is markedly prolonged, not corrected with mixing. Thrombin time is markedly prolonged, at 192 seconds. Impression: findings are most consistent with heparin effect. Clinical correlation is suggested. CPT: 85390.

22. Normal aggregation with collagen/epinephrine/ristocetin/arachidonic acid (AA) (high concentration)/adenosine diphosphate (ADP) (high concentration)

    Platelet aggregation study shows decreased aggregation with AA and ADP, both at low concentrations. Aggregation with AA and ADP at high concentration is adequate. Aggregation with collagen, epinephrine, and ristocetin is adequate. Impression: findings are consistent with mild platelet dysfunction. This pattern is suggestive of medication effect. Clinical correlation is suggested. CPT: 85576 x5.

23. Normal aggregation with collagen/ristocetin/arachidonic acid (AA) (high concentration)

    Platelet aggregation study results show decreased aggregation with ADP, epinephrine, and arachidonic acid at low concentration. Aggregation with arachidonic acid at high concentration is adequate. Aggregation with collagen and ristocetin is adequate. Impression: findings are consistent with platelet dysfunction. This pattern is suggestive of medication effect. Clinical correlation is suggested. CPT: 85576 x5.

24. Baseline partial thromboplastin time (PTT) is prolonged, not corrected with mixing

    Thrombin time is normal, at 18.2 seconds. Impression: findings are suggestive of an inhibitor in the intrinsic pathway (such as lupus anticoagulant or factor VIII inhibitor). CPT: 85390.

25. Baseline partial thromboplastin time (PTT) is markedly prolonged; this is partially corrected with mixing. Thrombin time is prolonged, at 38.6 seconds. Impression: findings are suggestive of heparin effect. Clinical correlation is suggested. CPT: 85390.

26. Abnormal platelet aggregation study results due to low platelet count cannot be ruled out.

    Platelet aggregation study results show decreased aggregation with all reagents tested (arachidonic acid [AA], adenosine diphosphate [ADP], collagen, epinephrine, and ristocetin). Loss of secondary aggregation is observed with AA and ADP. Platelet count on sample tested is 76,000/cmm. Impression: abnormal platelet aggregation study results due to low platelet count cannot be ruled out. Repeated testing at a later time when platelet count is higher than 100,000/ cmm is suggested if clinically indicated. CPT: 85576 x5.

27. Baseline prothrombin time (PT) is slightly prolonged, corrected with mixing. Baseline partial thromboplastin time (PTT) is markedly prolonged, not corrected with mixing. Thrombin time is normal, at 19.8 seconds. Impression: findings are most consistent with an inhibitor. Further testing for lupus anticoagulant is suggested if clinically indicated. CPT: 85390.

28. Abnormal aggregation with all reagents, in chronic renal insufficiency

    Platelet aggregation study results show decreased aggregation to arachidonic acid, adenosine diphosphate (ADP), collagen, epinephrine, and ristocetin. Loss of secondary aggregation is observed with ADP. Impression: dysfunctional platelets. The pattern observed in this study is not specific, but it is typically observed in chronic renal insufficiency. Clinical correlation is suggested. CPT: 85576 x5.

29. Normal aggregation with arachidonic acid (AA)/ristocetin

    Platelet aggregation study results show adequate aggregation with AA and ristocetin, decreased aggregation with adenosine diphosphate (ADP), collagen, and epinephrine. Loss of secondary aggregation is observed with ADP and epinephrine. Impression: findings are consistent with platelet dysfunction. This pattern is suggestive of medication effect. Clinical correlation is suggested. CPT: 85576 x5.

30. Baseline prothrombin time (PT) and partial thromboplastin time (PTT) are markedly prolonged, essentially corrected with mixing. Thrombin time is prolonged, at 58.1 seconds. Fibrinogen level is low, at 220. Liver function test results are markedly abnormal.

    Impression: findings are suggestive of factor deficiency in the common and in intrinsic and extrinsic pathways secondary to liver disorder. CPT: 85390.

31. Baseline prothrombin time (PT) is markedly prolonged, partially corrected with mixing. Baseline partial thromboplastin time (PTT) is markedly prolonged, not corrected with mixing. Thrombin time is markedly prolonged (more than 100 seconds)

    Impression: findings are most consistent with the combined effect of warfarin sodium and heparin. Clinical correlation is suggested. CPT: 85390.

32. Positive heparin-induced thrombocytopenia (HIT) test results

    Heparin-induced platelet aggregation study results show significant increase in aggregation with heparin added (as much as 30% higher than that with normal saline used as baseline). Impression: positive for heparin-associated antibody via heparin-induced platelet aggregation method. CPT: 85576.

33. Positive heparin-induced thrombocytopenia (HIT) test results; also, results via enzyme-linked immunosorbent assay (ELISA)

    Heparin-induced platelet aggregation study results show significant increase in aggregation with heparin added (as much as 31% higher than that with normal saline used as baseline). Impression: Positive results for heparin-associated antibody via the heparin-induced platelet aggregation method. Note: results of heparin-associated antibody test via ELISA (March 14, 2008) were also positive. CPT: 85576.

34. Positive results on the dilute Russell viper venom (dRVVT) and hexagonal phospholipid (Hex PL) test, partial thromboplastin time (PTT) not corrected in mixing study

    Positive results for lupus anticoagulant. Note: mixing study results show no correction for prolonged PTT. CPT: 85390.

35. Markedly-decreased aggregation with arachidonic acid (AA), adenosine diphosphate (ADP), collagen, and epinephrine

    Platelet aggregation study shows markedly decreased aggregation with AA, ADP, collagen, and epinephrine. Aggregation with ristocetin is adequate. Impression: findings are consistent with platelet dysfunction. This pattern may be observed with: medication effect, uremia, platelet storage pool disease, myeloproliferative disorder, or Glanzmann thrombasthenia. Clinical correlation is suggested. CPT: 85576 x5.

36. Baseline partial thromboplastin time (PTT) is prolonged, corrected with mixing

    Thrombin time is normal, at 20.7 seconds. Impression: results are suggestive of factor deficiency in the intrinsic pathway (factors VIII, IX, XI, or XII). In this 88-year-old woman, further testing for factors XI, XII, and von Willebrand panel (which includes factor VIII and von Willebrand factor) is suggested if clinically indicated. CPT: 85390.

37. Negative factor VIII inhibitor screen results

    Negative results for factor VIII inhibitor via screen panel, using 1:1 and 1:3 partial thromboplastin time (PTT) mixing study of control plasma and patient plasma. CPT: 85390.

38. Positive factor VIII inhibitor screen results

    Positive results for factor VIII inhibitor via screen panel, using 1:1 and 1:3 partial thromboplastin time (PTT) mixing study of control plasma and patient plasma. CPT: 85390.

39. Negative factor IX inhibitor screen results

    Negative results for factor IX inhibitor via screen panel, using 1:1 and 1:3 partial thromboplastin time (PTT) mixing study of control plasma and patient plasma. CPT: 85390.

40. Positive factor IX inhibitor screen

    Positive results for factor IX inhibitor via screen panel, using 1:1 and 1:3 partial thromboplastin time (PTT) mixing study of control plasma and patient plasma. CPT: 85390.

41. Normal aggregation with ristocetin

    Results of a platelet aggregation study with ristocetin show adequate aggregation with ristocetin at all concentrations. Impression: no evidence of von Willebrand disease or Bernard-Soulier syndrome. CPT: 85576.

42. Medication effect vs platelet storage pool disease

    Platelet aggregation study results show adequate aggregation with arachidonic acid and ristocetin and markedly decreased aggregation with adenosine diphosphate (ADP), collagen, and epinephrine. Loss of secondary aggregation is observed with ADP and epinephrine. Impression: findings are consistent with platelet dysfunction. This pattern is suggestive of medication effect or platelet storage pool disease. Clinical correlation is suggested. If medication effect is not the case in this patient, electron microscopic study of platelets may be needed to rule out platelet storage pool disease. CPT: 85576 x5.

43. Deficiency in Fitzgerald factor or Fletcher factor

    Baseline partial thromboplastin time (PTT) is prolonged, corrected with mixing. Thrombin time is normal, at 19.2 seconds. Subsequent assays for factors VIII, IX, XI, and XII all show normal results. Lupus anticoagulant results are also negative. Impression: results are suggestive of deficiency in Fitzgerald factor or Fletcher factor. Further testing for these factors is suggested if clinically indicated. CPT: 85390.

44. Medication effect (including nonsteroidal anti-inflammatory drugs [NSAIDs])

    Platelet aggregation study results show decreased aggregation with adenosine diphosphate [ADP], with loss of secondary aggregation. Aggregation with collagen is adequate. Specimen is of inadequate quantity for testing with ristocetin, arachidonic acid, and epinephrine. Impression: findings are consistent with platelet dysfunction. This pattern is suggestive of medication effect (including NSAIDs). Clinical correlation is suggested. CPT: 85576x2.

45. Baseline partial thromboplastin time (PTT) is markedly prolonged, partially corrected with immediate mixing; no correction after 1-hour incubation (factor VIII inhibitor).

    Thrombin time is normal, at 16.6 seconds. Impression: findings are consistent with an inhibitor in the intrinsic pathway. Subsequent testing shows very low factor VIII level (<0.7%), consistent with the presence of factor VIII inhibitor. CPT: 85390.

46. No evidence of platelet dysfunction, specimen quantity insufficient for all reagent concentrations

    Platelet aggregation study results show adequate aggregation with all reagents tested (arachidonic acid [AA], adenosine diphosphate [ADP], collagen, epinephrine, and ristocetin). No loss of secondary aggregation is observed with ADP or epinephrine. Note that aggregation with AA and epinephrine at low reagent concentration was not performed due to insufficient amount of platelets available for testing. Impression: the overall aggregation results show no evidence of platelet dysfunction. CPT: 85576 x5.

47. Negative heparin aggregation (with positive enzyme-linked immunosorbent assay [ELISA] results)

    Heparin-induced platelet aggregation study results show no significant increase in aggregation with heparin added (as much as 6% higher than that with normal saline used as baseline). Impression: negative for heparin-associated antibody via heparin-induced platelet aggregation method. Note: heparin-associated antibody results via ELISA (on November 1, 2008) were positive. Because the heparin-induced platelet aggregation method is more specific but less sensitive to detect such an antibody, clinical correlation is suggested. CPT: 85576.

48. Baseline partial thromboplastin time (PTT) is markedly prolonged, corrected with immediate mixing, but not corrected after 1-hour incubation (factor VIII inhibitor)

    Baseline prothrombin time (PT) is normal. Baseline partial thromboplastin time (PTT) is markedly prolonged, corrected with immediate mixing, but not corrected after 1-hour incubation (time-dependent inhibitor). Thrombin time is normal, at 16.4 seconds. Impression: findings are consistent with factor VIII inhibitor in this patient, with a history of factor VIII inhibitor. CPT: 85390.

49. Platelet dysfunction: medication effect vs platelet storage pool disease

    Platelet aggregation study results show moderately decreased aggregation with arachidonic acid, (adenosine diphosphate) ADP, and collagen; markedly decreased with epinephrine. Aggregation with ristocetin is adequate. Impression: findings are consistent with platelet dysfunction. This pattern may be observed with medication effect or platelet storage pool disease. Review of medical record showed positive family history for heavy bleeding and easy bruising. If medication effect (especially nonsteroidal anti-inflammatory drugs [NSAIDs]) can be ruled out, electron microscopic study of platelets would be useful to rule out platelet storage pool disease. Clinical correlation is suggested. CPT: 85576 x5.

50. Negative heparin aggregation (also, negative enzyme-linked immunosorbent assay [ELISA] results)

    Heparin-induced platelet aggregation study results show no significant increase in aggregation with heparin added (as much as 11% higher than that with normal saline used as baseline). Impression: negative for heparin-associated antibody via the heparin-induced platelet aggregation method. Note: heparin-associated antibody test results via ELISA (December 1, 2008) were also negative. CPT: 85576.

51. Nondiagnostic factor VIII inhibitor screen due to lupus anticoagulant (LA) (signed out as negative factor VIII inhibitor screen)

    Results of factor VIII inhibitor testing (via screen panel using 1:1 and 1:3 partial thromboplastin time [PTT] mixing study of control plasma and patient plasma) are not diagnostic due to interference by LA in this patient. However, factor VIII was found to be in the normal range (176%). This factor VIII level effectively shows no evidence of factor VIII inhibitor. CPT: 85390.

52. Platelet dysfunction due to medication effect (aspirin and clopidogrel per medical history [hx])

    Platelet aggregation study results show markedly decreased aggregation with arachidonic acid, adenosine diphosphate [ADP], collagen, and epinephrine. Loss of secondary aggregation is observed with ADP and epinephrine. Aggregation with ristocetin is adequate. Impression: findings are consistent with platelet dysfunction. This pattern is consistent with medication effect (aspirin and clopidogrel, in this patient). CPT: 85576 x5.

53. Platelet dysfunction due to medication effect (most likely nonsteroidal anti-inflammatory drugs [NSAIDs]).

    Platelet aggregation study results show markedly decreased aggregation with arachidonic acid (AA), moderately decreased with adenosine diphosphate [ADP], and epinephrine. Loss of secondary aggregation is observed with ADP and epinephrine. Aggregation with ristocetin and collagen is adequate. Impression: findings are consistent with platelet dysfunction. This pattern is suggestive of medication effect (most likely NSAIDs). Note that this patient has a history of von Willebrand disease (vWD) and is currently undergoing treatment. The normal aggregation with ristocetin is most likely due to treatment. Abnormal aggregation with AA, ADP, and epinephrine is not due to vWD. Clinical correlation is suggested. CPT: 85576 x5.

54. Positive results for von Willebrand disease (vWD)

    von Willebrand panel results show decrease in von Willebrand factor levels (antigen and functional). Factor VIII is borderline normal. Impression: results are consistent with vWD. CPT: 85390

55. Platelet dysfunction due to medication effect (most likely nonsteroidal anti-inflammatory drugs [NSAIDs]).

    Platelet aggregation study shows markedly decreased aggregation with arachidonic acid and moderately decreased aggregation with adenosine diphosphate [ADP], collagen, and epinephrine. Loss of secondary aggregation is observed with ADP and epinephrine. Aggregation with ristocetin is adequate. Impression: findings are consistent with platelet dysfunction. This pattern is suggestive of medication effect (most likely NSAIDs). Clinical correlation is suggested. CPT: 85576 x5.

56. Mild platelet dysfunction suggestive of medication effect (most likely nonsteroidal anti-inflammatory drugs [NSAIDs]).

    Platelet aggregation study results show a slight-to-moderate decrease in aggregation with arachidonic acid and a slight decrease with collagen. Aggregation with adenosine diphosphate [ADP], epinephrine, and ristocetin is adequate. Impression: findings are consistent with mild platelet dysfunction. This pattern is suggestive of medication effect (most likely NSAIDs). Clinical correlation is suggested. CPT: 85576 x5.

57. Negative dilute Russell viper venom test (dRVVT) results/ positive hexagonal phospholipid (Hex PL) results/positive platelet neutralization procedure (PNP) results; positive results for lupus anticoagulant. Details of the testing: partial thromboplastin time(PTT) is prolonged with no correction in mixing study; dRVVT results are prolonged, at 167.7 seconds; Hex PL neutralization results are positive; PNP results show presence of lupus anticoagulant (clotting time goes from >200 seconds down to 112.2 seconds); results of testing for factors VIII and IX show apparent increase in factor level with serial sample dilutions; factors VIII and IX are in the normal ranges with higher dilution, indicating the absence of inhibitors against these factors; and thrombin time is normal, at 17.0 seconds, which rules out heparin as an interference substance. CPT: 85390.

58. Negative dilute Russell viper venom test (dRVVT) results/negative hexagonal phospholipid (Hex PL) neutralization results

    Negative results for lupus anticoagulant with all tests performed. CPT: 85390.

59. Positive dilute Russell viper venom test (dRVVT) results/positive hexagonal phospholipid (Hex PL) neutralization results

    Positive results for lupus anticoagulant with all tests performed. Thrombin time is normal, at 17.0 seconds, which rules out heparin as an interference substance. CPT: 85390.

60. Negative dilute Russell viper venom test (dRVVT) results/positive hexagonal phospholipid (Hex PL) neutralization results/platelet neutralization procedure (PNP) not performed due to normal baseline partial thromboplastin time (PTT).

    Nondiagnostic for lupus anticoagulant (normal prothrombin time [PT] and PTT). Repeated lupus anticoagulant testing is suggested at a later time if clinically indicated. CPT: 85390.

61. Negative dilute Russell viper venom test (dRVVT) results/negative hexagonal phospholipid (Hex PL) neutralization results/positive platelet neutralization procedure (PNP) results

    Borderline-positive results for lupus anticoagulant (prolonged partial thromboplastin time [PTT], borderline-negative dRVVT results, negative Hex PL neutralization results, and positive PNP results). Thrombin time is normal, at 17.0 seconds, which rules out heparin as an interference substance. Repeated lupus anticoagulant testing is suggested at a later time if clinically indicated. CPT: 85390.

62. Positive dilute Russell viper venom test (dRVVT) results/negative hexagonal phospholipid (Hex PL) neutralization results/positive platelet neutralization procedure (PNP) results.

    Positive results for lupus anticoagulant. Thrombin time is normal, at 17.0 seconds, which rules out heparin as an interference substance. CPT: 85390.

63. Negative dilute Russell viper venom test (dRVVT) results/positive hexagonal phospholipid (Hex PL) neutralization results/positive platelet neutralization procedure (PNP) results

    Positive results for lupus anticoagulant. Thrombin time is normal, at 17.0 seconds, which rules out heparin as an interference substance. CPT: 85390.

64. Positive dilute Russell viper venom test (dRVVT) results/Negative hexagonal phospholipid (Hex PL) neutralization results/negative platelet neutralization procedure (PNP) results

    Negative results for lupus anticoagulant. Thrombin time is normal, at 15.2 seconds, which rules out heparin as an interference substance. CPT: 85390.

65. Negative dilute Russell viper venom test (dRVVT) results/positive hexagonal phospholipid (Hex PL) results/Negative platelet neutralization procedure (PNP) results

    Negative for lupus anticoagulant. Thrombin time is normal, at 16.7 seconds, which rules out heparin as an interference substance. CPT: 85390.

66. Positive dilute Russell viper venom test (dRVVT) results/negative hexagonal phospholipid (Hex PL) neutralization/platelet neutralization procedure (PNP) not performed

    Nondiagnostic for lupus anticoagulant. Thrombin time is markedly prolonged (>100 seconds), which indicates heparin as an interference substance that prolongs dRVVT results. Repeated testing after patient has stopped taking heparin is suggested if clinically indicated. CPT: 85390.

67. Positive dilute Russell viper venom test (dRVVT) results/negative hexagonal phospholipid neutralization (Hex PNP) results/positive platelet neutralization procedure (PNP) results

    Nondiagnostic for lupus anticoagulant. Thrombin time is markedly prolonged (>100 seconds), which indicates heparin as an interference substance. The positive PNP result is most consistent with heparin effect (false positive). Repeated testing after patient has stopped taking heparin is suggested if clinically indicated. CPT: 85390.

68. Positive dilute Russell viper venom test (dRVVT) results/negative hexagonal phospholipid (Hex PL) neutralization results/platelet neutralization procedure (PNP) not performed

    Negative results for lupus anticoagulant (prolonged dRVVT, negative Hex PL results). PNP was not performed due to normal baseline partial thromboplastin time (PTT) of 34.3 seconds. Thrombin time is normal, at 18.9 seconds. Prothrombin time (PT) is prolonged, at 24.8 seconds. Lupus anticoagulant is unlikely, with normal PTT and prolonged PT. CPT: 85390.

69. Negative dilute Russell viper venom test (dRVVT)/positive hexagonal phospholipid (Hex PL) neutralization/positive platelet neutralization procedure (PNP)

    Nondiagnostic for lupus anticoagulant. Thrombin time is markedly prolonged, at 60.4 seconds, which indicates heparin as an interference substance. The positive Hex PNP and PNP results are most consistent with heparin effect (false positive). Repeated testing after patient has stopped taking heparin is suggested if clinically indicated. CPT: 85390.

70. Positive dilute Russell viper venom test (dRVVT) results/negative hexagonal phospholipid (Hex PL) neutralization results/platelet neutralization procedure not performed

    Nondiagnostic for lupus anticoagulant. Specimen quantity is insufficient for further testing (via PNP). Re-collection of specimens is suggested for a definitive diagnosis. CPT: 85390.

71. Clopidogrel bisulfate effect

    Platelet aggregation study results show adequate aggregation with arachidonic acid, collagen, epinephrine, and ristocetin. Aggregation is moderately decreased with loss of secondary aggregation at a low concentration of adenosine diphosphate (ADP). Aggregation is adequate at high ADP concentration, however. Impression: findings are consistent with mild-to-moderate decrease in platelet dysfunction. This pattern is consistent with medication effect (patient taking clopidogrel bisulfate).CPT: 85576 x5.

72. Prolonged partial thromboplastin time (PTT) with von Willebrand disease

    Baseline PTT is prolonged, corrected with mixing. Thrombin time is normal, at 16.6 seconds. Impression: results are suggestive of factor deficiency in the intrinsic pathway. This is most likely due to low factor VIII level in this patient, who has a history of von Willebrand disease. Correlation with factor VIII level and von Willebrand factor level is suggested. CPT: 85390.

73. Uremia or high gamma globulin concentrations

    Platelet aggregation study results show a moderate-to-marked decrease in aggregation to arachidonic acid, adenosine diphosphate (ADP), collagen, epinephrine, and ristocetin. Loss of secondary aggregation is observed with ADP and epinephrine. Impression: dysfunctional platelets. The pattern observed in this study is not specific but it is often seen in chronic renal insufficiency or very high gamma globulin concentrations associated with some lymphoid or plasma cell neoplasms. Clinical correlation is suggested. CPT: 85576 x5.

74. Normal platelet function

    Platelet aggregation study results show a decrease in aggregation with arachidonic acid (AA) at low concentration and normal aggregation with higher AA concentration. Aggregation with adenosine diphosphate (ADP), epinephrine, collagen, and ristocetin is adequate. Impression: findings are consistent with adequate platelet dysfunction. The only decrease in aggregation with AA at a low concentration is unlikely to be of clinical significance. Clinical correlation is suggested. CPT: 85576 x5.

75. Direct thrombin inhibitor

    Baseline prothrombin time (PT) and partial thromboplastin time (PTT) are markedly prolonged, not corrected with mixing. Thrombin time is markedly prolonged, at greater than 100 seconds. Impression: findings are most consistent with a direct thrombin inhibitor (such as argatroban). Clinical correlation is suggested. CPT: 85390.

76. Aspirin ingestion or renal disease

    Platelet aggregation study results show decreased aggregation to arachidonic acid and adenosine diphosphate (ADP). Aggregation is borderline with collagen, epinephrine, and ristocetin. Loss of secondary aggregation is observed with ADP. Impression: mild platelet dysfunction. The pattern observed in this study is not specific but it is typically observed with medication effect or chronic renal insufficiency. Clinical correlation is suggested. CPT: 85576 x5.

77. Medication or renal disease

    Platelet aggregation study results show decreased aggregation to arachidonic acid, adenosine diphosphate (ADP), collagen, and epinephrine. Loss of secondary aggregation is observed with ADP and epinephrine. Aggregation with ristocetin is adequate. Impression: dysfunctional platelets. This pattern is suggestive of medication effect (including nonsteroidal anti-inflammatory drugs [NSAIDs]). In this patient with end-stage renal disease, dysfunctional platelets due to uremia may also contribute to catheter bleeding at the catheter site. Clinical correlation is suggested. CPT: 85576x5.

78. Normal platelet function

    Platelet aggregation study results show adequate aggregation with arachidonic acid, collagen, epinephrine, and ristocetin. Aggregation with adenosine diphosphate (ADP) is decreased with low concentrations but normal at high concentrations. Impression: essentially normal platelet function. The decrease in ADP only at low concentrations is unlikely to be associated with clinical significance. Clinical correlation is suggested. CPT: 85576 x5.

79. Aspirin and clopidogrel bisulfate

    Platelet aggregation study shows markedly decreased aggregation with arachidonic acid and adenosine diphosphate (ADP), moderately decreased with collagen and epinephrine. Loss of secondary aggregation is observed with ADP and epinephrine. Aggregation with ristocetin is adequate. Impression: findings are consistent with platelet dysfunction. This pattern is consistent with medication effect (patient was prescribed a regimen of aspirin and clopidogrel bisulfate). Clinical correlation is suggested. CPT: 85576 x5.

80. Normal platelet function

    Platelet aggregation study shows adequate aggregation with ristocetin at all concentrations. Impression: no evidence of von Willebrand disease or Bernard-Soulier syndrome. CPT: 85576.

81. Normal platelet function

    Platelet aggregation study results show adequate aggregation with all reagents tested (arachidonic acid, adenosine diphosphate [ADP], collagen, epinephrine, and ristocetin). No loss of secondary aggregation is observed with ADP or epinephrine. Note that only aggregation with ADP at low reagent concentrations is slightly decreased. Impression: the overall aggregation results show no evidence of platelet dysfunction. CPT: 85576 x5.

82. Aspirin ingestion or renal disorder

    Platelet aggregation study results show decreased aggregation to arachidonic acid, adenosine diphosphate (ADP), collagen, epinephrine, and ristocetin. Loss of secondary aggregation is observed with ADP and epinephrine. Impression: dysfunctional platelets. The pattern seen in this study is not specific but may be observed in chronic renal insufficiency or with medication effect. Clinical correlation is suggested. CPT: 85576 x5.

83. Factor deficiency in the common and/or in intrinsic and extrinsic pathways

    Baseline prothrombin time (PT) and partial thromboplastin time (PTT) are prolonged, corrected with mixing. Thrombin time is prolonged, at 64.4 seconds. Impression: findings are suggestive of factor deficiency in the common and/or in intrinsic and extrinsic pathways. CPT: 85390.

84. Mild von Willebrand disease (vWD), most likely subclinical

    von Willebrand panel results show normal level of factor VIII (88%), von Willebrand factor (vWF) levels are slightly decreased (vWF aggregate, 46%), vWF functional, 48%). These results are suggestive of mild vWD, most likely subclinical. Because the levels are borderline, repeated testing is suggested at a later time. CPT: 85390.

85. Factor inhibitor in intrinsic pathway

    Baseline prothrombin time (PT) is moderately prolonged, not corrected with mixing. Baseline partial thromboplastin time (PTT) is markedly prolonged, not corrected with mixing. Thrombin time is slightly prolonged, at 23.6 seconds. Impression: findings are most consistent with an inhibitor. Further testing for lupus anticoagulant is suggested if clinically indicated. CPT: 85390.

86. Negative for lupus anticoagulant

Negative for lupus anticoagulant with all tests performed (dilute Russell viper venom test [dRVVT] and hexagonal phospholipid neutralization). Note: previous lupus anticoagulant testing results were positive. These findings are indicative of transient lupus anticoagulant. Repeated testing at a later time is suggested. CPT: 85390.

87. Nondiagnostic for lupus anticoagulant

Nondiagnostic for lupus anticoagulant (prolonged dilute Russell viper venom test [dRVVT], negative hexagonal phospholipid neutralization results, and positive platelet neutralization procedure results). Thrombin time could not be performed to rule out heparin effect (due to insufficient specimen material). Note that heparin may cause false-positive results for dRVVT and platelet neutralization procedure. Re-collection of sample is suggested. CPT: 85390.

88. Nondiagnostic for lupus anticoagulant

Nondiagnostic for lupus anticoagulant (prolonged dilute Russell viper venom test [dRVVT] with negative hexagonal phospholipid neutralization). Platelet neutralization procedure was not performed due to normal baseline partial thromboplastin time (PTT), at 35.3 seconds. Prothrombin time (PT) is prolonged, at 20.4 seconds. The prolonged dRVVT results are likely due to vitamin K deficiency. Repeated lupus anticoagulant testing is suggested at a later time if clinically indicated. CPT: 85390.

89. Medication vs platelet storage pool disease

Platelet aggregation study results show adequate aggregation with arachidonic acid, collagen, and ristocetin, decreased aggregation with adenosine diphosphate (ADP) (only at low ADP concentration), and epinephrine. Loss of secondary aggregation is observed with ADP and epinephrine. Impression: findings are consistent with mild platelet dysfunction. This pattern is suggestive of medication effect. If medication can be ruled out, platelet storage pool disease may be considered. Clinical correlation is suggested. CPT: 85576 x5.

90. Aspirin and clopidogrel bisulfate effect

Platelet aggregation study results show markedly decreased aggregation with arachidonic acid (AA) (less than 4% on a 100% scale), and adenosine diphosphate (ADP) (less than 9% on a 100% scale). Impression: findings are consistent with effective inhibition of platelets by aspirin and clopidogrel bisulfate in this patient, who is taking both. CPT: 85576x2.

91. Negative for lupus anticoagulant

Negative for lupus anticoagulant with all tests performed (dilute Russell viper venom test [dRVVT] and hexagonal phospholipid neutralization). CPT: 85390.

92. Platelet aggregation: not diagnostic due to marked lipemia and hemolysis

Platelet aggregation study results show decreased aggregation with all reagents tested (marked decrease with arachidonic acid, adenosine diphosphate [ADP], collagen, epinephrine, and mild decrease with ristocetin). Gross examination of specimens revealed marked lipemia and hemolysis. Impression: Nondiagnostic results due to marked lipemic and hemolyzed specimen. Repeated testing at a later time after lipemia and hemolysis have been resolved is suggested if clinically indicated. Another option is to assess platelet function via thromboelastography, which is not affected by lipemia and hemolysis (whole blood collected in 1 blue-top tube). CPT: 85576 x5.

93. Platelet aggregation: only decrease with epinephrine

Platelet aggregation study results show adequate aggregation with arachidonic acid, adenosine diphosphate (ADP), collagen, and ristocetin. Aggregation with epinephrine is decreased. Impression: decrease in aggregation with only epinephrine (with adequate aggregation with all other reagents) has been described in patients who demonstrate no evidence of platelet dysfunction. The results of this study are not supportive of platelet dysfunction. Clinical correlation is suggested. CPT: 85576 x5.

94. Nondiagnostic for lupus anticoagulant

Nondiagnostic for lupus anticoagulant (borderline-negative dilute Russell viper venom test [dRVVT], positive hexagonal phospholipid neutralization). Thrombin time is normal, at 18.6 seconds. Platelet neutralization procedure is nondiagnostic due to shortened partial thromboplastin time (PTT) of patient plasma with platelet phospholipid and also with normal saline. Repeated testing at a later time is suggested. CPT: 85390.

95. Nondiagnostic for lupus anticoagulant

Nondiagnostic for lupus anticoagulant (prolonged dilute Russell viper venom test [dRVVT] results, positive hexagonal phospholipid [Hex PL] neutralization results). Thrombin time is markedly prolonged (>100 seconds), which indicates heparin as an interference substance that may yield false-positive dRVVT results and Hex PL neutralization results. Repeated testing after patient has stopped taking heparin is suggested if clinically indicated. CPT: 85390.

96. Negative for lupus anticoagulant

Negative for lupus anticoagulant (normal dilute Russell viper venom test results, positive hexagonal phospholipid neutralization results). Platelet neutralization procedure was not performed due to normal baseline partial thromboplastin time (PTT) (32.2 seconds). Thrombin time is normal, at 14.4 seconds. Prothrombin time (PT) is also normal, at 13.7 seconds. Lupus anticoagulant is unlikely with normal prothrombin time (PT)/ partial thromboplastin time (PTT). CPT: 85390.

97. Positive test results for von Willebrand disease (vWD), mild

von Willebrand panel results shows normal factor VIII and borderline levels of von Willebrand factor (vWF) (antigen and functional). Impression: results are suggestive of mild vWD. Clinical correlation is suggested. CPT: 85390.

98. von Willebrand disease (vWD), type II

von Willebrand panel results show decrease in von Willebrand factor (vWF) functional level. vWF antigen level is borderline normal. Factor VIII is also decreased. Impression: results are consistent with vWD. The discrepancy between vWF antigen and functional levels suggests vWD type II. Multimer testing is suggested for subtyping of vWD if clinically indicated. CPT: 85390.

99. Borderline-negative results for lupus anticoagulant

Borderline-negative results for lupus anticoagulant (prolonged dilute Russell viper venom test [dRVVT] results, negative hexagonal phospholipid neutralization results, and borderline-negative platelet neutralization procedure results). Thrombin time is normal, at 22.1 seconds, which rules out heparin as an interference substance. Repeated testing for lupus anticoagulant is suggested at a later time. CPT: 85390.

100. Nondiagnostic for lupus anticoagulant

Nondiagnostic for lupus anticoagulant (prolonged dilute Russell viper venom test [dRVVT] results, negative hexagonal phospholipid neutralization results). Thrombin time is normal, at 17.3 seconds. Platelet neutralization procedure is nondiagnostic due to shortened partial thromboplastin time (PTT) of patient plasma with platelet phospholipid and also with normal saline. Repeated testing at a later time is suggested. CPT: 85390.

101. von Willebrand disease (vWD), type II

von Willebrand panel results show a slight decrease in factor VIII and von Willebrand factor (vWF) antigen and a moderate decrease in vWF functional level. Impression: results are consistent with vWD. The discrepancy between vWF antigen and functional levels suggests vWD type II. Multimer testing is suggested for subtyping of vWD if clinically indicated. CPT: 85390.

102. von Willebrand disease (vWD) type II, mild

von Willebrand panel results show normal factor VIII and von Willebrand factor (vWF) antigen, mild decrease in vWF functional level. Impression: results are consistent with mild vWD. The discrepancy between vWF antigen and functional levels suggests vWD type II. Multimer testing is suggested for subtyping of vWD if clinically indicated. CPT: 85390.

103. Factor XII deficiency

Baseline partial thromboplastin time (PTT) is prolonged, corrected with mixing. Thrombin time is normal, at 17.7 seconds. Impression: results are consistent with factor deficiency in the intrinsic pathway. Subsequent testing showed normal factor IX (85%), factor VIII (143%), factor XI (91%), and low factor XII (11%). These results are diagnostic of factor XII efficiency in this 9-year-old male patient. CPT: 85390.

104. Mild medication effect (nonsteroidal anti-inflammatory drugs [NSAIDs])

Platelet aggregation study results show a slight decrease in aggregation with arachidonic acid and adenosine diphosphate. Aggregation with collagen, epinephrine, and ristocetin is adequate. Impression: findings are consistent with mild platelet dysfunction, most likely not clinically significant. This pattern is suggestive of medication effect (most likely NSAIDs). Clinical correlation is suggested. CPT: 85576 x5.

105. Selective abnormality in collagen due to L-asparaginase

Platelet aggregation study results show adequate aggregation with arachidonic acid, adenosine diphosphate, epinephrine, and ristocetin. Aggregation with collagen is markedly decreased. Impression: findings are consistent with platelet dysfunction. Selective abnormality in collagen aggregation appears to result from therapy, with the use of L-asparaginase, in particular, being implicated. Clinical correlation is suggested. CPT: 85576 x5.

106. Platelet dysfunction in patient with history of myelodysplastic syndrome [MDS]

Platelet aggregation study results show adequate aggregation with arachidonic acid, adenosine diphosphate, and ristocetin. Aggregation with epinephrine and collagen is decreased. Impression: platelet dysfunction. Selective decrease in aggregation with collagen is suggestive of collagen-receptor defect. Decrease in aggregation with epinephrine is nonspecific. Platelet dysfunction in this patient with history of MDS may be associated with this hematological disorder. Clinical correlation is suggested. CPT:85576 x5.

107. von Willebrand disease (vWD), type IIB

Platelet aggregation study results show adequate aggregation with arachidonic acid, adenosine diphosphate, collagen, and epinephrine. No loss of secondary aggregation is observed. Aggregation with ristocetin is adequate at high concentration and increased with low concentration (compared with that of normal control specimens). Impression: findings are suggestive of vWD, type IIB. vWD panel results are suggested for definitive diagnosis if clinically indicated. CPT: 85576 x5.

108. Therapeutic results for antiplatelet medications in total artificial heart

Platelet aggregation study results show markedly decreased aggregation with arachidonic acid, adenosine diphosphate (ADP), and epinephrine. Aggregation with ristocetin and collagen is slightly moderately decreased (most likely due to low platelet count of 74,000 after concentration; a control specimen with low platelet count is also run in parallel for comparison). Impression: findings are consistent with medication effect (in this patient who is taking aspirin and dipyridamole). Current dosages of aspirin and dipyridamole appear to be adequate according to total artificial heart antiplatelet medication protocol (markedly decreased aggregation with arachidonic acid, ADP, and epinephrine but not with collagen). Findings were reported to Dr Y. Bai on September 10, 2012 at 14:30. CPT: 85576 x5.

109. Platelet aggregation with bivalirudin (all aggregations are suppressed, 13%-32%)

Platelet aggregation study results show moderate-to-marked decrease (13%-32%) in aggregation to arachidonic acid, adenosine diphosphate, collagen, epinephrine, and ristocetin

Impression: dysfunctional platelets. The pattern seen in this study is most likely associated with the antiplatelet activity of bivalirudin (which this patient is currently taking). Clinical correlation is suggested. CPT: 85576 x5.

110. Aspirin effect

Platelet aggregation study results show markedly decreased aggregation with arachidonic acid and moderately decreased aggregation with adenosine diphosphate (ADP) at low concentration, collagen, and epinephrine. Loss of secondary aggregation is observed with ADP and epinephrine. Aggregation with high ADP concentration (50 µM/mL) is adequate (58%). Aggregation with ristocetin is adequate.

Impression: findings are consistent with platelet dysfunction. This pattern is suggestive of medication effect (most likely nonsteroidal anti-inflammatory drugs [NSAIDs]; patient is taking 162 mg/day of aspirin). Clinical correlation is suggested. CPT: 85576 x5.

111. Platelet aggregation, aspirin, and ticagrelor

Platelet aggregation study results show markedly decreased aggregation with arachidonic acid, moderately decreased aggregation with adenosine diphosphate (ADP) at low concentration, collagen, and epinephrine

Loss of secondary aggregation is observed with ADP and epinephrine. Aggregation with high ADP concentration (50µM/mL) is slightly decreased (50%). Aggregation with ristocetin is adequate.

Impression: findings are consistent with platelet dysfunction. This pattern is suggestive of medication effect (most likely nonsteroidal anti-inflammatory drugs [NSAIDs] and ticagrelor; patient is taking 82 mg/day of aspirin, ticagrelor 90 mg twice daily). Clinical correlation is suggested. CPT: 85576 x5.

112. Normal platelet aggregation at low platelet count

Platelet aggregation study results show adequate aggregation with all reagents tested (arachidonic acid, adenosine diphosphate, collagen, epinephrine, and ristocetin). Note that the platelet count of the patient is low (66,000). The aggregation results for the specimen from this patient are compared with those from a positive-testing control specimen with a comparable platelet count (65,000). Impression: no evidence of platelet dysfunction. CPT: 85576 x5.

113. Total artificial heart (TAH), therapeutic range

Platelet aggregation study results show markedly decreased aggregation with arachidonic acid, mild decrease with adenosine diphosphate (ADP), collagen, and epinephrine. Loss of secondary aggregation is observed with ADP and epinephrine. Aggregation with ristocetin is adequate. Impression: findings are consistent with platelet dysfunction. This pattern is consistent with medication effect (aspirin and dipyridamole in this patient with TAH implantation). The results indicate that antiplatelet medications are in therapeutic ranges for the TAH. CPT: 85576 x5.

114. Platelet aggregation, aspirin, no residual clopidogrel bisulfate

Platelet aggregation study results show decreased aggregation with arachidonic acid, adenosine diphosphate (ADP), collagen, and epinephrine. Loss of secondary aggregation is observed with ADP and epinephrine. Aggregation with high concentration of ADP (50 µM/mL) is adequate (60%). Aggregation with ristocetin is also adequate.

Impression: findings are consistent with platelet dysfunction. This pattern is suggestive of medication effect (most likely nonsteroidal anti-inflammatory drugs [NSAIDs]). Per electronic medical record review, patient is taking aspirin (325 mg twice daily) and clopidogrel bisulfate (75 mg twice daily). Platelet function of the patient is affected by aspirin but not clopidogrel bisulfate at this time. Clinical correlation is suggested. CPT: 85576 x5.

115. Platelet aggregation, total artificial heart (TAH), need to increase aspirin dosage

Platelet aggregation study results shows decreased aggregation with arachidonic acid and epinephrine. Aggregation with adenosine diphosphate (ADP), collagen, and ristocetin is adequate. Impression: findings are consistent with medication effect (aspirin and dipyridamole). ADP response is high for this patient with TAH implantation. Increased dosage of aspirin is suggested to keep antiplatelet medications in therapeutic range. CPT: 85576 x5.

116. Platelet aggregation, total artificial heart (TAH) therapeutic range

Platelet aggregation study results show markedly decreased aggregation with arachidonic acid and moderately decreased with epinephrine. There is also moderate decrease with adenosine diphosphate (ADP). Aggregation with high ADP concentration (50 µM/mL) is adequate (79%). Aggregation with collagen and ristocetin is adequate.

Impression: findings are consistent with platelet dysfunction. This pattern is consistent with medication effect (aspirin and dipyridamole in this patient with TAH implantation). The results indicate that antiplatelet medications are in therapeutic ranges per TAH protocol. CPT: 85576 x5.

117. Platelet aggregation, aspirin, and uremia

Platelet aggregation study results show decreased aggregation to arachidonic acid (AA), adenosine diphosphate (ADP), collagen, epinephrine, and ristocetin. Loss of secondary aggregation is observed with ADP and epinephrine.

Impression: dysfunctional platelets. The decreased aggregation to AA, ADP, collagen, and epinephrine is most likely due to medication effect (this patient is taking aspirin). The pattern of decreased aggregation to all agonists observed in this study is not specific but it is typically observed in chronic renal insufficiency (this patient has a creatinine level of 4.0 and blood urea nitrogen level of 93). Clinical correlation is suggested. CPT: 85576 x5.

118. Clopidogrel bisulfate effect

Platelet aggregation study results show decreased aggregation with arachidonic acid, adenosine diphosphate (ADP) (all 3 concentrations), collagen, and epinephrine. Loss of secondary aggregation is observed with ADP and epinephrine. Aggregation with ristocetin is adequate. Impression: findings are consistent with platelet dysfunction. This pattern is consistent with medication effect (nonsteroidal anti-inflammatory drugs [NSAIDs] and ADP-P2Y12 inhibitor). The patient was taking aspirin and clopidogrel bisulfate before admission to the hospital on October 29, 2013. After admission, the patient took only aspirin. The decreased aggregation with ADP (49%) at high concentration (50 µM/ml) indicates the inhibitory effect of clopidogrel bisulfate. CPT: 85576 x5.

119. Platelet aggregation, abciximab

Platelet aggregation study results show markedly decreased aggregation with arachidonic acid, adenosine diphosphate (ADP), collagen, and epinephrine. Aggregation with ristocetin is adequate. Impression: findings are consistent with platelet dysfunction. This pattern is consistent with the effects of abciximab, which the patient is taking. CPT: 85576 x5.

Appendix 2: Thromboelastograph Templates

1. Normal

   Thromboelastography results are essentially within or close to reference ranges. These indicate adequate hemostasis. If there is evidence of bleeding, consider anatomical or surgical bleeding. CPT:85390.

2. Factor deficiency/heparin/coumadin

   Thromboelastography results show markedly prolonged reaction time (R value). This finding is suggestive of factor deficiency or anticoagulants. In case of factor deficiency, transfusion of fresh-frozen plasma is suggested (10-20 ml/kg body weight, or 4-6 U for an average adult). CPT:85390.

3. Thrombocytopenia/platelet dysfunction

   Thromboelastography results show markedly decreased value of maximum amplitude (MA). This finding is suggestive of severe thrombocytopenia or platelet dysfunction. For thrombocytopenia or other platelet defects, transfuse with platelets (6 U/70 kg body weight). CPT:85390.

4. Thrombocytopenia/platelet dysfunction, mild

   Thromboelastography results show slightly decreased value of maximum amplitude (MA)]. This finding is suggestive of mild thrombocytopenia or platelet dysfunction. For thrombocytopenia or other platelet defects, transfuse with platelets (6 U/70 kg body weight). CPT:85390.

5. Fibrinolysis

   Thromboelastography results show markedly prolonged value of lysis after 30 minutes (Ly30). This finding is suggestive of fibrinolysis, therapeutic (tissue plasminogen activator [tPA], streptokinase, or urokinase) or pathologic. For pathologic fibrinolysis, epsilon-aminocaproic acid (EACA) (5 g/70 kg body weight) or other antifibrinolytics is suggested. CPT:85390.

6. Platelet hypercoagulation/fibrinolysis

   Thromboelastography results show increased values of maximum amplitude (MA) and prolonged value of lysis after 30 minutes (Ly30). These findings are suggestive of platelet hypercoagulation and fibrinolysis, therapeutic (tissue plasminogen activator [tPA], streptokinase, or urokinase) or pathologic. For pathologic fibrinolysis, antifibrinolytics are contraindicated because microvascular thrombosis may be exacerbated. CPT:85390.

7. Hypofibrinogenemia/thrombocytopenia/platelet dysfunction

   Thromboelastography results show decreased value of angle alpha and maximum amplitude (MA). These findings are suggestive of hypofibrinogenemia vs thrombocytopenia or platelet dysfunction. Transfusion with cryoprecipitate is suggested (6 U/70 kg body weight). For thrombocytopenia or other platelet defects, transfuse with platelets (6 U/70 kg body weight). CPT:85390.

8. Factor deficiency/heparin/coumadin and hypofibrinogenemia

   Thromboelastography results show markedly prolonged value of reaction time (R) and markedly decreased value of angle alpha. These findings are suggestive of factor deficiency (or anticoagulants) and severe hypofibrinogenemia. In case of factor deficiency, transfusion of fresh-frozen plasma is suggested (10-20 ml/kg body weight, or 4-6 U for an average adult). For hypofibrinogenemia, transfusion with cryoprecipitate is suggested (6 U/70 kg body weight). CPT:85390.

9. Primary and secondary hemostatic disorders

   Thromboelastography results show markedly prolonged value of reaction time (R), markedly decreased value of maximum amplitude (MA) and angle alpha. This finding is suggestive of defects in primary hemostasis (platelets) and secondary hemostasis (clotting factors). Transfusion with multiple blood components is suggested: fresh-frozen plasma (10-20 ml/kg body weight, or 4-6 U for an average adult), cryoprecipitate (6 U/70 kg body weight), and platelets (6 U/70 kg body weight). CPT:85390.

10. Platelet and enzymatic hypercoagulation

    Thromboelastography results show slightly shortened value of reaction time (R) and slightly increased values of angle alpha and maximum amplitude (MA). These findings are suggestive of mild platelet and enzymatic hypercoagulation, which may be observed in early phase of disseminated intravascular coagulation (DIC). Monitoring for DIC using a DIC panel may be indicated. CPT:85390.

11. Platelet hypercoagulation

    Thromboelastography results show increased values of maximum amplitude (MA). This finding is suggestive of platelet hypercoagulation. Note: the platelet count of the patient is elevated, at 1031. CPT:85390.

12. Enzymatic hypercoagulation

    Thromboelastography results show shortened value of rection time (R) and slightly increased value of angle alpha. These findings are suggestive of enzymatic hypercoagulation. CPT:85390.

13. Disseminated intravascular coagulation (DIC) with hypocoagulable state (stage 2)

    Transfusion with fresh-frozen plasma (2 U/70 kg body weight), cryoprecipitate (6 U/70 kg body weight), and platelets (6 U/70 kg body weight) is recommended. CPT:85390.

14. Platelet hypercoagulation

    Thromboelastography results show increased values of angle alpha and maximum amplitude (MA). These findings are suggestive of platelet hypercoagulation. CPT:85390.

15. Thrombocytopenia or platelet dysfunction /fibrinolysis

    Thromboelastography results show markedly decreased value of maximum amplitude (MA). This finding is suggestive of severe thrombocytopenia or platelet dysfunction. Thromboelastography results also show markedly prolonged value of Ly30. This finding is suggestive of fibrinolysis, therapeutic (tissue plasminogen activator [tPA], streptokinase, or urokinase) or pathologic. It is suggested that in case of uremia, the patient should be prescribed desmopressin acetate (DDAVP) (0.3 µg/kg body weight). For thrombocytopenia or other platelet defects, transfuse with platelets (6 U/70 kg body weight). For pathologic fibrinolysis, epsilon-aminocaproic acid (EACA) (5 g/70 kg body weight) or other antifibrinolytics is suggested. CPT:85390.

16. Mild hypofibrinogenemia

    Thromboelastography results show slightly decreased value of angle alpha. This finding is suggestive of mild hypofibrinogenemia. Transfusion with cryoprecipitate is suggested (6 U/70 kg body weight). CPT:85390.

17. Mild platelet hypercoagulation

    Thromboelastography results show slightly increased values of angle alpha and maximum amplitude (MA)). These findings are suggestive of mild platelet hypercoagulation. CPT:85390.

18. Enzymatic hypercoagulation

    Thromboelastography results show shortened value of reaction time (R). This finding is suggestive of enzymatic hypercoagulation. CPT:85390.

19. Hypofibrinogenemia vs thrombocytopenia or platelet dysfunction, also hyperfibrinolysis

    Thromboelastography results show markedly decreased value of maximum amplitude (MA) and angle alpha, as well as markedly increased value of lysis after 30 minutes (LY30). This finding is suggestive of hypofibrinogenemia vs thrombocytopenia or platelet dysfunction, as well as hyperfibrinolysis: therapeutic (tissue plasminogen activator [tPA], streptokinase, or urokinase) or pathologic. Transfusion with multiple blood components are suggested: cryoprecipitate (6 U/70 kg body weight), and platelets (6 U/70 kg body weight). For pathologic fibrinolysis, epsilon-aminocaproic acid (EACA) (5 g/70 kg body weight) or other antifibrinolytics is suggested. CPT:85390.

20. Increased level of fibrinogen

    Thromboelastography results show increased value of angle alpha. This finding is suggestive of increased level of fibrinogen. CPT:85390.

21. Normal

    Thromboelastography results are within reference ranges. These indicate adequate hemostasis. If there is evidence of bleeding, consider anatomical or surgical bleeding. CPT:85390.

22. Disseminated intravascular coagulation (DIC)

    Thromboelastography results show markedly prolonged value of R, markedly decreased value of maximum amplitude (MA) and angle alpha, and markedly prolonged value of lysis after 30 minutes (Ly30). This finding is suggestive of defects in primary hemostasis (platelets) and secondary hemostasis (clotting factors), as well as hyper fibrinolysis, such as observed in DIC. Transfusion with multiple blood components is suggested: fresh-frozen plasma (10-20 ml/kg body weight, or 4-6 U for an average adult), cryoprecipitate (6 U/70 kg body weight), and platelets (6 U/70 kg body weight). For pathologic fibrinolysis, epsilon-aminocaproic acid (EACA) (5 g/70 kg body weight) or other antifibrinolytics is suggested. CPT:85390.

23. Platelet hypercoagulation/hyperfibrinolysis

    Thromboelastography results show increased values of angle alpha and maximum amplitude (MA). These findings are suggestive of platelet hypercoagulation. Thromboelastography results also show prolonged value of Ly30. This finding is suggestive of fibrinolysis: therapeutic (tissue plasminogen activator [tPA], streptokinase, or urokinase) or pathologic. For pathologic fibrinolysis, antifibrinolytics are contraindicated because microvascular thrombosis may be exacerbated. CPT:85390.

24. Platelet and enzymatic hypercoagulation/hyperfibrinolysis

    Thromboelastography results show shortened value of reaction time (R) and increased values of angle alpha and maximum amplitude (MA). These findings are suggestive of platelet and enzymatic hypercoagulation. Lysis after 30 minutes (LY30) is also markedly increased, consistent with hyperfibrinolysis. For pathologic fibrinolysis, antifibrinolytics are contraindicated because microvascular thrombosis may be exacerbated. CPT:85390.

25. Early phase of disseminated intravascular coagulation (DIC)

    Thromboelastography results show shortened value of reaction time (R) and increased value of maximum amplitude (MA). These findings are suggestive of platelet and enzymatic hypercoagulation, which may be observed in the early phase of DIC. Monitoring for DIC using the DIC panel may be indicated. CPT:85390.

26. Hypofibrinogenemia and hyperfibrinolysis

    Thromboelastography results show decreased value of angle alpha. This finding is suggestive of hypofibrinogenemia. Transfusion with cryoprecipitate is suggested (6 U/70 kg body weight). Thromboelastography results also show prolonged value of lysis after 30 minutes (Ly30). This finding is suggestive of fibrinolysis: therapeutic (tissue plasminogen activator [tPA], streptokinase, or urokinase) or pathologic. For pathologic fibrinolysis, epsilon-aminocaproic acid (EACA) (5 g/70 kg body weight) or other antifibrinolytics are suggested. CPT:85390.

27. Platelet/enzymatic hypercoagulation and hyperfibrinolysis

    Thromboelastography results show shortened value of reaction time (R) and increased values of angle alpha and maximum amplitude (MA). These findings are suggestive of platelet and enzymatic hypercoagulation. LY30 is also increased, consistent with hyperfibrinolysis. For pathologic fibrinolysis, antifibrinolytics are contraindicated because microvascular thrombosis may be exacerbated. The thromboelastography parameters are suggestive of an early phase of disseminated intravascular coagulation (DIC). Monitoring for DIC using the DIC panel may be indicated. CPT:85390.

28. Enzymatic hypercoagulation

    Thromboelastograph (with heparinase) results show shortened value of reaction time (R). This finding is suggestive of enzymatic hypercoagulation. CPT:85390.

29. Markedly prolonged reaction time (R) and markedly decreased maximum amplitude (MA), angle alpha

    Thromboelastography results show markedly prolonged value of reaction time (R). This finding is suggestive of defects in secondary hemostasis (clotting factors) or anticoagulant effect. If patient is not taking anticoagulant medication, transfusion with blood components is suggested: fresh-frozen plasma (10-20 ml/kg body weight, or 4-6 U for an average adult). Maximum amplitude (MA) and angle alpha are also markedly decreased but these results are not diagnostic due to markedly prolonged R. CPT:85390.

30. Heparin effect: markedly prolonged reaction time (R) and markedly decreased maximum amplitude (MA), angle alpha; all normalized with heparinase

    Thromboelastography results show markedly prolonged value of reaction time (R) and markedly decreased value of maximum amplitude (MA) and angle alpha. Thromboelastography with heparinase was performed, which shows normal values of all parameters (R, angle alpha, MAs). Impression: findings are consistent with heparin effect. CPT:85390.

31. Heparin effect: markedly prolonged reaction time (R) and markedly decreased maximum amplitude (MA), angle alpha; all normalized with heparinase

    Thromboelastography with heparinase was performed, which shows normal values of all parameters (R, angle alpha, MAs, Ly30). Thromboelastography (without heparinase) results show markedly prolonged value of R and markedly decreased value of MA and angle alpha. Impression: findings are consistent with heparin effect. CPT:85390.

32. Normal results of thromboelastography (performed with heparinase)

    Thromboelastography (performed with heparinase) results are within reference ranges. These indicate adequate hemostasis; also, there is no evidence of heparin in the specimen. If there is evidence of bleeding, consider anatomical or surgical bleeding. CPT:85390.

33. Enzymatic hypercoagulation and thrombocytopenia or platelet dysfunction

    Thromboelastography results show shortened value of reaction time (R) and decreased value of maximum amplitude (MA). These findings are suggestive of enzymatic hypercoagulation and thrombocytopenia or platelet dysfunction. CPT:85390.

34. Enzymatic hypercoagulation and hyperfibrinolysis

    Thromboelastography results show shortened value of reaction time (R) and increased values of angle alpha. These findings are suggestive of enzymatic hypercoagulation. LY30 is also increased, consistent with hyperfibrinolysis. For pathologic fibrinolysis, antifibrinolytics are contraindicated because microvascular thrombosis may be exacerbated. The thromboelastography parameters are suggestive of an early phase of disseminated intravascular coagulation (DIC). Monitoring for DIC using a DIC panel may be indicated. CPT:85390.

35. Enzymatic hypercoagulation and thrombocytopenia or platelet dysfunction

    Thromboelastography results show shortened value of reaction time (R), increased value of angle alpha, and decreased value of maximum amplitude (MA). These findings are suggestive of enzymatic hypercoagulation and thrombocytopenia or platelet dysfunction. For thrombocytopenia or other platelet defects, transfuse with platelets (6 U/70 kg body weight). CPT:85390.

36. Enzymatic hypocoagulation or anticoagulant

    Thromboelastography results show markedly prolonged value of reaction time (R). This finding is suggestive of defects in secondary hemostasis (clotting factors) or anticoagulant effect. If patient is not taking an anticoagulant, transfusion with blood components is suggested: fresh-frozen plasma (10-20 ml/kg body weight, or 4 U for an average adult). Maximum amplitude (MA)and angle alpha are also markedly decreased, but these results are not diagnostic due to markedly prolonged R. Testing for fibrinogen and platelet count is suggested if clinically indicated. CPT:85390.

37. Hyperfibrinogenemia and hyperfibrinolysis

    Thromboelastography results show increased value of angle alpha. This is suggestive of hyperfibrinogenemia. Thromboelastography results also show prolonged value of lysis after 30 minutes (Ly30). This finding is suggestive of fibrinolysis: therapeutic (tissue plasminogen activator [tPA], streptokinase, or urokinase) or pathologic. For pathologic fibrinolysis (early disseminated intravascular coagulation [DIC]), antifibrinolytics are contraindicated because microvascular thrombosis may be exacerbated. Clinical correlation is suggested. CPT:85390.

38. Heparin effect

    Thromboelastography results show markedly prolonged value of reaction time (R), markedly decreased value of angle alpha and maximum amplitude (MA). Thromboelastography with heparinase was performed, which shows normal values for all parameters (R, angle alpha, MA). Impression: findings are consistent with heparin effect.