如 何 診 斷 出 血 病 患

摘　　要

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當我們看到一個容易出血或止血異常的病人，首先要確定他的出血是全身或局部性血管的問題。如果是全身性問題也要確定他真的患有異常出血或止血不正常，流血過長的情形。一個人受了傷會出血是每個人均會經驗到的事情，不足爲怪，問題在他是否在常識認知的正常時間內（五分鐘）止血。這要靠詢問病史的技巧，在日常生活中常碰到打針，輕微受傷，拔牙等足以考驗病人止血能力的事情(stress test)都要一一詢問病人做爲評估病人是否有異常出血問題的判斷，而不能依賴病人來告訴你，因爲他常常不瞭解或忽略異常出血的事情。

在詢問病史當中，需要瞭解他的異常出血是從小就有或以後才出現，有無家族病史，以確定是先天性或後天性的情形。如果是後天性的原因，有無肝病、尿毒症、血液病或全身性紅斑性狼瘡等的病史，或者與葯物有關包括 Aspirin, NSAID, Heparin and oral anticoagulants 等。

有了這些臨床資料，再加上對於出血症的鑑別診斷，可以區分是因血小板血管問題或血液凝固問題所引起。如此對病人有了充份的瞭解和把握，下一個步驟要如何檢查病人才能獲得正確的診斷便會有一個方向。經過身體檢查後便可以決定做什麼檢查。包括 Platelet cont, BT, PT and PTT 以及接下來的特殊檢查。

以上事例很清楚驗證詢問病史、身體檢查是很重要的課題，比接下來的實驗室檢查有時還要重要，甚至不必檢查也可以有正確的診斷，一般說來三者相輔相成才有好的結果。

Approach to bleeding

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The evaluation of patients for the possibility of a systemic bleeding diathesis is usually undertaken in one of three general clinical settings.

1. Patients with active or past histories of unexpected bleeding require diagnostic studies.

   A systemic coagulopathy or by an anatomic or mechanical problem of the vasculature.
2. Patients may be found incidentally to have abnormalities of screening laboratory tests of hemostasis.
   (1) Whether or not the finding is clinically relevant. e.g., a hereditary deficiency of one of the contact activation coagulation factors.
   (2) Patients with lupus anticoagulant
   (3) Patients with heparin induced thrombocytopenia
3. Patients without previous coagulation problems frequently undergo routine testing for bleeding rick prior to the performance of invasive procedures or surgery.

The above examples illustrate the critical importance of considering the history, physical examination, and screening laboratory tests or complementary facets of the clinical approach to patients with suspected systemic coagulopathies. Each part alone is not only inadequate but actually be misleading.

1. Screening history

   A history taken to evaluate hemostasis should answer these questions.

   (1) Has the patient experienced abnormal bleeding or bruising? If so, are symptoms of recent origin, suggesting an acquired disorder, or do they date back to childhood, suggesting a hereditary disorder?
      (i) It is critical to probe systematically for the patient's hemostatic responses to specific challenges to the coagulation system that may occur throughout life ——— a "stress test" of hemostasis.
      (ii) The history of normal hemostasis following events is just as important to record as episodes of excessive bleeding.
      (iii) The finding that a patient has recently withstood surgery without bleeding complications in many ways constitutes a better test of systemic hemostasis than any laboratory test could possibly provide.
   (2) Is there a history of acquired disorder that could impair hemostasis for example, chronic liver disease, systemic lupus erythematosus, uremia, a hematologic malignancy?

(3) Is the patient taking a drug that could interfere with hemostasis?

(i) Aspirin

(ii)Other NSAID

(iii)Heparin or oral anticoagulants

(4)Have other member of the family bled abnormally?

In general, patients with defects of primary hemostasis (e.g., thrombocytopenia, or abnormal platelet function and platelet-vessel wall interactions) tend to exhibit superficial hemorrhage into the skin or mucous membranes that occurs either spontaneously or immediately after trauma and usually stops, after compression to the wound . In contrast, patients with abnormalities of secondary hemostasis (e.g., coagulation factor deficiencies) characteristically develop extensive deep tissue hemorrhage, such as hemathroses, hematomas , or retroperitoneal bleeding, that may be delayed in onset for up to 24 – 48 hours after surgery or trauma, and may stop after compression but rebleeds after release of the pressure. The additional history of delayed wound healing may suggest factor XIII deficiency. Patients with complex coagulopathies (e.g., DIC, liver failure) may exhibit mixed patterns of bleeding that are associated with both primary and secondary hemostasis defects.

2. Physical examinations

(1) Evidence of abnormal bleeding into the skin

Large ecchymoses (bruises), petechise, small purpuric spots with a firm center (palpable purpnra), bleedings at the puncture sites.

(2) Abnormal elasticity of the skin and hyperextensibilily of joints or evidence of a hereditary connective tissure disorder associated with vascular bleeding.

(3) Stigmatas of chronic liver disease :spider angiomas, palmar erythema, dilated abdominal veins, an enlarged liver and spleen.

3. Laboratory tests

(1) Screening tests

Platelet count, bleeding time, PT and PTT.

(2) Specialized tests

(i) A diagnostic test when one or more of the screening tests is abnormal.

(ii) Certain specialized tests may be indicated when screening tests are normal but the clinical index of suspicion for an unusual systemic coagulopathy is high.

4. Individual laboratory tests

(1) Platelet count

(i)The report of a low platelet count determined by electronic counting machines must always be followed by examination of the peripheral blood smear.

(ii)Pseudothrombocytopenia is the phenomenon of a spuriously low platelet

count that may be due to a variety of laboratory artifacts and should be suspected when an unexpectedly low platelet count is reported in a patient who has had no clinical bleeding problems.

(2) Bleeding time

(i) It is widely recognized as the best screening test of in vivo primary hemostasis independent of blood coagulation reaction.

(ii) Its role as a routine preoperative screening test to predicl surgical hemorrhage has been seriously challenged.

(iii) A bleeding time technique in which a standardized incision is made and in which the hemostatic plugs must hold against a back pressure should be used.

(iv) There is generally a direct inverse correlation between the degree of thrombocytopenia with a platelet count below 100,000/mm^3 and the degree of prolongation of the bleeding time until the platelet cound falls below about 10,000/mm^3.

(v) Severe anemia causes prolongation of the bleeding time.

(vi) Exaggerated prolongations of the bleeding time are observed following the ingestion of aspirin in individuals who have mild underlying disorders of primary hemostasis.

(vii) The sensitivity and specificity of the bleeding time as a test of primary hemostasis have been challenged.

(3) PT&PTT

(i) False-positive prolongation of PT and PTT may occur as a result of underfilling the test tube.

(ii) PTT may be falsely prolonged in patient with polycythemia or when blood is contaminated with even trace amount of heparin.

(iii) Thromboplastins prepared from human brain are generally more responsive to reduction in the coagnlation factors affected by PT than those prepared from rabbit brain.

(iv) Marked variation in the sensitivity of the PTT reagents to the lupus anticoagulant due to the variation in the choice of PTT activator as well as in the amount, type and physical properties of the phospholipid used in the assay has been documented.

(v) The sensitivity of PTT to detect the coagulation factor deficiency is about 30% for factor VIII and IX, PT is significantly affected by factor VII levels of lese than 50%.