OUTLINE

• OVERVIEW OF HEMOSTASIS
• ETIOLOGY & OVERVIEW
• APPROACH IN DENTAL SETTING
A: OVERVIEW OF HEMOSTASIS
HEMOSTASIS

• Refers to the stoppage of blood
• Normal when it seals a blood vessel to prevent blood loss/hemorrhage
• Abnormal when it causes inappropriate blood clotting, or is insufficient to stop blood flow from the vasculature
• Balance of pro-coagulants and anti-coagulants that maintains:
  • Blood flow
  • Integrity of the vasculature
Hemostasis Mechanism

• Vessel spasm
  • Transient -1 min
  • TXA$_2$ -prostaglandin f.Plts-vasoconstriction

• Formation of the platelet plug
  • Plts in contact with vessel wall
  • Adhesion(vWF) and aggregation(granules) Plts

• Blood coagulation/insoluble fibrin clot
  • Activation of coagulation pathway
  • Fibrinogen to Fibrin mesh
Hemostasis Mechan.

• Clot retraction
  • Occurs 20-60min after a clot has formed
  • Squeezes serum from the clot
  • Joining the edges of the broken vessel

• Clot dissolution
  • Fibrinolysis - begins shortly after its formation
  • Allows blood flow to be reestablished
  • Balance between activators and inhibitors
  • Plasminogen to Plasmin - digest fibrin strands
  • Allows permanent tissue repair
Coagulation Cascade

• Continuous factor activation and coordinated assembly of enzymes kept in check by circulating anticoagulant proteins

• Enzyme complexes: serine proteases, co-factors, zymogen substrates assembled in a phospholipid membrane surface

• Formation of complexes is low under normal circumstances, circulating anti-coagulants inactivates them to avoid clot formation
Coagulation Cascade

- Liver is the major site of synthesis
- In severe liver disease all coagulation factors are diminished except factor VIII
- Vitamin K dependent factors: II, VII, IX, and X
- Protein C and S are naturally occurring anticoagulants which synthesis is also vitamin K dependent
- Warfarin blocks liver uptake of vitamin K, decreasing the synthesis of vitamin K dependent factors
Coagulation cascade

- Artificially divided into extrinsic and intrinsic pathways that converge into the common pathway leading to thrombin and fibrin generation
- Extrinsic pathway assessed in the laboratory by the Prothrombin time (PT)
- PT-sensitive to deficiencies of factors II, VII, V, and X, all these associated with bleeding complications
- PT-to monitor warfarin (coumadin) therapy
Coagulation cascade

• PT-INR—international normalized ratio, the degree of prolongation of PT by warfarin depends on the strength of the reagents used in the lab, which could vary among labs.

• INR created To standardize the variations and allow for global application of anticoagulant recommendations

• INR = patient PT/mean control PT
Coagulation cascade

- PTT- partial thromboplastin time, assess the intrinsic pathway
- Sensitive to deficiencies of factor, II, V, VIII, IX, X, XI
- To monitor Heparin therapy
- Heparin binds to antithrombin III, and increases its ability to inactivate thrombin, factor Xa and others
Intrinsic Pathway
- Surface contact
  - XII → XIIα
  - Fitzgerald
    - XI → XIα
    - PF3
      - IX → IXα
      - Ca++
    - Ca++
    - PF3
    - VIII → VIIIα

Extrinsic Pathway
- III (Tissue thromboplastin)
  - Ca++
    - VIIα ← VII

Common Pathway
- X → Xα
  - V → Vα
    - Ca++, PF3
      - Prothrombin (II) → Thrombin
      - XIII
        - XIIIα
      - Fibrinogen (I) → Fibrin (monomer)
        - Stable fibrin (polymer)
Coagulation cascade
Fibrinolysis

• Plasminogen present in the blood inactive, is activated to plasmin by plasminogen activators, formed in the vascular endothelium, liver and kidneys
• Plasmin digests the fibrin strands and certain clotting factors such as fibrinogen, V, VIII, II, and XII
• Plasmin is inactivated by alpha2-plasmin inhibitor which limits fibrinolysis to the local clot
Fibrinolysis

• Two naturally occurring plasminogen activators:
  • Tissue-type PA
  • Urokinase-type PA
• Released in response to:
  • Vasoactive drugs
  • Venous occlusion
  • Increased body temperature
  • exercise
CLOTTING VS ANTICLOTTING MECHANISMS
B: ETIOLOGY AND OVERVIEW OF BLEEDING DISORDERS
Evaluation of Bleeding disorders

• Careful history
  • Description of bleeding, epistaxis, menorrhagia, hematoma formation
  • Circumstances associated, trauma, dental procedure, surgery, expontaneous
  • Blood products required to stop bleeding
  • Concomitant illnesses, liver Ds
  • Family Hx of bleeding: query several generations and second degree relations such as maternal uncles when Hemophilia is suggested in a boy
Evaluation of Bleeding

- **Physical exam**
  - Origin of the bleeding/
    distinguish between small vessel
    bleeding(petechial-pinpoint) and
    large vessel (purpura-hematoma)
  - Petechiae skin/mucosae
    associated with plts
    disorders(QLT/QNT), vascular
    abn, vWF def. Bleeding into the
    tissues, organs, and joints
    associated with factors deficiency
    (hemophilias)
Evaluation of Bleeding

- **Laboratory evaluation:**
  - Screening test-Plt count and Diff.
  - PT / PTT to determine factor def.
  - Mixing studies, distinguishes between factor def. and circulating inhibitors
  - Thrombin time, assays fibrinogen level and its functional capability
  - In vivo bleeding time, for plt function, prolonged by thrombocytopenia <100,000/mcl and qualitative plts defects
  - In vitro bleeding time, PFA100, prolonged by qualitative defect
Causes of bleeding

1. Vascular
2. Thrombocytopenia or abn. Plt. Functions
3. Low levels of multiple coagulation factors from vit K def. or liver disease
4. Single factor def. either inherited or acquired
5. Consumptive coagulopathies such as Disseminated Intravascular Coagulation (DIC)
6. Circulating inhibitors to coagulation factors, AB to factor VIII
1-Vascular causes of bleeding

- **Acquired Vascular purpura** (bruising) - caused by intrinsic struct. abn. of blood vessels
- **Senile purpura** - abn. subc. tissues in older patients, also seen in steroid therapy - atrophy subc. tissue and fragile blood vessels, collagen breakdown
- **Scurvy purpura/vit C def.** - bleeding around individual hair fibers, and in classic saddle pattern over the upper thighs
  - Edentulous patients?
  - Bleeding gums in scurvy caused by gingivitis not by subc. deffect.
Vascular causes -2

- **Congenital defects of vessel wall** causing bruising:
  - *Pseudoxanthoma elasticum* (abn. elastic fibers) severe GI + GU bleeding + bruising of the skin
  - *Ehlers-Danlos syndrome* (abn. collagen in vessels and subc.tissue) bruising of the skin
  - *Hereditary hemorrhagic telangetasias* (osler-weber-rendu syndrome) degeneration vessel wall-angiomatous lesions/ blisters in lips and GI bleeding-iron def.anemia
Vascular causes - 3

- By inflammatory infiltration of blood vessels (vasculitis)
- Sudden onset of Raised palpable purpuric rash plus fever
- Septic vasculitis - meningococcemia
- Aseptic vasculitis - Henoch Schonlein purpura
2-Bleeding caused by platelet disorders

Thrombocytopenia

- Decreased production
  - nutritional B12/folate deficiency
  - congenital Alport’s synd. Fanconi’s anemia May-Hegglin anomaly
  - marrow damage
    - Aplastic anemia
    - cytotoxic
    - Malignancy

- sequestration
  - splenomegaly
  - liver Ds.
  - Malignancy
  - Myelofibrosis

- increased destruction
  - Immune-Med.
    - ITP
    - Drug-induced
    - Systemic-SLE/HIV
  - Non-Immune
    - DIC
    - TTP
    - Antiphospholipid

- hemodilution
  - massive transfusion
  - Cardiopulmonary bypass
Idiopathic-autoimmune Thrombocytopenic Purpura (ITP)

• Essential of Dx
  • Isolated thrombocytopenia
  • Other cell lines are normal
  • Spleen not palpable
  • Nl bone marrow /Nl or increased megakaryocytes
ITP

- Clinical diagnosis
- IgG ab formed and binds to Plts.
- Plts are not destroyed by direct lysis
- Ab-coated Plts removed by the spleen
- Childhood, after viral infection (varicella) and self limited
- More chronic course in adults
- Peak incidence 20-50 years
ITP

- 2:1 female predominance
- Mucosal or skin bleeding: epistaxis, oral bleeding, menorrhagia, purpura and petechiae
- Systemically well, not febrile
- No organomegally
ITP-Lab Findings

- Hallmark-Thrombocytopenia, sometimes less than 10-20,000/mcl
- Large Plts (megathrombocytes)
- 10% Evans’s syndrome - decreased Plts + autoimmune Hemolytic anemia
- Normal bone marrow
- Normal coagulation studies
ITP-Diff.Dx.

• Myelodysplasia - abn.marrow cells
• DIC, TTP, HUS, and sepsis will have systemic symptoms
• Use of drugs such as sulfonamides should be questioned - possible cause
• Drug induced - stops after DC the medications
ITP-Treatment

• Self limiting in children-spontaneous remission
  • 10% recurrent thrombocytopenia, resolved by splenectomy

• Adults
  • Prednisone 1-2mg/kg/day
  • No Plt transfusion unless significant bleeding
  • Severe thrombocytopenia <5000/mcl or surgery or severe bleeding-
    • Methylprednisolone 1g/d for 3 days
    • IVIG 2g/kg in divided doses over 2-5 days
  • Splenectomy
  • If no results - Cytoxan, Rituxan
Heparin Induced Thrombocytopenia. HIT

- Immune mediated, ab against heparin-plt factor 4 complex
- Catastrophic thrombotic complications
- More frequent with UFH than with LMWH
- Cross-reaction between the two
- Discontinuation of Heparin is critical
- Short term anticoagulation: with direct thrombin inhibitors, Lepirudin, Argatroban
- Warfarin should not be given on a short-term basis or alone without thrombin inhibitor coverage increased incidence of limb thrombosis
Disseminated Intravascular Coagulation (DIC)

- Essential of Diagnosis
  - Consumptive thrombocytopenia
  - Underlying serious illness
  - Macroangiopathic hemolytic anemia may be present
  - Hypofibrinogenemia, thrombocytopenia, fibrin degradation products and prolonged prothrombin time
Disseminated Intravascular Coagulation (DIC) Causes

- Sepsis or endotoxins
  - Gram-negative
- Malignant disease
  - Adenocarcinoma, Acute promyelocytic leukemia
- Primary vascular disorders
  - Vasculitis, giant hemangioma, aortic aneurysm, cardiac mural thrombus
- Exogenous causes
  - Snake venom,
- Tissue damage
  - Trauma, closed head injury, burns, hypo-perfusion and hypotension
DIC - DEATH IS COMING

- Overwhelming production of Thrombin and Fibrin
- Deposition of Fibrin in vasculature
- Inadequate Fibrinolysis
- Thrombotic or microangiopathic vasculopathy
- Organ damage (clot -ischemia)
- Exhausting the Bone marrow and liver synthetic capability
- Thrombocytopenia and decreased coag. factors
- Mucosal bleeding (GI), oozing from IV puncture sites
DIC symptoms

• Thrombosis and bleeding
• Thrombosis -digital ischemia and gangrene
• Spontaneous bleeding and oozing from IV sites and wounds
• Trousseau’s syndrome: refers to sub-acute DIC in cancer patients, with recurrent thrombosis
DIC Laboratory

- Hypofibrinogenemia
- Elevated fibrin degradation products, D-dimer
- Thrombocytopenia
- Prolonged PT and possible PTT
- Fragmented RBCs in slide
DIC treatment

• Diagnosis and Treatment underlying disorder (antibiotics, chemotherapy)
• Mild cases- no specific therapy
• Heparin therapy is contraindicated in cases that any increased of bleeding is unacceptable (neurological procedures)
• Heparin therapy-helpful in cases when DIC is causing serious clinical consequences
• Heparin dose 500-750 units per hour
DIC treatment

• Replacement therapy (supportive)
  • Maintain Plts level 30-50,000/mcl
  • Fibrinogen - Cryoprecipitate (1 unit to rise Fibrinogen by 6-8mg/dl)
  • Coagulation factors - fresh frozen plasma
TTP-thrombotic thrombocytopenic purpura

- Autoimmune activation and clearance of Plts due to increased Plt adhesion w/o activation of Coag. cascade
- Absent or decreased ADAMTS13
  - vWF-cleaving protease
- Inherited or acquired
- NI PT and PTT
- Associated with pregnancy, chemotherapy and HIV infection
TTP

- Pentad
  - Fever
  - Thrombocytopenia
  - Microangiopathic hemolysis
  - Neurologic symptoms
  - Renal insufficiency

- Schistocytes and increased serum LDH

- Clinical Dx

- Rx
  - Plasmapheresis-plasma exchange-replaced with fresh frozen plasma
  - Steroids and antiplatelet drugs
  - ASA, dipyridamole
  - Purified or recombinant ADAMTS13 possible in the future
HUS - Hemolytic Uremic Syndrome

- Sometimes considered part of TTP
- However HUS
  - not associated with neurologic impairment
  - Primary in children
  - In adults with hemorrhagic colitis
    - Toxin produced by E.coli 0157.H7
- Responsive to the same treatment as TTP
3-Bleeding by Qualitative Plts defects

• **Acquired** decreased aggregation: Aspirin (irreversible), NSAIDs (reversible)

• **Congenital:**
  • Rare, Receptors defects: Bernard-Soulier syndrome / Glanzmann’s thrombasthenias
  • Granules Defects:
    • Storage pool ds.-decreased dense granules/ decreased plt aggregation
    • Hermansky-Pudlack syndrome- dense granules defect/associated with oculocutaneous albinism
    • Chediak-Higashi syndrome: rare granule disorder, mild bleeding, partial albinism, recurrent pyogenic infections
    • Gray platelet syndrome: loss of alpha granules, colorless or gray platelets
Qualitative Plts defects

- All the platelets granule disorders are successfully treated by avoiding Aspirin and antiplatelets drugs, hormonal regulation of menses in women, and by Plt transfusion when bleeding occurs.
Von Willebrand’s Disease essentials

• Autosomal dominant pattern of inheritance
• Most common congenital disorder of hemostasis
• vWF in EC and Megakaryocytes
  • In plasma mediates “torquetting” of Plts and adhesion to the damage endothelium
  • Carrier for factor VIII, protection from early clearance
• Mucosal bleeding and Prolonged Btime at baseline or after Aspirin (dec.adhesion)
• Dec.factor VIII level – prolonged PTT
General considerations

- Phenotypically grouped into Three major subtypes
- Most common is type 1 - quantitative decreased in vWF
- Equiv. dec. in VIII and vWF antigen and Rcof activity
- Mild-mod bleeding
- Rx : DDAVP
Von Willebrand’s Disease

• Type 2 – qualitative defect, dec. factor VIII affinity
• Type 3 – complete deficiency,
  • no response to DDAVP and severe bleeding episodes
  • May mimic hemophilia
• Require vWF concentrates
Signs and symptoms

- Affects both men and women
- Most cases mild
- Most bleeding is mucosal, epistaxis, gingival bleeding, menorrhagia
- In most cases incisional bleeding occurs after surgery or dental extractions
- With vWF the clinical picture varies with the degree of deficiency
Laboratory Test

• Platelet number/morphology-normal
• Bleeding time usually prolonged
• Factor VIII coagulant activity
• Factor VIII antigen assay: measures the immunologic presence of vWF.
• Ristocetin cofactor activity assay: measures functional properties of vWF in mediating Plt adhesion
Treatment

• Avoid aspirin
• Patients need to be prepared in case of surgery, perform Btime.
• Desmopressin acetate is useful for mild type I von Willebrand disease
• Should be considered first, second choice factor VIII concentrates
Acquired vWF deficiency

- Abnl. clearance of multimer vWF
- Associated with thrombocytosis:
  - Monoclonal gammopathy
  - Myeloma
  - Malignant lymphoproliferative Ds
  - Critical aortic stenosis
- Rx: treat underlying Ds, IVIG, surgical repair of aortic valve
4. Bleeding/coag.factor def. Hemophilia A

- X-linked recessive pattern with only males affected, rarely affects females
- 6x more common than Hemophilia B
- Deficiency of factor VIII coagulant, quantitatively
- Normal factor VIII antigen
- Severe if factor VIII:C levels are less than 1%, moderate 1-5%, mild >5%
Signs and Symptoms

- Most common SEVERE bleeding disorder
- Second most common CONGENITAL bleeding disorder after vWF Deficiency
- Both severe Hemop.A and B develop in childhood
- Bleeding can occur anywhere, more common into the joints (Hemarthroses), muscles, GI, and retroperitoneal space.
Signs and Symptoms

- **Mild cases**: bleed only after major trauma or surgery
- **Moderate**: bleed with mild trauma or surgery
- **Severe**: bleed spontaneously with severe complications:
  - Severe deformities
  - Arthritis
  - Muscle atrophy
  - Contractures
Laboratory Tests

- PTT is prolonged
- PT: wnl
- Bleeding time: wnl
- Fibrinogen level: wnl
- Level of factor VIII:C decreased
- vWF levels are normal
- Platelet count: wnl
Differential Diagnosis

• Factor IX hemophilia: clinically indistinguishable, factors assays is the only way to differentiate

• Von Willebrand factor: abnormal levels of factor VIII antigen

• Important to ID female carriers in families - low or normal VIII:C and normal levels of factor VIII antigen
Treatment

• Factor VIII concentrates, either heat treated or recombinant. Recombinant is safe and effective, though expensive
• Mild cases can be treated with desmopressin acetate
• Avoid ASA
• Phys.therapy/ortho care/joint replacement
<table>
<thead>
<tr>
<th>Condition</th>
<th>PT</th>
<th>APTT</th>
<th>Bleeding time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemophilia A</td>
<td>Normal</td>
<td>Increased</td>
<td>Normal</td>
</tr>
<tr>
<td>Hemophilia B</td>
<td>Normal</td>
<td>Increased</td>
<td>normal</td>
</tr>
<tr>
<td>Von Willebrand disease</td>
<td>Normal</td>
<td>Increased</td>
<td>increased</td>
</tr>
</tbody>
</table>
Hemophilia B

- X-linked recessive inheritance
- Males only affected
- Low levels of factor IX coagulant activity
- Also known as Christmas disease or factor IX hemophilia
- Most common factor IX quantitatively reduced
Hemophilia B

- Less frequent than classic hemophilia
- Clinically and genetically identical
- PTT is prolonged
- Factor IX levels are reduced
- Other Labs the same as factor VIII hemophilia
Summary

• Vascular fragility
• subcutaneous bleeding
• Factor deficiencies
• spontaneous soft tissue and joint bleeding
• Evaluation begins with
• a good History
• Medical Hx and ROS is important
• PE
• evaluates site of bleeding
• Screening tests:
• Plt count, PT, PTT, and Btime
• If PT or PTT abn,
• mixing studies
• Plt count <20,000/mcl and PT/PTT are
• Bone Marrow biopsy check for nl precursors, if dec.
• underproduction
• If NL - Peripheral destruction
• Run anti-Plts antibodies, not very specific, clinical diagnosis
C: APPROACH TO BLEEDING DISORDER IN DENTAL TREATMENT
Evaluation of bleeding disorders

1. Take history
2. Physical examination
3. Screening clinical laboratory tests
4. Observation of excessive bleeding following a surgical procedure
History

- Bleeding problems in relatives
- Bleeding problems following operations and tooth extractions, trauma
- Use of drugs for prevention of coagulation or pain
- Spontaneous bleeding from nose mouth etc.
Physical examination

➢ Jaundice
➢ Petechiae: < 0.2 cm
➢ Purpura: 0.2 cm-1 cm
➢ Eccymoses: > 1 cm
➢ Spider angioma
➢ Oral ulcer
➢ Hyperplasia of gingiva
➢ Hemarthrosis
# Screening laboratory tests

<table>
<thead>
<tr>
<th>Test</th>
<th>Primary/Secondary</th>
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</thead>
<tbody>
<tr>
<td>1. Platelet count</td>
<td></td>
</tr>
<tr>
<td>2. BT (Bleeding Time)</td>
<td>primary</td>
</tr>
<tr>
<td>3. PT (Prothrombin Time)</td>
<td></td>
</tr>
<tr>
<td>4. aPTT (active Partial Thrombopastin Time)</td>
<td>secondary</td>
</tr>
<tr>
<td>5. TT (Thrombin Time)</td>
<td></td>
</tr>
</tbody>
</table>
Platelet count

➢ Test platelet phase: evaluation of platelet function
➢ Normal (140,000 to 400,000/mm3)
➢ Thrombocytopenia : < 140,000/mm3
➢ Clinical bleeding problem : <50,000/mm3
➢ Spontaneous bleeding with life threatening : <20,000/mm3
PT (Prothrombin Time)

- Activated by tissue thromboplastin
- Tests extrinsic (factor VII) and common (I, II, V, X) pathways
- Normal (11-15 sec)
- Coumarin therapy - PT at 1.5 to 2.5 time
- International normalized ratio = INR, (1) surgery can be done under INR < 3.0 (2) when INR = 3.0-3.5, consultation is needed (3) delay surgery when INR > 3.5
Activated PTT (aPTT)

- Activated by contact activator (kaolin)
- Tests intrinsic and common pathway
- Normal (25-35 sec)
- Heparin therapy - PTT in 50-65 sec range by promote AT III
TT (Thrombin Time)

- Activated by thrombin
- Tests ability to form initial clot from fibrinogen
- Normal (9 to 13 seconds)
<table>
<thead>
<tr>
<th>Condition</th>
<th>Tests Performed</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. No historical bleeding problem</td>
<td>Following surgical procedure</td>
</tr>
<tr>
<td>2. History bleeding problem</td>
<td>PT, aPTT, TT, BT</td>
</tr>
<tr>
<td>3. Aspirin therapy</td>
<td>BT, aPTT</td>
</tr>
<tr>
<td>4. Coumarin therapy</td>
<td>PT</td>
</tr>
<tr>
<td>5. Renal dialysis (heparin)</td>
<td>aPTT</td>
</tr>
<tr>
<td>6. Possible liver disease</td>
<td>BT, PT</td>
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<tr>
<td>7. Chronic leukemia</td>
<td>BT</td>
</tr>
<tr>
<td>8. Long term antibiotic therapy</td>
<td>PT</td>
</tr>
<tr>
<td>9. Vascular wall alteration</td>
<td>BT</td>
</tr>
<tr>
<td>10. Cancer (fibrinogenolysis)</td>
<td>TT</td>
</tr>
</tbody>
</table>

**DENTAL MANAGEMENT OF THE MEDICALLY COMPROMISED PATIENT**
<table>
<thead>
<tr>
<th>condition</th>
<th>Platelet count</th>
<th>BT</th>
<th>PTT</th>
<th>PT</th>
<th>TT</th>
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<tr>
<td>1. Aspirin therapy</td>
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<tr>
<td>2. Coumarin therapy</td>
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<td>-</td>
<td>++</td>
<td>++</td>
<td>-</td>
</tr>
<tr>
<td>3. Heparin therapy</td>
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<td>+</td>
<td>++</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4. Liver disease</td>
<td>+</td>
<td>+</td>
<td>++</td>
<td>++</td>
<td>+</td>
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<tr>
<td>5. leukemia</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
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<tr>
<td>6. Long term antibiotic</td>
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<td>++</td>
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<tr>
<td>7. Vascular wall defect</td>
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<td>-</td>
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<tr>
<td>8. thrombocytopenia</td>
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<td>-</td>
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<td>9. hemophilia</td>
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<tr>
<td>10. fibrinogenolysis</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>++</td>
</tr>
</tbody>
</table>

-: normal, +: may be abnormal, ++: abnormal
Patient at low risk

➢ 1. patient with no history of bleeding disorders, normal examinations, no medications associated with bleeding disorders and normal bleeding parameters

➢ 2. patients with nonspecific history of excessive bleeding with normal bleeding parameters (PT, PTT, BT, platelet count, are within normal time)
Patient at moderate risk

- 1. patients in chronic oral anticoagulant therapy (coumadin)
- 2. patients on chronic aspirin therapy
Patient at high risk

➢ 1. patients with known bleeding disorders
   Thrombocytopenia
   Thrombocytopenia
   Clotting factor defects

➢ 2. Patient without known bleeding disorders
   found to
   have abnormal platelet count, BT, PT, PTT
Dental management of bleeding disorders

➢ Replacement therapy:
  1. platelet concentrate: thrombocytopenia (1 unit = 30,000/uL enough for 1 day)
  2. Fresh frozen plasma: liver disease, Hemophilia B, vWD type III
  3. Factor VIII, IX concentrate: Hemophilia A (1 unit/kg can add 2%, so 50 unit/kg add 100%)
  4. Factor IX concentrate: Hemophilia B
  5. 1-desamino-8-darginine vasopressin (DDAVP): Hemophilia A, vWD type I, II

➢ Antifibrinolytic therapy:
  1. E-aminocaproic acid (EACA, Plasllloid)
  2. Tranexamic acid (AMCA, Transamin)
Local hemostatic methods

- splints, pressure packs, sutures; gelfoam with thrombin, surgicel, oxycel, microfibrillar collagen(avitene), topical AHF
Heparin (anticoagulant)

- Complex inhibited (IXa, Xa, Xla, XIIa)
- Used in deep vein thrombosis, renal dialysis
- Rapid onset, Duration 4-6hrs (given IV)
- Monitoring by aPTT: 50-65 sec
- Discontinue 6 hrs before surgery then reinstituting therapy 6-12hrs post-op
- Protamine sulfate can reverse the effect
Coumarin (Vit k antagonist)

- Inhibit Vit K action (Factor II, VII, IX, X)
- Used venous thrombosis, cerebrovascular disease
- Duration half-life 40hrs
- Monitored by PT : INR 1.5-2.5
- PT > 2.5, reduction coumarin dosage (2-3 days)
- Vit. K can reverse the effect
Aspirin (antiplatelet)

➢ Inhibit cycloxygenase, TxA2 formation
➢ Analgesic drug impairs platelet function
➢ Aterial thrombosis, MI
➢ Tests-BT, aPTT
➢ If tests are abnormal, MD should be consulted before dental surgery is done
➢ Stop aspirin for 5 days, substitute alternative drug in consultation with MD
Hemophilia-dental management -1

➢ Preventive dentistry
   1. tooth brushing, flossing, rubber cup prophylaxis &
      topical fluoride, supragingival scaling
   2. without prior replacement therapy

➢ Pain control
   1. block anaesthesia: factor level > 50%
   2. Avoid aspirin, NSAIDs
Hemophilia-dental management -2

➢ Orthodontic treatment:
  1. no contraindication in well-motivated patients
  2. care with placement of bands and wires

➢ Operative dentistry
  1. rubber dam to protect tissue against accidental laceration
  2. wedges should be place to protect and retract papilla
Hemophilia-dental management - 3

➢ Pulp therapy
  1. Preferable to extraction
  2. Avoid overinstrumentation and overfilling

➢ Periodontal therapy
  1. no contraindication of probing and supragingival scaling
  2. deep scaling, curettage, surgery need replacement therapy
Hemophilia-dental management

➤ Oral surgery:

1. Dental extraction: 40%-50% level
2. Maxillofacial surgery (including surgery extraction of impaction teeth): 80-100%
3. Antifibrinolytic therapy & local hemostatic measure
4. do not open lingual tissue in lower molar regions to avoid hemorrhage track down a endanger airway
Summary

➢ History, PE, Lab data
➢ Consultation with physician
➢ Antibiotics to prevent post-op infection
➢ Avoid aspirin and NSAIDs
➢ Local hemostatic measure is very important
The End

• Thank you!

SLIDES AVAILABLE AT WITHIN 24 HRS: http://profiles.uonbi.ac.ke/andrew/

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