All that you didn’t know about platelets!

CME, Vadodara, Sunday, 27th December 2015

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Bombay Hospital Institute of Medical Sciences, Mumbai
Spurious thrombocytopenia
Spurious thrombocytopenia

• Harris (Bengal) syndrome

• EDTA induced platelet agglutination

• Partially clotted blood
## Thrombocytopenia: Different shades

<table>
<thead>
<tr>
<th>Severity</th>
<th>Platelet count (x $10^9$/L)</th>
<th>Prevalence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>150 – 400</td>
<td></td>
</tr>
<tr>
<td>Inconsequential</td>
<td>100 – 150</td>
<td>40</td>
</tr>
<tr>
<td>Mild</td>
<td>75 – 100</td>
<td>30</td>
</tr>
<tr>
<td>Moderate</td>
<td>30 – 75</td>
<td>20</td>
</tr>
<tr>
<td>Severe</td>
<td>&lt; 30</td>
<td>10</td>
</tr>
</tbody>
</table>

## Thrombocytopenia: Mechanism

<table>
<thead>
<tr>
<th>Production</th>
<th>Marrow disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Liver disorders</td>
</tr>
<tr>
<td>Destruction</td>
<td>Infections, sepsis, DIC</td>
</tr>
<tr>
<td></td>
<td>Drugs including heparin</td>
</tr>
<tr>
<td></td>
<td>Immune–mediated (ITP)</td>
</tr>
<tr>
<td>Sequestration</td>
<td>Hypersplenism</td>
</tr>
<tr>
<td>Dilution</td>
<td>Massive transfusion</td>
</tr>
<tr>
<td></td>
<td>Gestational thrombocytopenia</td>
</tr>
</tbody>
</table>
Infection induced thrombocytopenia
### Infection induced thrombocytopenia

<table>
<thead>
<tr>
<th></th>
<th>Dengue, Hanta</th>
<th>CMV, EBV, HCV</th>
<th>HIV – to remember</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Virus</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Bacteria</strong></td>
<td></td>
<td></td>
<td>Meningococcemia</td>
</tr>
<tr>
<td><strong>Parasites</strong></td>
<td></td>
<td></td>
<td>Malaria</td>
</tr>
</tbody>
</table>
Drug induced thrombocytopenia
## Drugs

<table>
<thead>
<tr>
<th>Production</th>
<th>Chemotherapy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Radiotherapy</td>
</tr>
<tr>
<td></td>
<td>Anti-folates</td>
</tr>
<tr>
<td></td>
<td>Others</td>
</tr>
<tr>
<td>Destruction</td>
<td>Heparin</td>
</tr>
<tr>
<td></td>
<td>Rifampicin</td>
</tr>
<tr>
<td></td>
<td>Quinine</td>
</tr>
<tr>
<td></td>
<td>Na–valproate</td>
</tr>
<tr>
<td></td>
<td>Others</td>
</tr>
</tbody>
</table>

Immune mediated thrombocytopenia
Pathogenesis of ITP

- Antibody mediated destruction
- T-lymphocyte mediated
- Inadequate production
What is new in the treatment of ITP?
H. Pylori
Anti CD20 Antibody

Rituximab
TPO analogs

- Eltrombopag (Revolade)
- Romiplostim (NPlate)
Thrombocytopenia with thrombosis
Q: One of the following disorders does not cause thrombosis & thrombocytopenia

A. Heparin induced thrombocytopenia
B. Antiphospholipid antibody syndrome
C. Evan’s syndrome
D. Thrombotic thrombocytopenic purpura
A: One of the following disorders does not cause thrombosis & thrombocytopenia

A. Heparin induced thrombocytopenia
B. Antiphospholipid antibody syndrome
C. Evan’s syndrome
D. Thrombotic thrombocytopenic purpura
TTP: Pathogenesis

Role of ADAMTS-13
TTP: Pathogenesis

Blood Flow

GPIIb

VWF

Vessel Wall

ADAMTS13

Normal Hemostasis

No ADAMTS13

TTP
Q: One of the following is the best treatment modality for TTP

A. FFP support
B. FFP & platelet support
C. Plasma exchange
D. Rituximab
A: One of the following is the best treatment modality for TTP

A. FFP support
B. FFP & platelet support
C. Plasma exchange
D. Rituximab
Plasma exchange for TTP

• It is an emergency
• Plasma exchange & not plasmapheresis
• In emergency : FFP
• Avoid platelet transfusion
• Cryosupernatant
• Follow up with LDH & platelet count

Contreras M et al. Transfusion. 2014;38:796–797
Investigations
Investigations

• History

• Examination

• CBC

• Blood film examination
Q: Give the diagnosis on PS in next slide

A. MAHA
B. Dengue
C. Malaria
D. CMV
Q: Give the diagnosis on PS in next slide

A. MAHA
B. Dengue
C. Malaria
D. CMV
A : The film shown to you is diagnostic of

A. MAHA
B. Dengue
C. Malaria
D. CMV
Blood film examination

• Platelets : Number, size, clumping
• RBC : Schistocytes, spherocytes, parasites
• WBC : Leukocytosis, leukaemia, dysplasia
Coagulation

• PT, PTT
• Fibrinogen
• D–Dimer
Blood chemistry

- Haemolysis: Bilirubin, LDH, Haptoglobin
- Hepatic dysfunction: Acute, chronic
- Renal dysfunction: HUS, TTP, DIC, SLE
USG abdomen for splenic enlargement and Doppler for portal hypertension
Bone marrow examination
Management
Management – 1

- Treating the cause
- Platelet transfusion
- IVGG, Anti D, steroids
- Antifibrinolytic agents
- NovoSeven

MCQ: Despite thrombocytopenia, platelet transfusion is most hazardous in

A. TTP & HIT-T

B. ITP & TTP

C. Hypersplenism & DIC

D. HLH & HUS
MCQ: Despite thrombocytopenia, platelet transfusion is most hazardous in

A. TTP & HIT–T

B. ITP & TTP

C. Hypersplenism & DIC

D. HLH & HUS
The peril of platelet transfusion

<table>
<thead>
<tr>
<th>Contraindicated</th>
<th>TTP, HIT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not indicated</td>
<td>ITP</td>
</tr>
<tr>
<td></td>
<td>Hypersplenism</td>
</tr>
<tr>
<td>Indicated</td>
<td>Production defects</td>
</tr>
<tr>
<td></td>
<td>Others: DIC, dilutional</td>
</tr>
</tbody>
</table>

Platelet count as the trigger for transfusion
(Clinical judgment is superior to the count)

• Severity
• Associated coagulation defects
• Stability
• Aetiology
