Investigation into the outcome of a liver transplant patient with Anti-Yta

Hamed Beig Mohammadi
Uyen Walker
Royal Prince Alfred Hospital
Blood bank
Patient History

Mr A.S, a 63 year-old Lebanese male presents with a Hb 86, which was rapidly falling.

*Clinical History*: Alcoholic liver disease, Hepatocellular carcinoma, Refractory ascites, hepatic encephalopathy, anxiety/depression (on liver transplant waiting list)
Initial Blood Bank work up

- **Blood Group**: A pos
- **Primary Antibody screens**: Negative (21/11/2013)
- **RBC phenotype**: R1r, K neg, Fy(a) neg
- **Direct Antiglobulin test**: Negative

**Transfusion history**: Nil

Patient treated with **1 pooled platelets and 3 RBCs**

(Rh matched/ K neg).
Liver Transplant activation

A month later! On 23/12/2013

SCI 3+
SCII 3+
SCIII 3+

3 Cells Screen = SCI 3+
SCII 3+
SCIII 3+

11 Cells Panel = 3+ (All cells) (Auto = Negative)

Enzyme panel = Negative

* Initial Transplant call aborted
Red Cell Reference Lab Results

- Antibody: Anti-Yt(a) (Cartwright)
  Anti-K not excluded.

- Red Cell phenotype: Yt(a) negative
Liver transplant

28/12/2013

Blood loss 2000ml

5 X RBC
3 X Fresh Frozen Plasma
1 X Pooled Platelet
20 X Cryoprecipitate
3.5 Litres 4% Albumin

Recombinant Factor VIIa

• 07/01/2014 and 08/01/2014

Post transplant

2 X RBC
Laboratory findings post-transplant

Direct Antiglobulin Test

<table>
<thead>
<tr>
<th>Date</th>
<th>30/12/2013 16:28:46</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cards</strong></td>
<td></td>
</tr>
<tr>
<td>Card ID</td>
<td>715506140060105541</td>
</tr>
<tr>
<td><strong>Well Reaction</strong></td>
<td></td>
</tr>
<tr>
<td>AHG</td>
<td>2+</td>
</tr>
<tr>
<td>IgG</td>
<td>2+</td>
</tr>
<tr>
<td>C3d</td>
<td>-</td>
</tr>
<tr>
<td>ctl</td>
<td>-</td>
</tr>
</tbody>
</table>

*IgG: positive C3d: negative*
Haemoglobin monitoring

1st presentation, given Yta Pos RBCs

Transplant, intra-op massive transfusion

Post transplant, additional 2x RBC in ICU

Pre-Tx
Hb 88
Plts 67
INR 2.3
Titration monitoring using pooled screening cells.
Transfusion support for liver transplant patients

“We would like 14 units of APos, K neg, Yta neg please”

Livers are nutritious!
Transfusion support for liver transplant patients

- Match for Rh and Kell units prior to transplant.
- Potential massive transfusion due to existing coagulopathy, portal hypertension, thrombosis, splenomegaly, prior abdominal surgery
- “Functional” recovery of new liver
- Family members typed for Yta, all were incompatible
Implications of incompatible blood during massive transfusion

Study at Jackson Memorial Hospital USA 2007¹, reviewed 2000 patients undergoing liver, intestinal and multivisceral transplants, in a period over 15 years.

- 115 had clinically significant antibodies, 46 had multiple antibodies.
- A mean of 18 packed RBC units (U) were transfused per patient.
Implications of incompatible blood during massive transfusion

• Transfusion strategy:
  First 5-10U: Antigen negative
  Massive Blood loss: Antigen unscreened
  Last 5-10U: Antigen negative

• All patients were successfully managed without delay in initiation of surgery or haemolytic complications during transplant or thereafter.
Characteristics of Cartwright system antigens and antibodies

<table>
<thead>
<tr>
<th>Antigens</th>
<th>Antibodies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yta is of high frequency (99.7%)</td>
<td>High titre, low avidity</td>
</tr>
<tr>
<td>located on red cell acetylcholinesterase (ACHE locus on chromosome 7q).</td>
<td>Can demonstrate variable reaction strength</td>
</tr>
<tr>
<td>Weaker expression of Yta on cord blood cells, Ytb has similar expression as adult cells.</td>
<td>IgG reacting by IAT.</td>
</tr>
<tr>
<td>Sensitive (variable) to ficin/papain, DTT, AET</td>
<td>Some Anti-Yta bind complement</td>
</tr>
<tr>
<td>Resistant to trypsin</td>
<td>Does not generally cause transfusion reactions</td>
</tr>
<tr>
<td></td>
<td>Does not cause HDN</td>
</tr>
<tr>
<td></td>
<td>Commonly found in individuals of Jewish descent.</td>
</tr>
</tbody>
</table>
Clinical significance of Anti-Yta

<table>
<thead>
<tr>
<th>Study</th>
<th>No of patients in study</th>
<th>Cr-labelled survival studies</th>
<th>MMA</th>
<th>Incompatible units transfused</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transfusion. 1977 Jan-Feb;17(1):65-6.</td>
<td>1</td>
<td>Rapid destruction (T1/2 in 6 hours)</td>
<td>n/a</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Transfusion. 1981 Nov-Dec;21(6):702-5.</td>
<td>1</td>
<td>Moderate destruction (T1/2 in 96 hours)</td>
<td>n/a</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Transfusion. 1985 Jul-Aug;25(4):381-4.</td>
<td>5</td>
<td>1 increased destruction, 3 normal survival 1 not performed</td>
<td>No activation</td>
<td></td>
<td>No evidence of acute or delayed HTR.</td>
</tr>
<tr>
<td>Sangre (Barc). 1993 Aug;38(4):327-9.</td>
<td>1</td>
<td>Normal survival</td>
<td>n/a</td>
<td>2</td>
<td>No adverse reaction</td>
</tr>
</tbody>
</table>
Conclusion

• Successful transplant for patient A.S without haemolytic complications observed
• In emergency or urgent transfusion, most patients receiving Yt-incompatible blood have fared well.
• Despite haemodilution, Anti-Yta was still present, with a mild decline in antibody titre.
• Clinical significance should be interpreted on a case-by-case basis.
Acknowledgements

• Mark Langshaw, Senior Hospital Scientist, Blood Bank Laboratory, Royal Prince Alfred Hospital.
• Bernadette Blayney, Senior Scientist, Blood Bank Laboratory, Royal Prince Alfred Hospital.
• Dr Lianne Khoo, Haematologist, Royal Prince Alfred Hospital.
• Ming Tian, Red Cell Reference Laboratory, ARCBS, NSW
References


