QUESTION 18 Anemia post op
A 67-year-old woman develops symptomatic anaemia and jaundice nine days after subtotal colectomy for adenocarcinoma of the colon, at the time of which she received a transfusion of 3 units of red cells. She is on therapy (which includes sulfasalazine) for ulcerative colitis.
Review of the pre-operative antibody screen shows this to be negative for red cell alloantibodies.
Her current full blood examination includes the following:
haemoglobin 82 g/L [120-155]
mean cell volume 99 fl [80-97]
reticulocyte count 162 x 10^9/L [8-104]
Her blood film is shown below.

The most likely cause of her anaemia is:
A. iron deficiency.
B. peri-operative blood loss.
C. red cell aplasia.
D. delayed haemolytic transfusion reaction.
E. megaloblastosis.

Delayed hemolytic transfusion reaction (DHTRs)
- Due to anamnestic antibody response occurring after reexposure to a foreign red cell antigen previously encountered by transfusion, transplantation or pregnancy
- Antibody is often Kidd or Rh system – undetectable on pretransfusion testing but increases rapidly in titer following transfusion
- Seen within 2-10 days after transfusion
- Hemolysis is usually extravascular, gradual and less severe than with acute reactions

Signs and symptoms
- Slight fever
- Falling hematocrit
- Mild increase in serum unconjugated bilirubin
- Spherocytosis on blood smear

- New positive direct antiglobulin test a
- New positive antibody screen

**Treatment**
- No treatment is required in absence of brisk hemolysis
- Future transfusions containing the implicated red cell antigen need to be avoided

**Acute Hemolytic reactions**
- Due to ABO incompatibility
- Rapid destruction of donor erythrocytes by preformed recipient antibodies
- May lead to DIC, shock, ARF due to acute tubular necrosis

**Clinical presentation**
- Classic presenting triad of fever, flank pain, and red or brown urine – rarely seen
- Fever and chills – only manifestation
- Direct coombs test positive
**Initial approach**
1. As fever is also a presenting sign of a “benign” FNHTR, one must rule out a haemolytic transfusion reaction

2. Stop transfusion
   - Bag containing transfused cell should have repeat typing and cross matching of this unit by blood bank

3. Maintain patient’s airway, blood pressure and heart rate

4. Begin infusion of N/Saline immediately to initiate diuresis and avoid hypotension
   - Avoid Ringer’s lactate solution due to calcium – initiate clotting
   - Avoid dextrose – may hemolyze an of remaining red cells in line

5. Obtain sample of a direct antiglobulin test, plasma free hemoglobin and repeat type and cross match
   - Save urine sample for hemoglobin testing

6. Blood bank should be alert immediately

**Treatment**
1. Start fluids – N/Saline
2. Maintain urine output above 100-200ml/hr
3. Cardiac monitoring if massive hemolysis
4. Vigorous supportive care

**Febrile Non haemolytic reactions**
- Most common transfusion reaction
- Fever, often chill and sometimes mild dyspnea
- Benign and causing no lasting sequelae
- Caused by cytokines such as interleukinIL 1, 6 and 8 and TNFa generated during storage of blood components
- Only 15% of pt who have an FNHTR will have 2nd reaction with further transfusions
- Associated with class I HLA Ab

**Management**
- Stop transfusion
- Determine that a haemolytic reaction is not taking place
- Administration of antipyretics
- Asprin should be avoided in thrombocytopenic patients
- Prestorage leukoreduction should be performed on all cellular components intended for transfusion if cost were not a factor

**Megaloblastosis**
From lecture by Carole Smith on Megloblastic anemia

**Causes:**
1. Vit B12 deficiency
   a) Malabsorption
      - Pernicious anemia
      - Total or partial gastrectomy
      - Stagnant loop syndrome
      - Crohn’s disease or ileal reseetion
      - Chronic atrophic gastritis
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b) Reduced Intake
   - Veganism (Vit B12 from meat)
   c) Impaired utilisation /inactivation – N2O abuse/overuse

2. Folic acid deficiency
3. Abnormalities of B12 or folate metabolism
4. Other defects of DNA synthesis

**Clinical features of megaloblastic anemia**

- Glossitis
- Mild jaundice – lemon tinted skin, sclera
- Neuropathy and madness
- LOW (megaloblastic gut)

**Ix**

- Very high MCV 105-150
- Blood film: hypersegmented neutrophil - 6 lobed nucleus

**Red cell aplasia**

Rare condition of profound anemia characterized by the absence of reticulocytes and virtual absence of erythroid precursors in the bone marrow

**Etiology**

Often idiopathic (4 – 20%)
Represents the prodrome to a myelodysplastic syndrome

Causes of acquired pure red cell aplasia

Idiopathic

Drugs (phenytoin, trimethoprim-sulfamethoxazole, zidovudine, chlorpropamide, recombinant human erythropoietins, mycophenolate)

Infection

Immune disorders

Lymphoid malignancies

Myeloid malignancies

Other cancers

Pregnancy

Site of suppression

At the stage between CFU (Colony Forming Unit erythroid) and proerythroblast

Lymphopoiesis, granulopoiesis and megakaryopoiesis are all normal

Clinical manifestations

Very low reticulocyte count

Normal white blood cell and platelet count

A bone marrow that shows normal myelopoiesis, lymphopoiesis and megakaryopoiesis but few if any erythroid precursors

**Back to the question**

A. iron deficiency.
   - this will show a microcytic anemia

B. peri-operative blood loss.
   - will show a normocytic anemia
   - patient was given a blood transfusion perioperatively
   - unlikely to be the cause 9 days post op

C. red cell aplasia.
   - reticulocyte count was high and therefore not red cell aplasia

D. delayed haemolytic transfusion reaction.
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- spherocytosis
E. megaloblastosis.
- no megalocytes (hypersegmented nucleus)