ISSUE
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QSP Monthly

HORIBA Medical

NEWSLETTER OF THE QUALITY SLIDE PROGRAM

This issue

October Slides P.1
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October Slides

Slide 1
CLL.

Slide 2
Nothing to report.

Slide 3
Nothing to report.

Slide 4
See case study on the right.

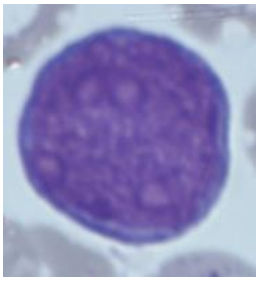
Slide 5
Leukemic phase of typical Mantle leukaemia.

Slide 6
Post-therapy follow-up of AML7.

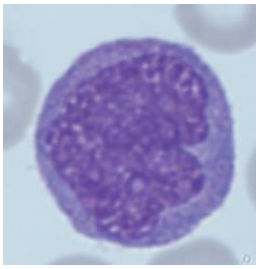
Monthly Digital Case Study
Presentation October 2022, Slide 4

FBC Results

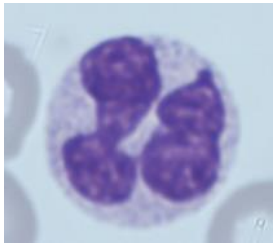
WBC 21.2* (10^3/mm3)
RBC 2.51* (10^6/mm3)
HGB 7.4* (g/dL)
HCT 22.7* (%)
MCV 90 (fL)
MCH 29.5 (pg)
MCHC 32.6 (g/dL)
PLT 104* (10^3/mm3)



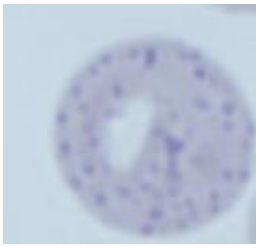
Blast Cell



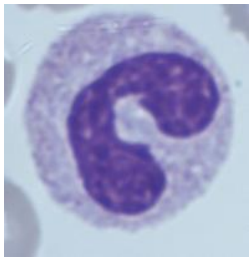
Blast Cell



Hypo granular Neutrophil



Basophilic Stippling



Hypo Segmented (left Shift) Neutrophil

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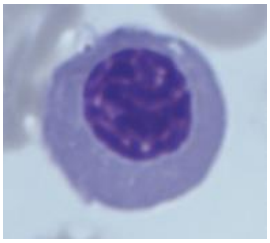
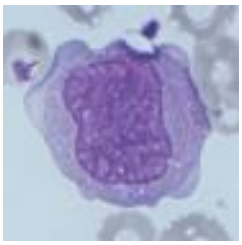
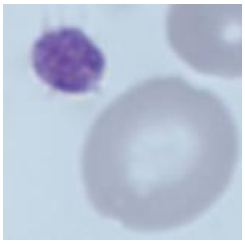


Promonocyte



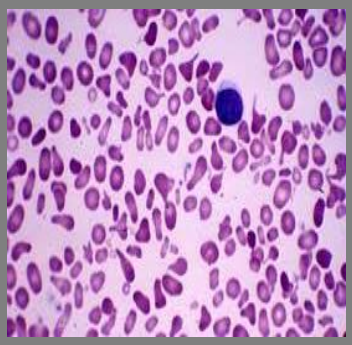
Nucleated Red Blood Cell



Macrothrombocyte

Monthly Morphology Quiz

Look closely at the slide below:



Which of the below would you associate with a film appearance like this?

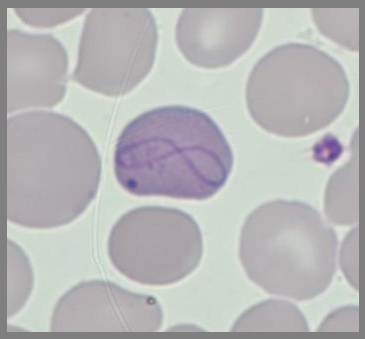
A) Sickle cell

B) Hereditary Elliptocytosis

C) Haemolysis

Last month's cell Quiz:

Look closely at this red cell. What is unusual about it and what could this indicate?



Answer:
Blood film shows a red cell with a cabot ring and a Howell-Jolly body. In this case, the patient was displaying a myelodysplastic picture with abnormalities in all cell lines and circulating megakaryocytes.

Slide review (continued)

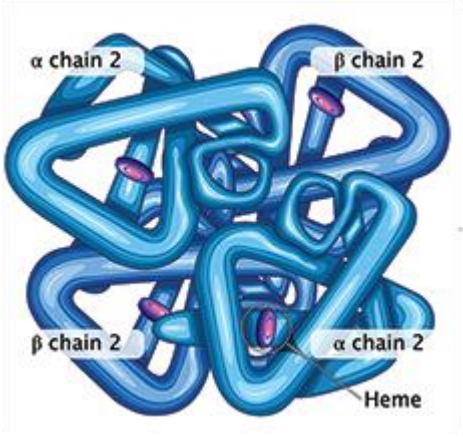
04 October 2022
Male (78years old).
Clinical Haematology Unit. Hyposegmented (++) and hypogranulated (+++) neutrophils. Monocytes/Promonocytes/Monoblasts (+++). Anaemia, Anisocytosis (++). Thrombocytopenia. AML post MDS?

Expert Comment: Pelger Neutrophils (+++), Macroplatelets.
Probable AML post MDS or AML probably with multilineage dysplasia.

Haemoglobin Structure and Function

The primary function of Haemoglobin (Hgb) is to transport oxygen from the lungs to the tissues. The haemoglobin molecule consists of four separate folded polypeptide chains. In normal adult haemoglobin the chains are named alpha and beta chains (known as HbA), which form a hydrophobic water repelling pocket around a heme group. The heme group is composed of a central Iron atom complexed to 4 Nitrogen atoms. Oxygen is capable of reversibly binding to the heme group. The Haemoglobin molecule can exist in 2 states – a tense (T) state which has low affinity for Oxygen and a Relaxed (R) state which has a high affinity for oxygen. This transition from one state to another enables Oxygen to be taken up in the presence of high oxygen concentration (lungs) and release oxygen in low oxygen concentration. Changes to the structure of the polypeptide chains of the haemoglobin molecule has a profound effect on the function of the haemoglobin.

The Haemoglobin molecule

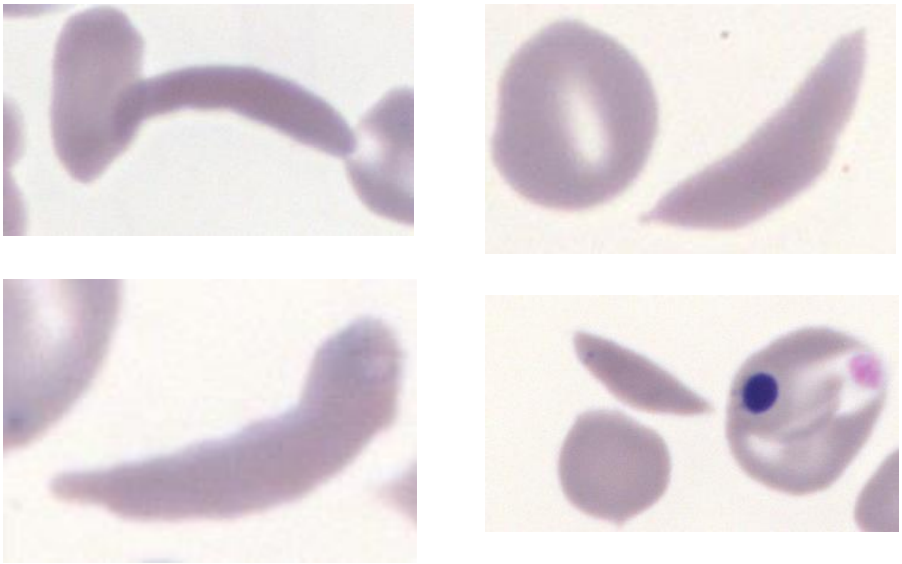


Sickle Haemoglobin HbS

Inheritance of an abnormal sickle cell gene causes the production of HbS. The mutation is on the HBB gene and causes a single amino acid substitution in the Beta chain, specifically replacing Glutamic Acid with Valine at position 6 of the Beta chain (Glu6Val or E6V). The inheritance of the abnormal gene is autosomal recessive, therefore it is possible to have either heterozygous beta chains with the Haemoglobin being made up of 50% HbA and HbS (Sickle cell trait) or homozygous where haemoglobin is entirely HbS (sickle cell anaemia).

In the presence of a low oxygen concentration, the replacement of Glutamic Acid with Valine causes the abnormal HbS sub-units to stick together and form long, rigid molecules that bends the red cell into a sickle like shape. Sickled red cells are destroyed prematurely, causing anaemia which may require blood transfusion. Red Cells containing HbS have increased adhesion which can cause the formation of heterocellular aggregates, which physically causes small vessel occlusion and resultant local hypoxia.

Sickle Cells



The chance of inheriting either sickle cell trait or sickle cell anaemia is dependent upon the genetic makeup of the parents. If both parents have sickle cell trait, then there is a 1 in 4 chance of offspring having homozygous sickle cell anaemia a 1 in 2 chance of having sickle cell trait and a 1 in 4 chance of having normal haemoglobin.

Sickle cell anaemia is most common in Africa, the Middle East, and India. People with sickle cell trait do have a relative resistance to Plasmodium Falciparum and are less likely to get the disease, have a lower parasite count, and are less likely to die.

Patients with sickle cell trait do not normally have any symptoms unless the body is under immense stress e.g. intense exercise or low oxygen environments.

Patients with Sickle Cell Anaemia start presenting with symptoms after about age 6 months and can present with many different symptoms including Vaso-Occlusive crisis, Acute Chest Syndrome, infections, stroke, PE, renal complication, and aplasia.

Patients often have sickle cell crisis where there is acute pain caused by vaso occlusions. A sudden drop in the already low Hb level may occur due to either splenic sequestration, aplastic crisis (often in conjunction with parvovirus B19 infection or other vital infections), or hyper haemolytic crisis (particularly if the patient is G6PD deficient).

Bibliography

Hemoglobin: Structure, Function and Allostery - PMC (nih.gov)

HBB gene: MedlinePlus Genetics

Sickle Cell Disease - StatPearls - NCBI Bookshelf (nih.gov)

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