ISSUE
37
JUNE
2023

QSP Monthly

NEWSLETTER OF THE QUALITY SLIDE PROGRAM

HORIBA Medical

This issue

June Slides

Slide 1
Nothing to report.

Slide 2
Hyperlymphocytosis associated with numerous smudge which may be compatible with lymphoproliferative syndrome or non-Hodgkin's lymphoma. Request immunophenotyping of circulating lymphocytes with establishment of a Matutes scoring and a cytogenetic assessment. Note: Presence of incised nuclei on numerous images of lymphocytes.

Slide 3
Microcytes (+). Anisocytosis (+). Schizocytes (+). Echinocytes (+). Hypochromia (++). Expert's comments: suspected iron deficiency.

Slide 4
Macrocytic anisocytosis (+). Spherocytes (+). Discreet myelemia. Expert's comments: Exclude a chronic monocytic myeloid leukaemia.

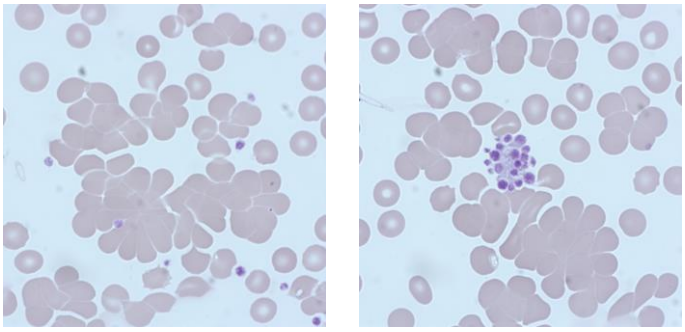
Slide 5
See monthly slide review on right.

Slide 6
Anaemia. Anisocytosis (+++). Microcytes (+++). Macrocyte (+++). Spherocytes (++). Polychromatophilic (++) RBCs. Presence of punctuated RBCs and some acidophilic erythroblasts. Reticulocytosis (+++): 18% Reticulocytes (RET#: 306x10^9/L). Expert's comment: Autoimmune haemolytic anaemia? (Presence of spherocytes)

Monthly Digital Case Study Presentation
June 2023, Slide 5

FBC Results

WBC	6.35	(10^3/mm3)
RBC	3.41*	(10^6/mm3)
HGB	14.2*	(g/dL)
HCT	30.7*	(%)
MCV	90	(fL)
MCH	41.6	(pg)
MCHC	46.3	(g/dL)
PLT	363	(10^3/mm3)
Neutrophils	53%	
Lymphocytes	19.9%	
Monocytes	21.3%	
Eosinophils	5.1%	
Basophils	0%	



Red blood cells agglutination consistent with the clinical and biological context.

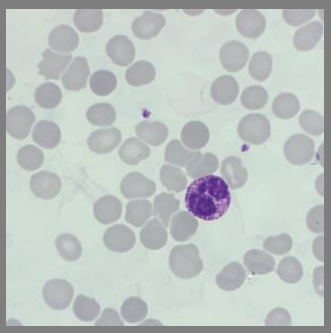
Clinical Details
05 June 2023 Male (64 years old)

Slide Information
Red blood cell flags. Agglutination of red blood cells (+++) due to the presence of a cold agglutinin verified after incubation of the sample at 37°C and rerun of this sample to check the results associated with the parameters which were alarmed.

Expert Comment
Look for a cold agglutinin. Cold agglutinin disease/ lymphoproliferative syndrome?

QSP Quality Slide Program 2.0

Cell Quiz



Name the white cell in the slide image above.

A. Blast cell

B. Eosinophil

C. Basophil

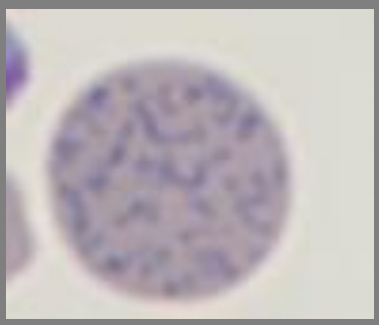
Last Month's Quiz

What feature can be seen in the red cell?

A. Basophilic Stippling

B. Bitten Out Cell

C. Cabot's Ring



Right answer:
Basophilic Stippling or punctate basophilia describes the presence of numerous small basophilic inclusions in the cytoplasm of the red cell. They are composed of aggregates of ribosomes, degenerating mitochondria and siderosomes. Basophilic Stippling is associated with conditions such as thalassaemia, haemolytic anaemia, myelodysplastic syndromes, sickle cell anaemia, heavy metal poisoning (lead, zinc, arsenic, etc.)

Haemoglobinopathies – Part 2

The most significant haemoglobinopathies result in either a change in the structure and quality of the haemoglobin, or a reduction in the quantity of haemoglobin produced.

Haemoglobinopathies are:
• not gender (x) linked
• more prevalent in some parts of the world

The likelihood of a person being a carrier of a haemoglobinopathy depends on ancestry. The type of mutation varies between ethnic groups. It is possible to inherit mutations in both alpha and beta globin genes at the same time. It is also possible (although rare) for an individual to have a '**de novo**' haemoglobin mutation. This is a genetic mutation that is not directly inherited from parents but is present only in that individual.

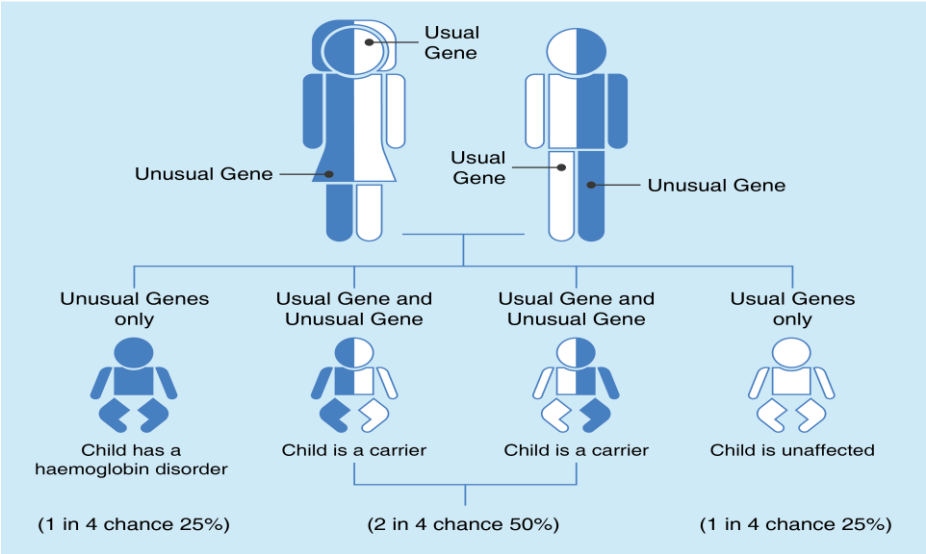
Inheritance

The genes to produce haemoglobin are inherited from both parents. Sickle cell disease or beta thalassaemia major are recessively inherited. If one unusual beta chain gene is inherited from one parent, the individual will be a carrier of the condition but will not be affected. This is known as having a **trait**. Carriers of haemoglobin variants are healthy and are unaware of their status unless detected during screening.

Should two unusual beta chain genes are inherited (one from each parent), the individual will have a haemoglobin disorder. The most common clinically significant conditions are beta thalassaemia major and sickle cell disease.

If both parents carry a significant haemoglobinopathy, the baby is at risk of inheriting a haemoglobin disorder. There is a:

• 1 in 4 (25%) chance of being completely unaffected
• 2 in 4 (50%) chance of being a carrier
• 1 in 4 (25%) chance of inheriting the condition:



Sickle Disease

Sickle carrier

If a person inherited one normal haemoglobin A gene and one sickle haemoglobin gene, they are a sickle carrier. With one typical haemoglobin gene and one altered form of the gene, people with the sickle cell trait make both typical haemoglobin and sickle cell haemoglobin.

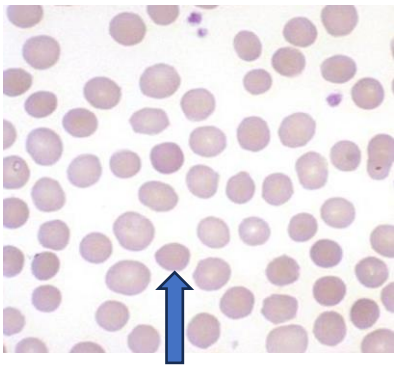
Their blood might contain some sickle cells, but they generally don't have symptoms. They're carriers of the disease, however, which means they would pass the gene to their children.

Sickle cell disease

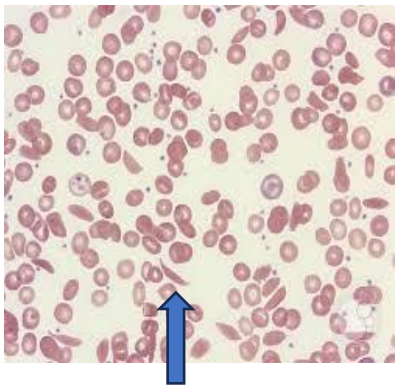
Sickle cell disease occurs when a person inherits two abnormal copies of the β-globin gene (HBB) that makes haemoglobin, one from each parent. This gene occurs in chromosome 11. Several subtypes exist, depending on the exact mutation in each haemoglobin gene.

Sickle cell disease most commonly affects people of West African, Caribbean, Middle Eastern and Indian ancestry. However, it can affect anyone from any population.

Sickle rbc's have a shorter than average life span (10-20 days), and this premature destruction results in anaemia. Unlike normal rbc's which are flexible, sickle cells are rigid and also sticky. This can cause clumping in vessel walls, resulting in obstruction and a reduced oxygen supply to various organs. This occurs frequently and manifests itself as periodic episodes of pain, known as "crisis". These Vaso-occlusive events can result in damage to eyes, kidneys, and lungs.



Normal red blood cells



Sickle red blood cells

As the spleen is managing increased numbers of dead rbc's, it becomes fibrous and enlarged. The immune function in the spleen declines, which results in the individual being more susceptible to infection. The hyposplenism would be indicated by the presence of Howell-Jolly bodies and target cells. In an attempt to compensate for the rbc loss, the bone marrow attempts to produce more cells and grow larger. This results in weakened bones. Patients also present with jaundice due to the rapid destruction of haem in the rbc's.

Treatment

- Bone marrow transplant is currently the only known cure (replacing diseased stem cells with healthy donor cells.
- Prophylactic antibiotics treatment to avoid crisis and relieve symptoms
- Drugs to promote formation of foetal haemoglobin to suppress sickling
- Periodic blood transfusions to prevent anaemia and prevent crisis

QSP 2.0

Available as a single use license and a site license which allows up to 50 concurrent users.
To find out more, contact us.

Bibliography

https://medlineplus.gov/lab-tests/hemoglobin-electrophoresis/

Essential Haematology
A.V .Hoffbrand &
J.E.Pettit

https://www.mayoclinic.org/

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1390791/

Editorial Team

Kelly Duffy
Andrew Fisher

About us

HORIBA UK Limited
Kyoto Close
Moulton Park
Northampton, UK
NN3 6FL

HORIBA Medical
Parc Euromédecine, 390
Rue du Caducée, 34790,
France

www.horiba.com/medical

QSP Quality Slide Program 2.0