

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

NAME : NAJMA ABDULLA KHALEEF

AGE : 40 YEARS OLD

ADRESS : MOSUL; SOQ AL-MAASH

OCCUPATION : HOUSE WIFE

Chief Complaint

pallor and easy fatigability for 2months.

History of Present illness

40 years old female patient presented with pallor associated with easy fatigability, poor appetite, nausea, vomiting, one month ago she was admitted to the hospital and diagnosed as anemia and received one pint of blood then discharged, one month later the previous symptoms returned with fever, night sweating, lethargy and weight loss

REVIEW OF OTHER SYSTEMS

- **C.N.S** : No history of epilepsy or any neurological deficit
- **RYSP.** : cough .with sputum
- **G.U.T**: no frequency ; no dysuria.



- **PAST MEDICAL HISTORY**

History of old T.B before 20 years

- **PAST SURGICAL HISTORY**

History of ectopic pregnancy & hysterectomy before 10 years

- **FAMILY HISTORY**

No family history of similar symptoms.

- **DRUG HISTORY**

No drug allergy.

On examination

- Middle age female
- conscious
- looked pale
- not dyspneic,
- not jaundiced,
- no lymphadenopathy
- no bruises on her skin.
- No gum bleeding
- No gum hypertrophy
- Chest: HVB
- Heart : normal double rhythm.
- Abdomen : soft abdomen ,no palpable liver or spleen
- Temperature was 37.3c⁰.
- pulse rate :100 Bpm.
- BP 120/70 mmHg

INVESTIGATION

- **ULTRA SOUND** OF Abdomen:
- Normal liver spleen and pancreas size and echogenecity No focal lesion
- Normal GB &biliary tree
- Normal both kidneys size & echogenecity
- RT kidney show mild dilatation of calyces seen
- No para aortic lymph gland enlargement seen
- No pelvic pathology detected.

BIOCHEMICAL

- RBS : 7.6 mmol/l NR up to 7.8
- TOTAL BILIRUBIN : 11 micromol/l NR up to 17
 - DIRECT: 5 micromol/l NR up to 4.2
 - INDIRECT: 6 micromol/l NR up to 6
- SGPT: 22 U/L NR up to 55
- SGOT: 38 U/L NR up to 39

BIOCHEMICAL *cont.*

• S.ALK:	204U/L	NR(98-279)
• S.LDH:	405 U/L	NR(240-480)
• S.ALB:	36g/l	NR(36-52)
• S.uric acid:	117 micromol/l	NR(155-357)
• Blood urea:	5.4 mmol/l	NR (2.5-7.5)
• S.creatinine:	72 micromol/l	NR (62-124)
• S.k :	3.4 mmol/l	NR(3.5-5.3)
• S.Ca:	1.9 mmol/l	NR(2.1-2.6)

VIRAL SCREENING

- HBV: **NEGATIVE**
- HCV: **NEGATIVE**
- HIV: **NEGATIVE**

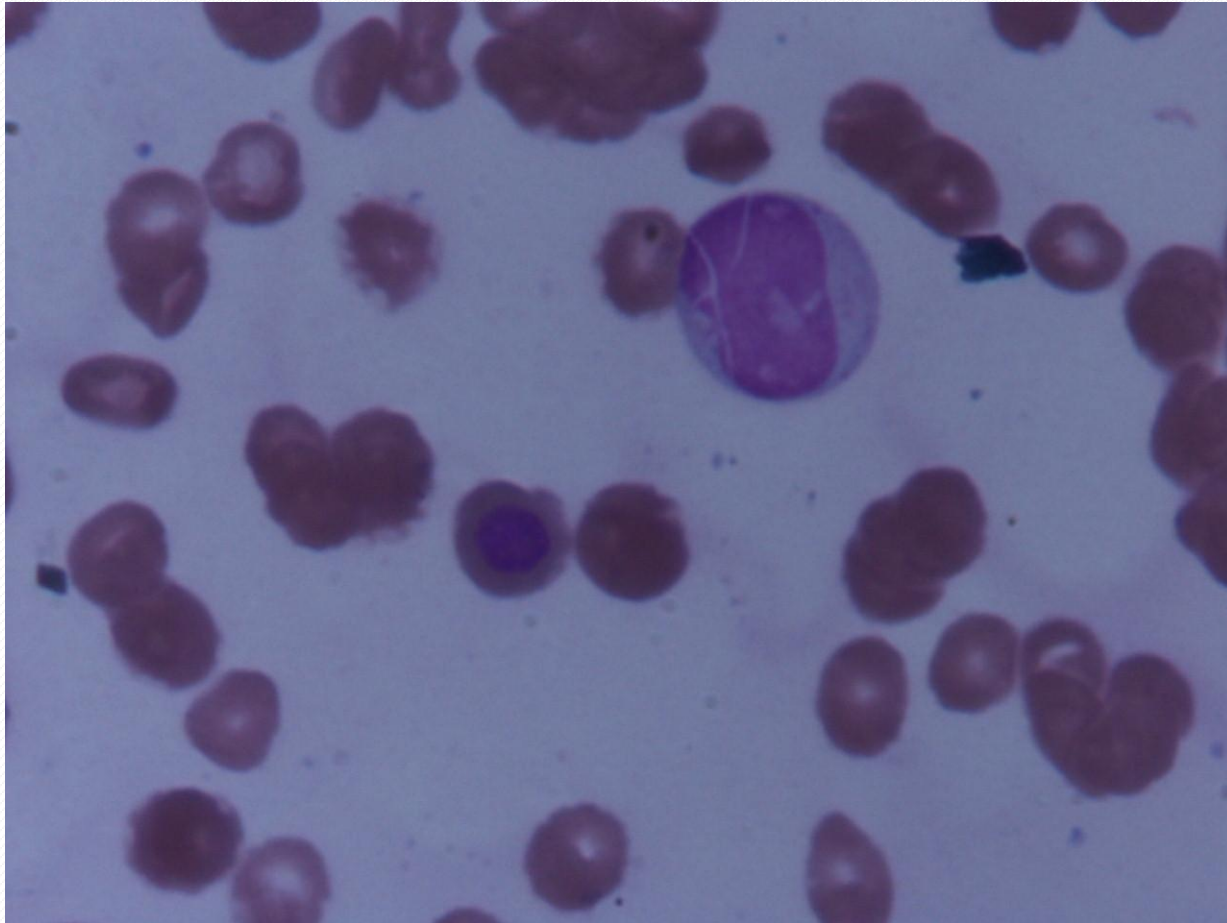
CBP: at 1/10/2013

Hb:	63 g/L	NR(120-150)g/L
PCV:	0.18 L/L	NR(0.36-0.46)L/L
MCV :	94.9 fl	NR(83-101)fl
MCH:	32.3pg	NR(27-32)pg
MCHC:	34.1%	NR(30-36)%
WBC:	5.3x10 ⁹ /L	NR(4-11X10 ⁹ /L)
Blasts:	56%	
Neutrophils	13%	
Lymphocytes	27%	
Monocytes	4%	
Platelets	20X10 ⁹ /L	NR(150-410X10 ⁹ /L)
ESR	149 mm/hr	

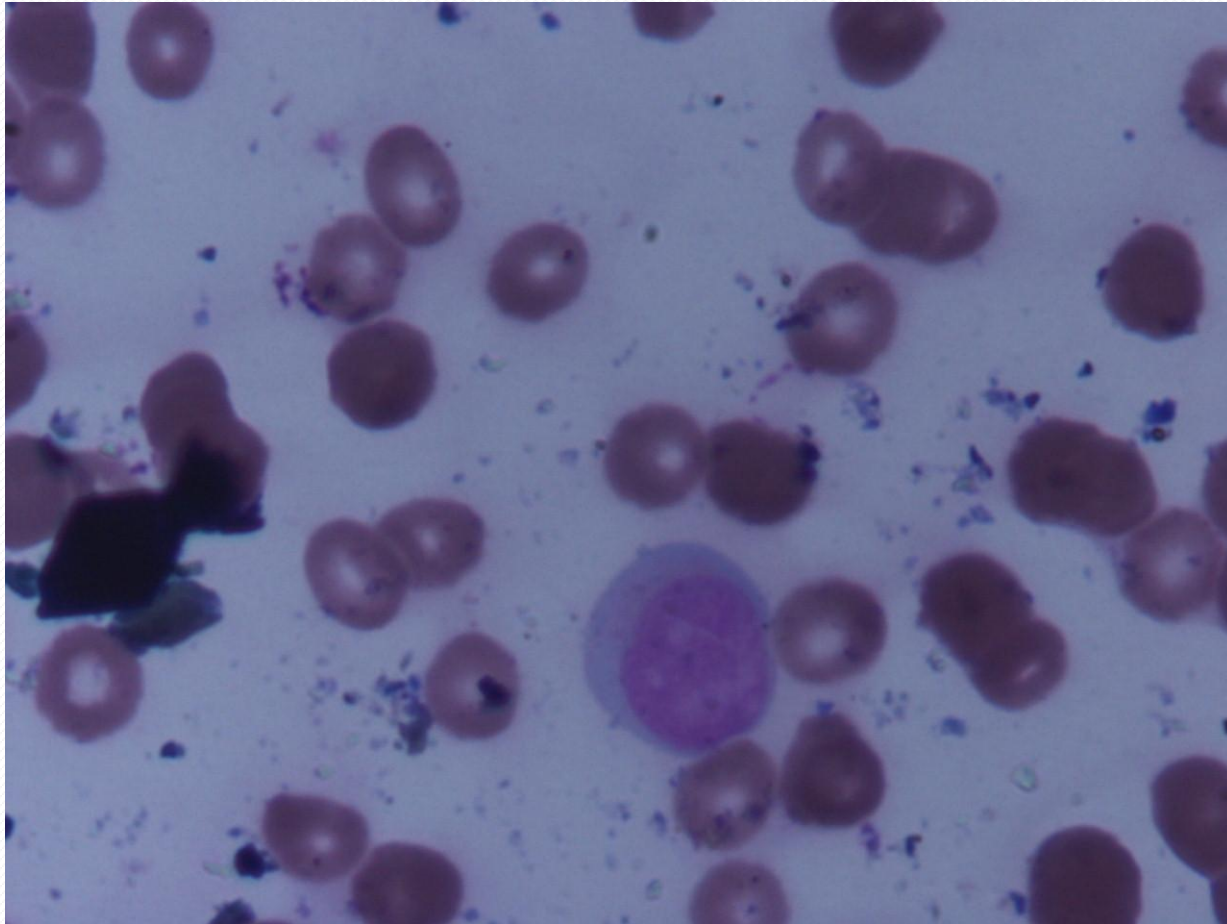
BLOOD FILM

- RBC :Normochromic
Normocytic , few normoblast
- WBC : blastaemia
- Platelets : severely reduced in film

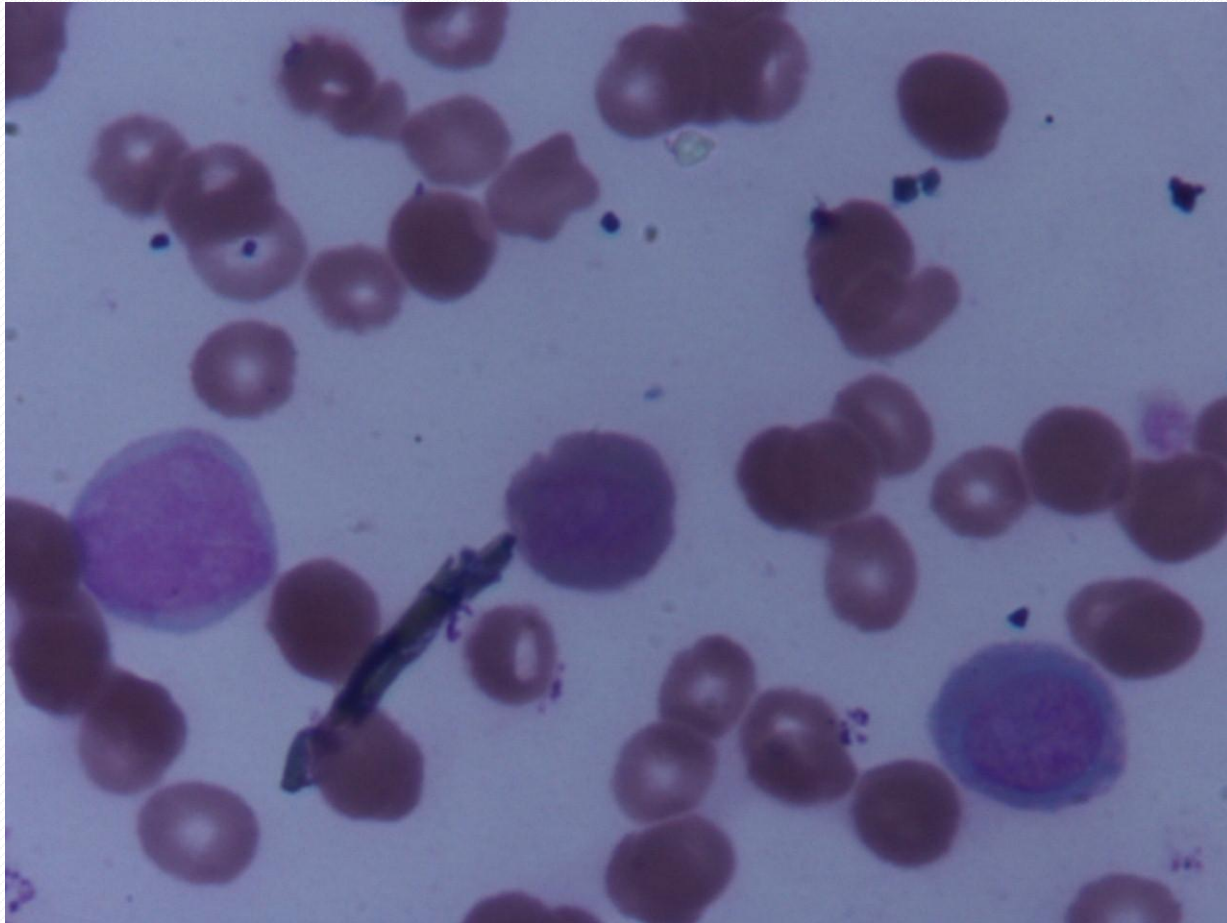
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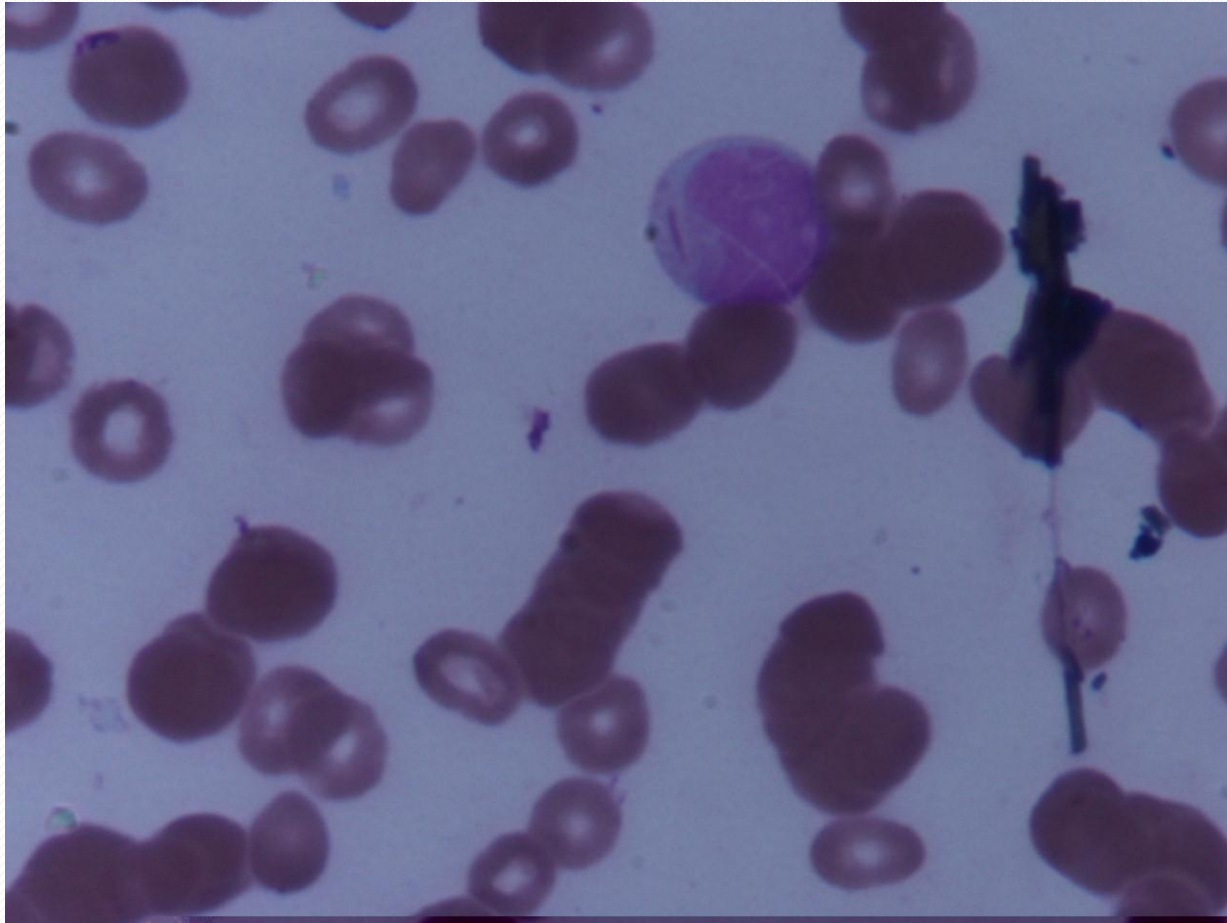
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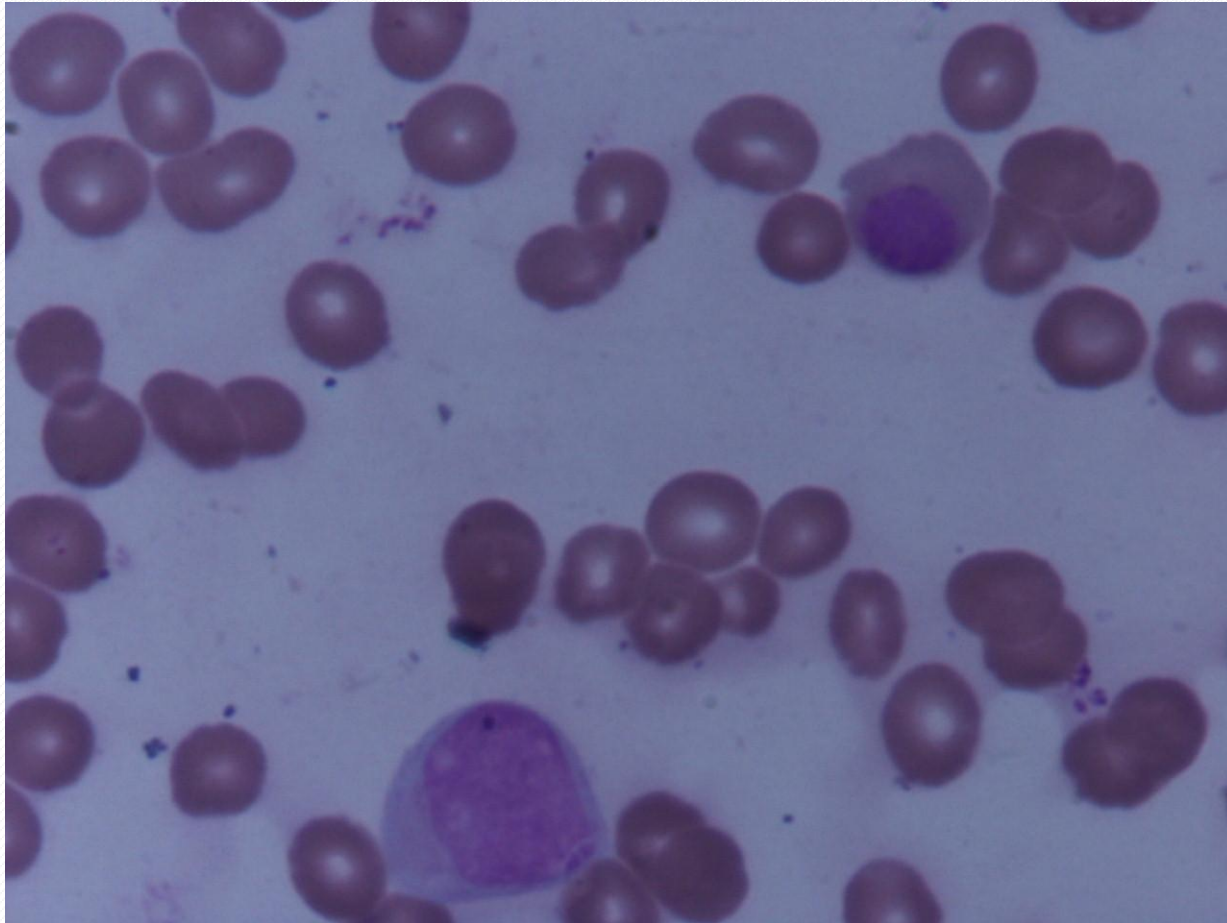
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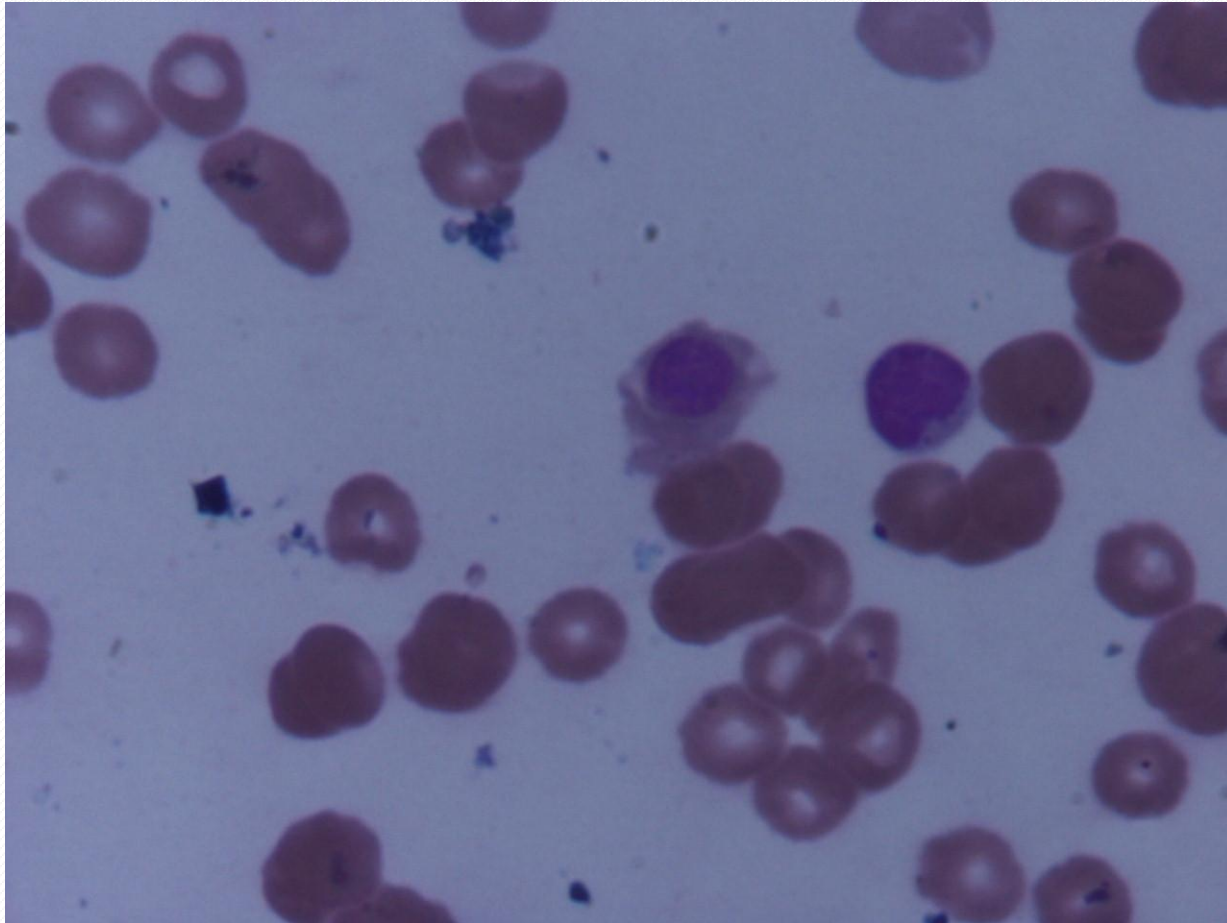
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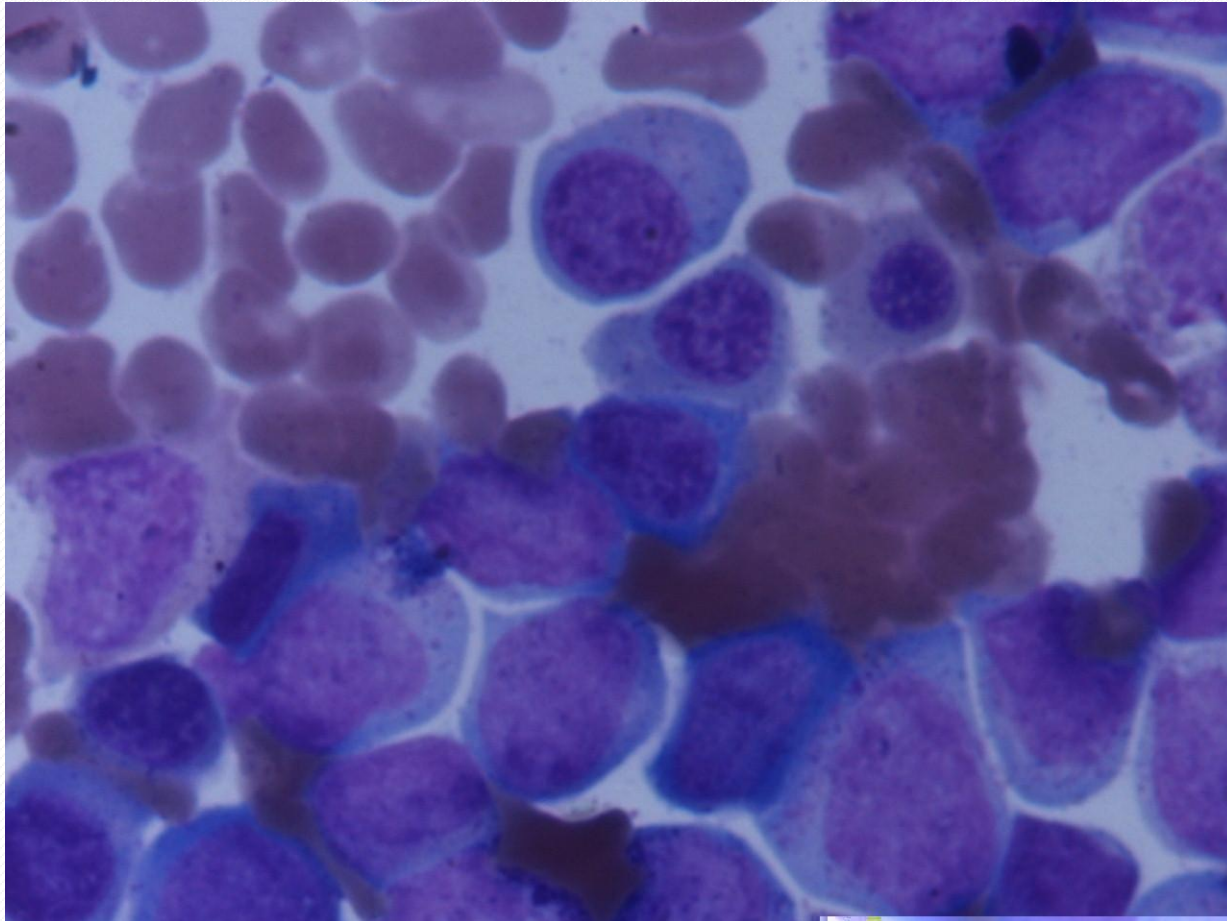


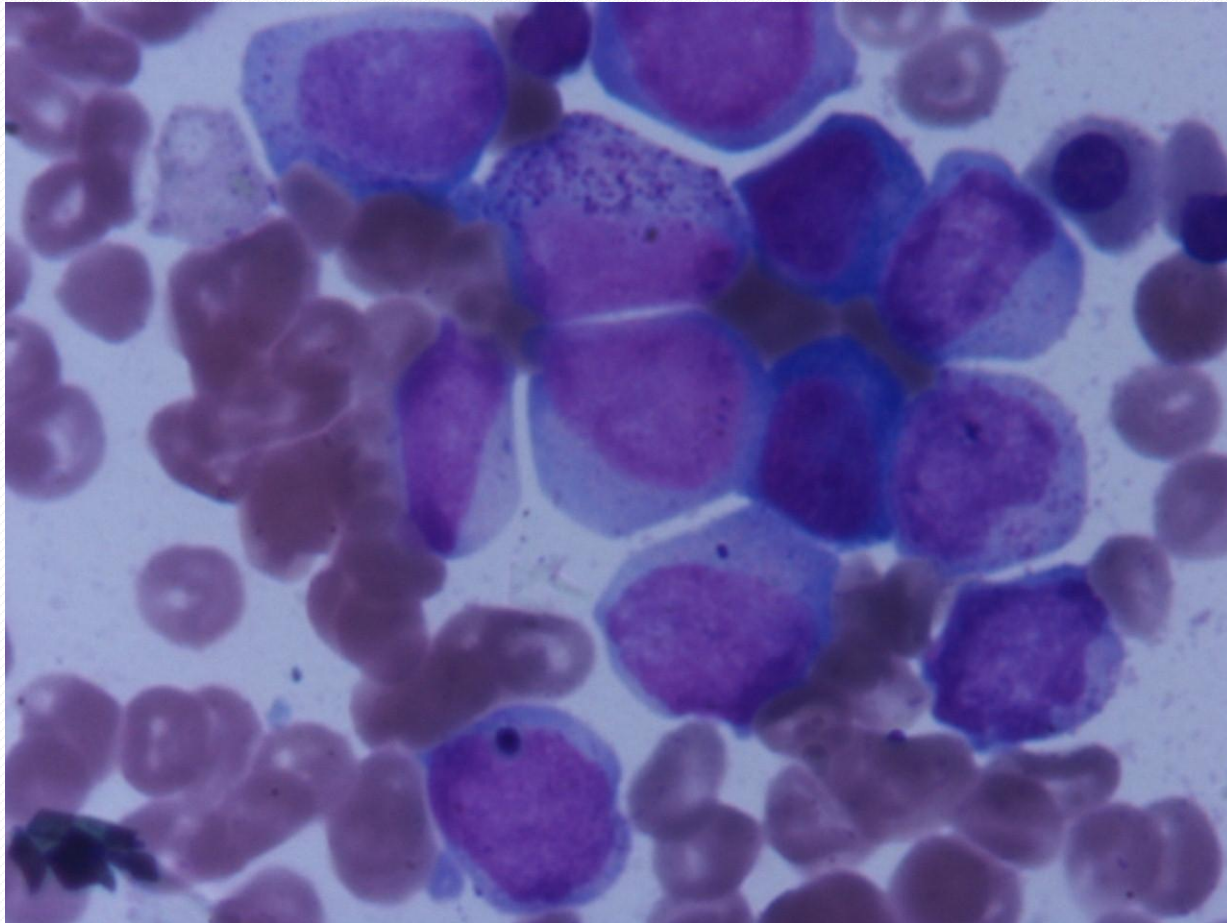
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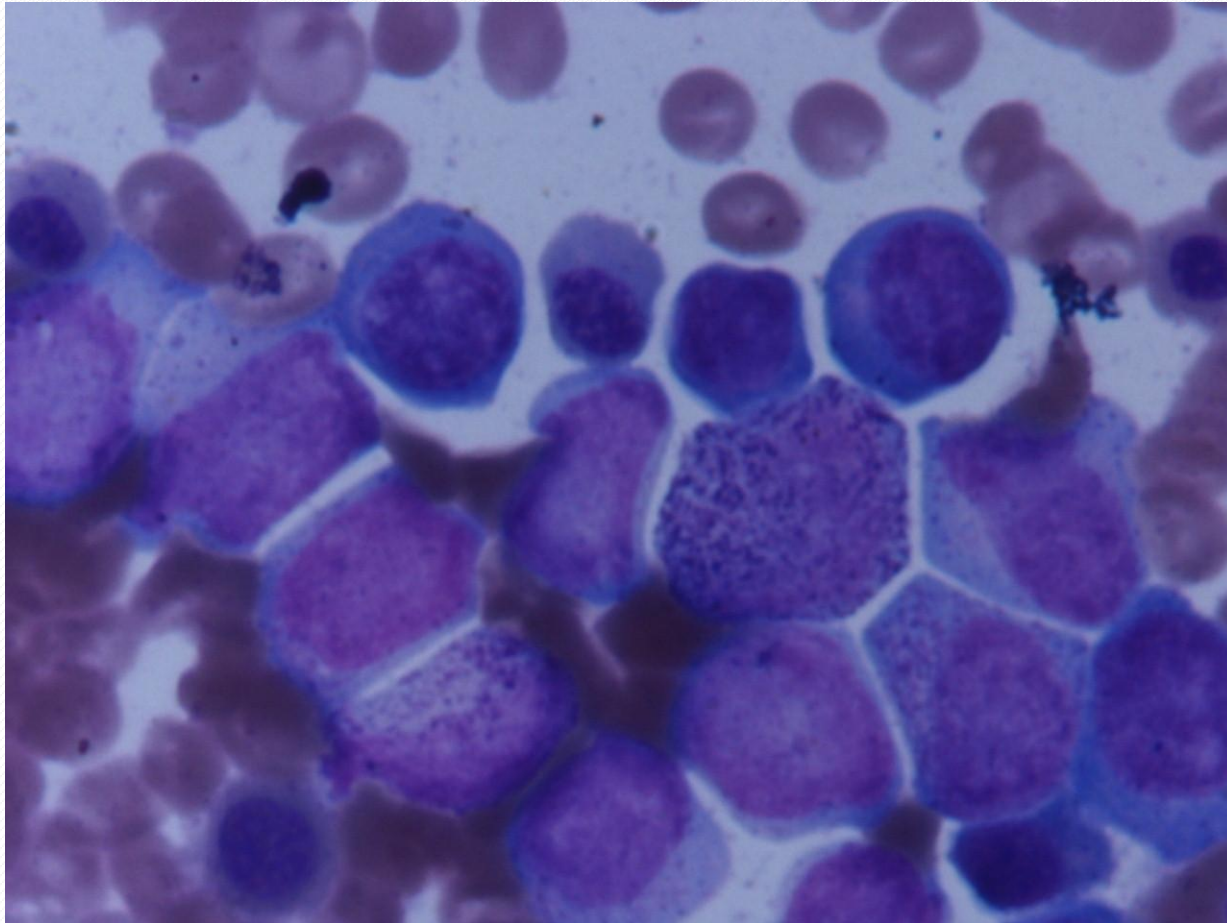


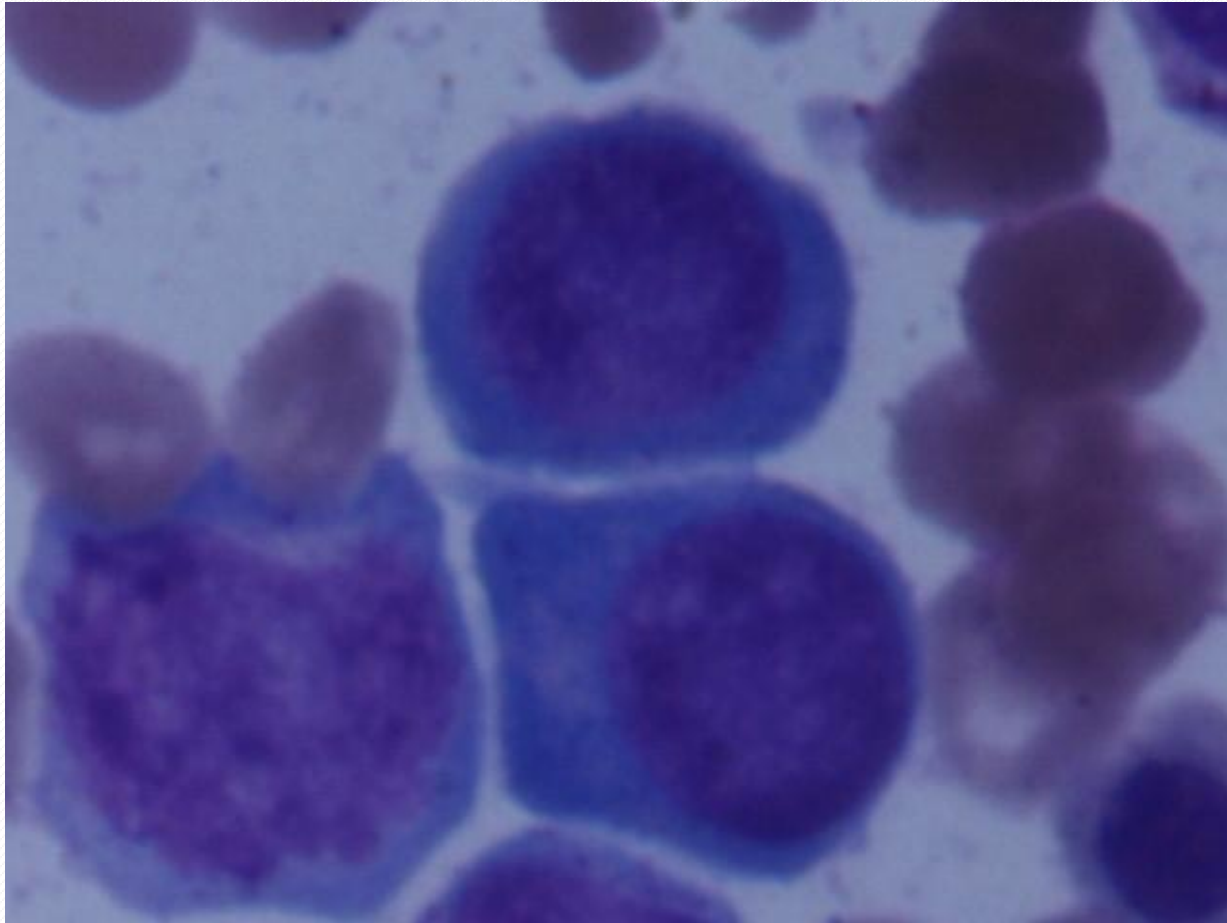
B.M aspiration report at 2/10/2013

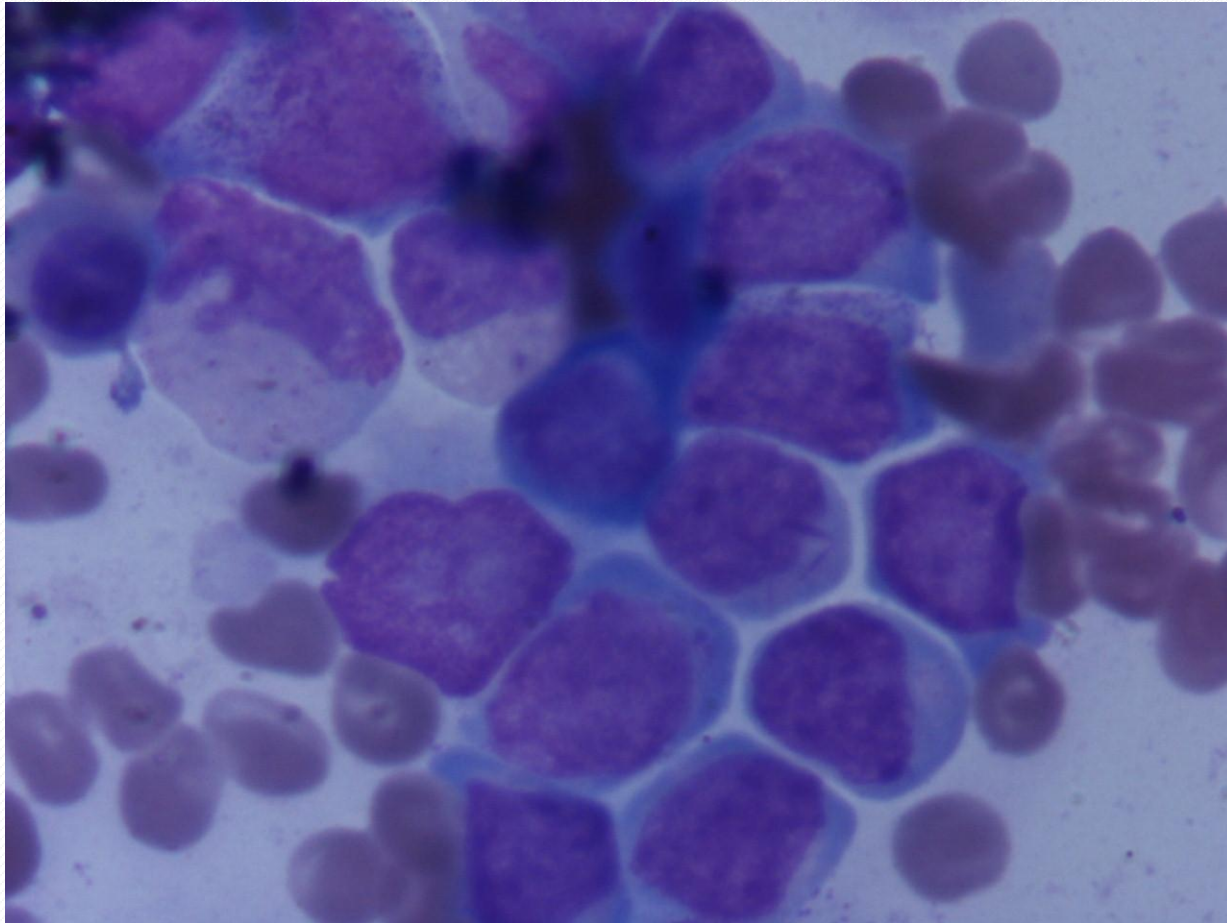
- Cellularity : hypercellular B.M fragments .
- Erythropoiesis : markedly increased activity forming 50% of all nucleated cells , megaloblastic maturation
- Leucopoiesis : reduced normal activity with diffuse infiltration of marrow by blast forming 31% of non erythroid element of marrow cells ,few contain Auer rod
- Megakaryocytes : markedly reduced
- Conclusion : Acute myeloid leukemia AML M6 (erythroleukemia)

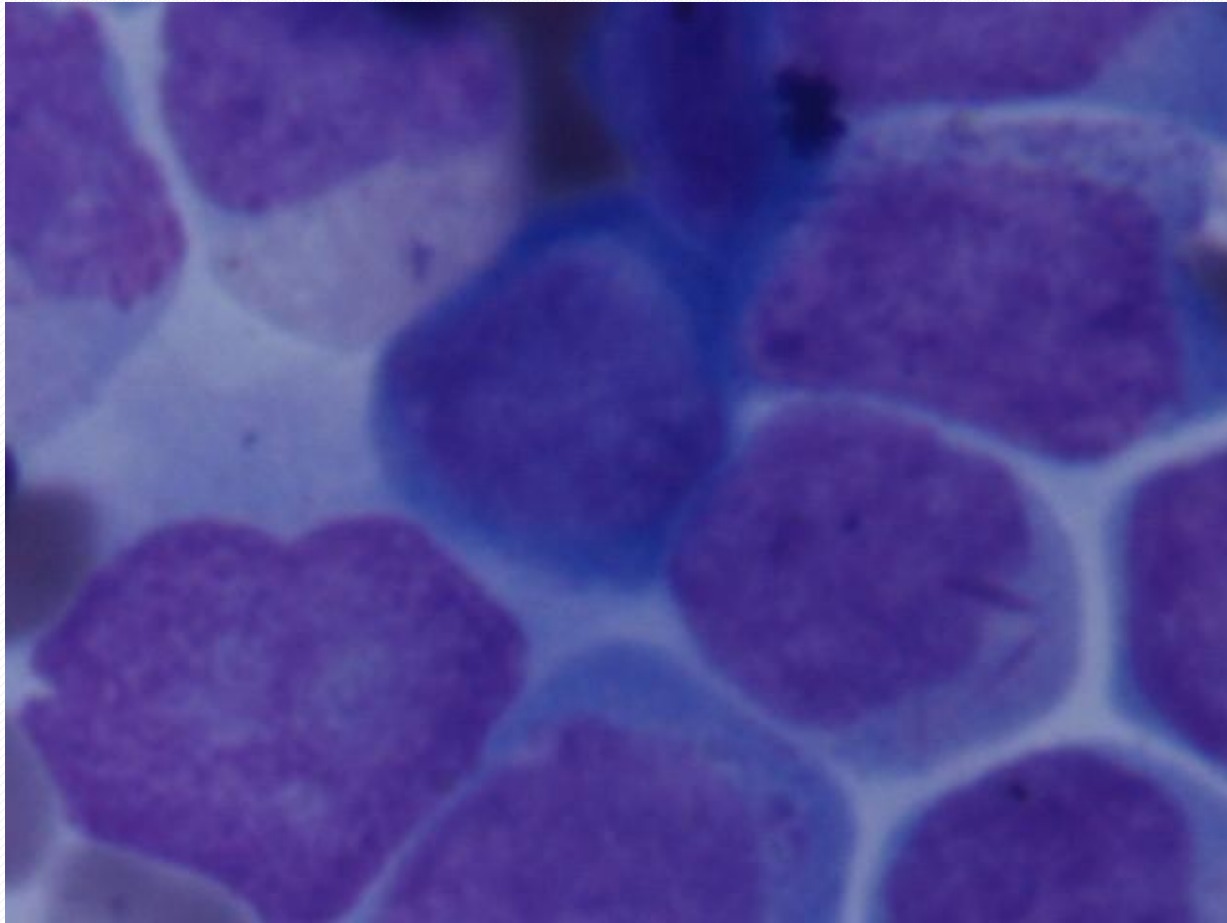


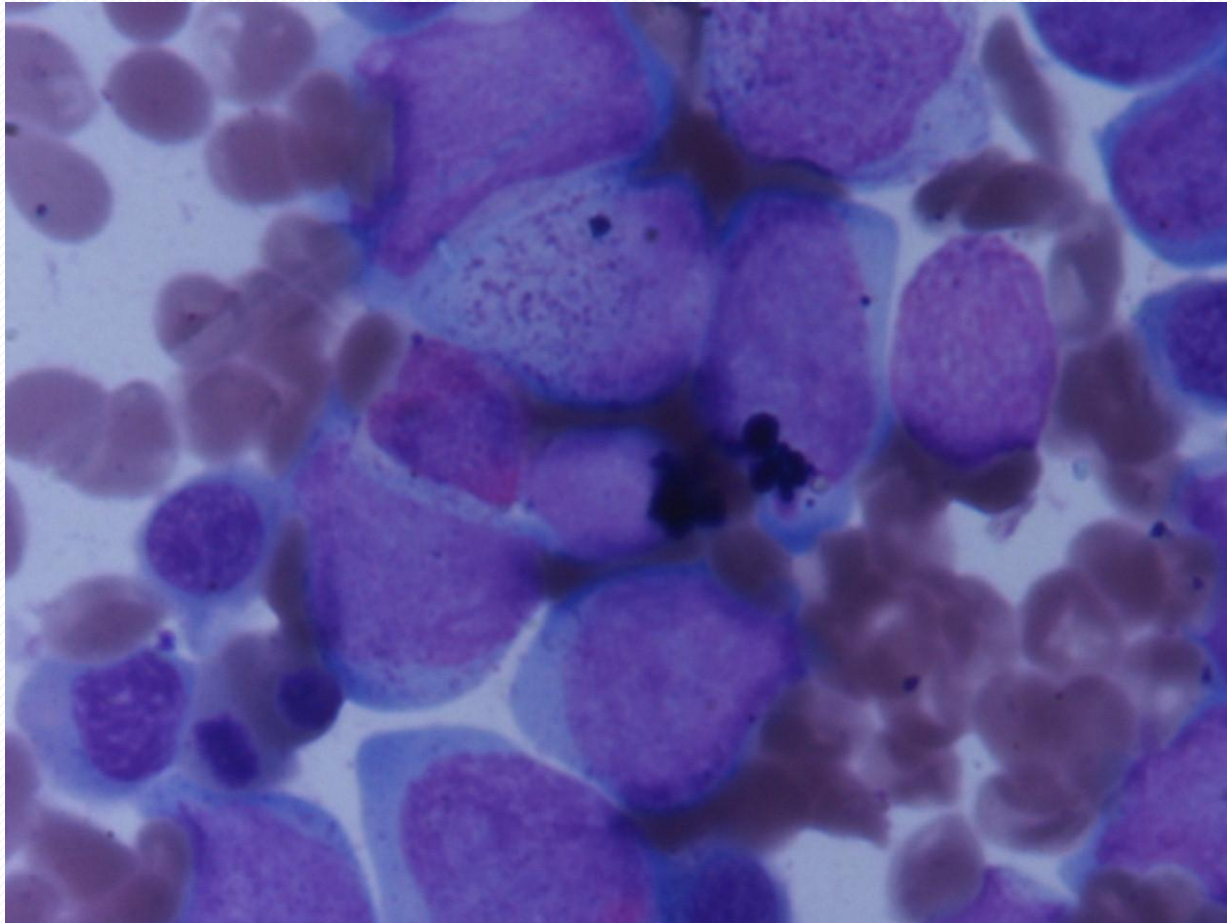












Acute erythroid leukemia

- Acute erythroid leukemia is a rare form of acute myeloid leukemia. It accounts for <5% of all acute myeloid leukemia cases. According to the World Health Organization 2008 classification, it falls under the category of acute myeloid leukemia and is further divided into two subtypes:
 - erythroid leukemia (erythroid/myeloid)
 - and pure erythroid leukemia. Currently, erythroleukemia . (erythroid/myeloid) is defined as 50% or more erythroid precursors and > 20% blasts of thenon-erythroid cells.
- By definition, pure erythroid leukemia is composed of > 80% erythroid precursors.


Differential diagnosis

Acute erythroid leukemia is a diagnosis of exclusion and difficulty. This review discusses its differential diagnosis, which present with erythroid proliferation, such as :

- myelodysplastic syndrome with erythroid proliferation,
- acute myeloid leukemia with myelodysplasia related changes
- myeloproliferative neoplasms with erythroblast transformation,
- acute myeloid leukemia with recurrent genetic abnormalities.
- Additionally, reactive conditions such as erythropoietin treatment, vitamin B12 and folate deficiency, toxin exposure and congenital dyserythropoiesis should be excluded.

Clinical presentation

- The incidence of this disease ranges from 3 to 8%. . There is a male preponderance. The age distribution appears to be bimodal, with a smaller peak below 20 years and a more definitive and broader peak in the seventh decade of life.
- In half of the cases, AML6 are secondary to chemotherapy or immunosuppressive therapy. AML6 more commonly complicate treatment with alkylating agents or benzene exposure. AML 6 may also develop as a blastic crisis of myeloproliferative disease or a final evolution of myelodysplastic syndrome.

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- The most common complaints reported are related to severe anemia.
 - One third of the patients were reported to have hemorrhage.
 - The presence of hepatomegaly or splenomegaly varies between 20 to 40% of the series.
 - Anemia is present in most patients, and is often severe (mean, 7.5 g/dl).

- **The bone marrow:** is hypercellular and shows major dysplasia in the red cells. Erythroid lineage is dysplastic, with macroblasts (asynchronous nucleo-cytoplasmic maturation) and basophilic stippling, lack of hemoglobinization and multinucleation. Multilineage dysplasia is present in most of the reports. Megakaryocytes are very often dysplastic, with abnormalities of segmentation of the nucleus or abnormalities of size (micromegakaryoblasts).

Cytochemistry

- A large variety of cytochemical stains are available for characterization of erythroid lineage.
- The periodic acid-Schiff reaction is always negative in normal erythroid differentiation. Aberrant positivity is very often observed in pronormoblasts and basophilic normoblasts in AML6.
- . Some authors reported occasional positivity of myeloperoxidase in erythroid cells in erythroleukemia.

Immunophenotyping

- The most known markers for AML6 have included glycoporphin A and transferrin receptor (CD71). However glycoporphin A has been reported to be completely negative in some cases of AML6 probably because it is a late erythroid marker, and CD71 is a non-specific activation marker which can be found in other AML.
- The platelet CD41 antigen and the myeloid markers as CD13 and CD33 can sometimes be positive.
- Probably the expression of Rh D antigen or of spectrin in AML6 could have some interest since they are expressed in immature erythroid cells.

- Markers like beta-sialoglycoprotein, Carbonic anhydrase 1, could be interesting tools to identify minimally differentiated M6. The most important role for antigenic erythroid markers is not in classical AML6, but rather to search for such a primitive erythroid phenotype among the acute undifferentiated leukemia.

Outcome

- The outcome is classically poor. In *de novo* AML6, treatment with intensive chemotherapy with anthracycline and aracytine gives a complete remission (CR) rate of about 62%. In other series, the level of CR does not exceed 10% to 40% especially in secondary AML6.. The median survival is about 23 weeks. The survival is related to karyotypic abnormalities.
- The allogeneic bone marrow transplantation seems to be the best treatment for patients with abnormalities of 5q or 7q. Even for therapy-related myelodysplasia, transplantation seems to be favorable for long-term disease-free survival.



THANK YOU