

Acute Myeloid Leukaemia

90 questions in three levels, with answer key

For NEET-PG, USMLE, DNB and DM revision and for classroom tests. Level 1 covers core facts; Level 2 tests applied reasoning (calculations, imaging and laboratory findings, trial interpretation, sequencing); Level 3 covers landmark trials and newer therapies. Answers and explanations are at the end. No drug doses.

Dr. Haritha Chiramana

MD (Internal Medicine), DM (Medical Oncology) · Senior Medical Oncologist & Hemato-Oncologist
C.R. Reddy Cancer Center, C.R. Reddy Superspeciality Hospital · info@drharitha.co.in · drharitha.co.in

18 October 2026 · Free for teaching with attribution (CC BY-NC-ND 4.0) · Online version with instant scoring:
drharitha.co.in/professionals/acute-myeloid-leukaemia-mcq

Level 1 · Basics

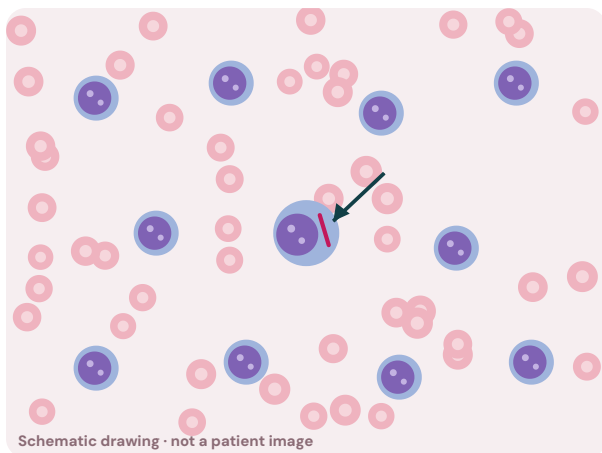
Q1. Diagnosis About what share of acute leukaemias in adults is acute myeloid leukaemia (AML)?

- A About 50%
- B About 80%
- C About 20%
- D Under 10%
- E Nearly 100%

Q2. Diagnosis Which statement about the white count at diagnosis of AML is correct?

- A It is always above 100,000/ μ L at diagnosis
- B It is normal only in APL, never in other AML
- C A count under 5,000/ μ L rules out AML
- D It is the test that makes the diagnosis
- E A normal or low count does not exclude AML

Q3. Diagnosis This schematic blood film is from a 55-year-old with fever and bruises. What is the pink rod-shaped inclusion marked by the arrow?



- A A Döhle body
- B A Howell-Jolly body
- C Toxic granulation
- D A Russell body
- E An Auer rod

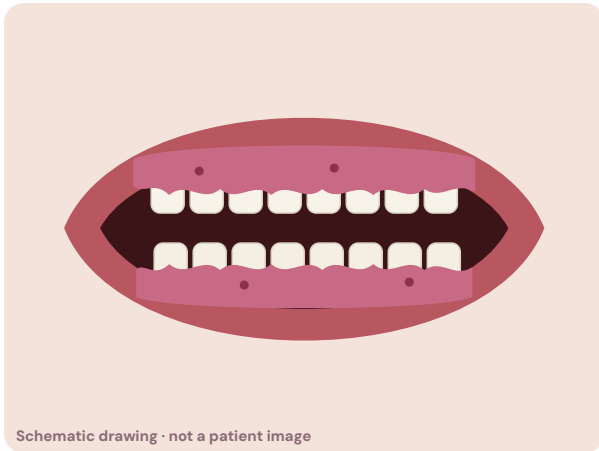
Q4. Diagnosis Which flow cytometry markers confirm myeloid lineage in a new acute leukaemia?

- A CD13, CD33, CD117 and MPO
- B CD20, CD5 and CD23
- C CD138 and CD38
- D CD3, CD7 and CD5
- E CD19, CD10, CD22 and CD79a

Q5. Diagnosis For most AML without a defining genetic lesion, what marrow or blood blast count is needed for the diagnosis?

- A 30% or more
- B 20% or more
- C 50% or more
- D 5% or more
- E 10% or more

Q6. Diagnosis This schematic drawing shows the gums of a 48-year-old with a new acute leukaemia. Which AML type does it suggest?



- A AML with t(8;21)
 - B Acute promyelocytic leukaemia
 - C Pure erythroid leukaemia
 - D Monocytic AML
 - E Chronic myeloid leukaemia in chronic phase
- Q7. Diagnosis** Which is a recognised cause of AML?
- A Earlier infectious mononucleosis
 - B Earlier alkylating chemotherapy
 - C Long-term low-dose aspirin use
 - D Chronic hepatitis C infection
 - E Long-standing iron deficiency
- Q8. Diagnosis** A mass biopsied from the orbit shows sheets of myeloid blasts; the marrow has 6% blasts. How is this classified?
- A A lymphoma until proved otherwise
 - B MDS, because marrow blasts are under 20%
 - C A small round cell tumour of the orbit
 - D AML (myeloid sarcoma), whatever the marrow
 - E A benign reactive inflammatory lesion
- Q9. Treatment** A 26-year-old has gum bleeding, a low fibrinogen and many granular cells on the smear. When should ATRA be started?
- A After the bleeding has stopped
 - B After the bone marrow report
 - C At once, the same day, on suspicion of APL
 - D Only if the white count is above 10,000/ μ L
 - E Only after PML::RARA is confirmed
- Q10. Diagnosis** Which genetic lesion defines acute promyelocytic leukaemia (APL)?
- A t(9;22) BCR::ABL1
 - B t(15;17) PML::RARA
 - C inv(16) CBFB::MYH11
 - D t(9;11) MLLT3::KMT2A
 - E t(8;21) RUNX1::RUNX1T1
- Q11. Treatment** What does "7+3" induction mean?
- A 7 weeks of tablets followed by 3 weeks off
 - B 7 cycles of azacitidine with 3 of venetoclax
 - C 7 days of anthracycline with cytarabine on days 1–3 only
 - D 7 days of ATRA with 3 days of arsenic
 - E 7 days of cytarabine with an anthracycline on days 1–3

- Q12. Treatment** A fit 45-year-old has AML with a FLT3 mutation. What is added to 7+3 induction?
- A Imatinib or dasatinib
 - B Rituximab or obinutuzumab
 - C Midostaurin or quizartinib
 - D ATRA or arsenic trioxide
 - E Venetoclax or azacitidine
- Q13. Treatment** Which drug is added to induction for CD33-positive core-binding-factor AML?
- A Midostaurin
 - B Blinatumomab
 - C Arsenic trioxide
 - D Ivosidenib
 - E Gemtuzumab ozogamicin
- Q14. Treatment** What is the standard first treatment for an older adult with AML who is unfit for intensive chemotherapy but not frail?
- A Venetoclax with azacitidine
 - B Imatinib
 - C Supportive care only, with no drugs
 - D ATRA with arsenic trioxide
 - E 7+3 induction
- Q15. Treatment** What is the preferred induction for low- or intermediate-risk APL (white count 10,000/ μ L or less)?
- A ATRA with arsenic trioxide
 - B 7+3 without ATRA
 - C Allogeneic transplant
 - D ATRA alone
 - E Venetoclax with azacitidine
- Q16. Treatment** A patient on induction has a temperature of 38.6°C and neutrophils of 200/ μ L. What is the first step?
- A Paracetamol now and review on the morning round
 - B Wait 24 hours for the blood culture results before antibiotics
 - C Granulocyte transfusion before any antibiotic
 - D Start an antifungal only, with no antibiotic
 - E Cultures, then an antipseudomonal beta-lactam within 60 min
- Q17. Treatment** A breathless patient has a white count of 200,000/ μ L and haemoglobin of 6 g/dL. What should be avoided if possible?
- A Starting induction
 - B Intravenous fluids
 - C Platelet transfusion if bleeding
 - D Hydroxyurea
 - E A rapid red cell transfusion
- Q18. Treatment** For a patient at high risk of tumour lysis, which drug is given with intravenous fluids, after checking for G6PD deficiency?
- A Furosemide
 - B Sodium bicarbonate
 - C Allopurinol alone
 - D Rasburicase
 - E Calcium gluconate
- Q19. Treatment** What happens to most patients in complete remission who receive no post-remission therapy?
- A They are cured
 - B Half are cured without further treatment
 - C They relapse
 - D They develop MDS but not AML
 - E They develop CML

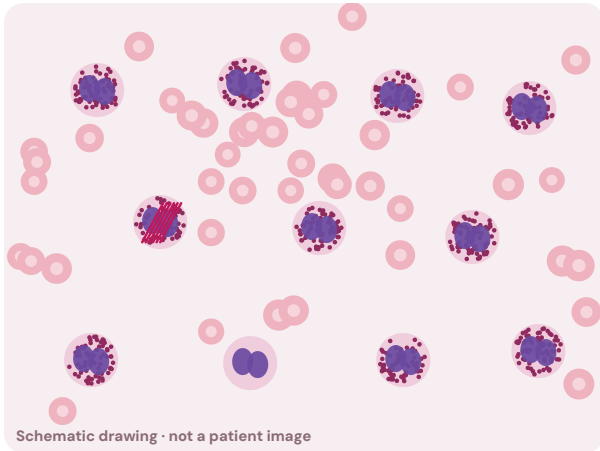
- Q20. Treatment** A fit 40-year-old with adverse-risk AML reaches first complete remission. What is the usual next step?
- A Allogeneic transplant in first remission
 - B Two years of ATRA maintenance
 - C Oral azacitidine maintenance alone
 - D Autologous stem cell transplant
 - E No further treatment, with close follow-up
- Q21. Diagnosis** Which genetic finding places AML in the ELN 2022 favourable group?
- A TP53 mutation
 - B t(6;9) DEK::NUP214
 - C A complex karyotype
 - D Monosomy 7
 - E t(8;21) RUNX1::RUNX1T1
- Q22. Diagnosis** How is a TP53 mutation in AML best described?
- A Adverse in every risk model
 - B Cured by ATRA
 - C Without prognostic meaning
 - D Favourable with intensive chemotherapy
 - E Favourable with venetoclax
- Q23. Treatment** A patient on ATRA for APL develops fever, breathlessness and weight gain. Which drug is started at once?
- A Heparin
 - B Dexamethasone
 - C Furosemide alone
 - D Erythropoietin
 - E Vancomycin alone
- Q24. Survivorship** Which is a recognised late effect of AML treatment?
- A Loss of hearing from daunorubicin
 - B Heart damage from anthracyclines
 - C Diabetes from 7+3
 - D Liver cirrhosis from ATRA
 - E Chronic kidney stones from cytarabine
- Q25. Survivorship** In English data (2001–2006), what was the main factor by which 5-year survival from AML fell?
- A Age at diagnosis
 - B Blood group
 - C Height
 - D Place of birth
 - E Sex
- Q26. Diagnosis** Where is a bone marrow sample taken in suspected AML?
- A Anterior tibia, as in adults generally
 - B Sternum, for both aspirate and biopsy
 - C Posterior iliac crest
 - D A vertebral body, under CT guidance
 - E The femoral head, under anaesthesia
- Q27. Treatment** When is a lumbar puncture done in an adult with newly diagnosed AML?
- A In APL, on the first day
 - B Only after transplant
 - C Only for neurological symptoms
 - D Before every cycle of chemotherapy
 - E In every patient at diagnosis

- Q28. Diagnosis** In the Vellore series of 380 patients with non-APL AML, why did most not start standard treatment?
- A Advanced age
 - B Lack of money
 - C Lack of a diagnosis
 - D Refusal of transfusion
 - E Preference for herbal medicine
- Q29. Diagnosis** Which tests are sent at once in every suspected case of AML to look for DIC?
- A Serum protein electrophoresis
 - B HbA1c and a lipid profile
 - C PT, aPTT, fibrinogen and D-dimer
 - D Troponin and creatine kinase
 - E Serum ferritin and transferrin
- Q30. Diagnosis** A 35-year-old has had "viral fever" for 3 weeks with pallor and new bruises. What is the right test today?
- A A chest X-ray only
 - B A dengue test and a review in a week
 - C A complete blood count with a smear
 - D Another course of oral antibiotics
 - E A vitamin B12 level only

Level 2 · Applied & innovative

- Q31. Diagnosis** A marrow shows 12% blasts with t(8;21) RUNX1::RUNX1T1. What is the diagnosis?
- A MDS/AML
 - B MDS with excess blasts
 - C AML
 - D CML in blast phase
 - E A reactive marrow after infection
- Q32. Diagnosis** A marrow has 8% blasts and an NPM1 mutation. How does the WHO 5th edition classify it?
- A Clonal haematopoiesis only
 - B AML with mutated NPM1
 - C MDS with low blasts
 - D A normal marrow for age
 - E MDS/AML (ICC term)
- Q33. Diagnosis** Under ICC 2022, how is a marrow with 15% blasts and no defining genetic lesion classified?
- A Acute leukaemia of ambiguous lineage
 - B MDS/AML
 - C Myeloproliferative neoplasm
 - D MDS with low blasts
 - E AML
- Q34. Diagnosis** A patient with a white count of 250,000/ μ L has a potassium of 7.2 mmol/L but a normal ECG. What should you do first?
- A Ignore it, as high counts never affect potassium
 - B Give insulin-dextrose and calcium at once
 - C Recheck potassium on a heparinised plasma sample
 - D Give a potassium binder and stop fluids
 - E Start urgent haemodialysis

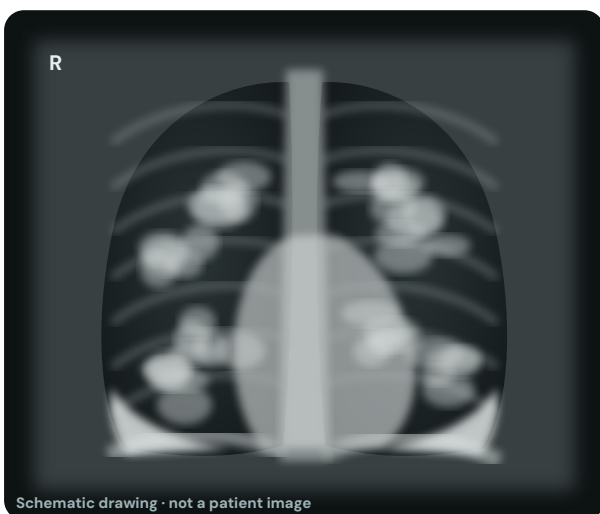
Q35. Diagnosis This schematic blood film is from a 28-year-old with gum bleeding and a fibrinogen of 90 mg/dL. What is the immediate step?



- A Insert a central line for induction today
 - B Start heparin for the clotting disorder
 - C Lumbar puncture to look for CNS disease first
 - D Start ATRA now, before PML::RARA is confirmed
 - E Wait for FISH results before any treatment
- Q36. Treatment** Which transfusion targets are used during induction for APL?

- A Platelets only for bleeding; fibrinogen not measured
 - B Platelets $\geq 150,000/\mu\text{L}$; fibrinogen > 400 mg/dL
 - C Plasma only, with no platelet transfusion at all
 - D Platelets $\geq 10,000/\mu\text{L}$; no fibrinogen target needed
 - E Platelets $\geq 30,000$ – $50,000/\mu\text{L}$; fibrinogen > 150 mg/dL
- Q37. Treatment** APL is confirmed in a 32-year-old with a white count of $14,000/\mu\text{L}$. Which induction fits?
- A ATRA + arsenic alone, exactly as for low risk
 - B 7+3 alone, with ATRA kept for relapse only
 - C High-risk treatment: ATRA with an anthracycline
 - D Venetoclax with azacitidine as first induction
 - E Arsenic alone, without ATRA, until counts fall

Q38. Treatment On day 9 of ATRA and arsenic for APL, a patient has fever, breathlessness, a 6 kg weight gain and this schematic chest X-ray. What is the best plan?



- A Dexamethasone now; usually continue the drugs
- B Stop ATRA and arsenic permanently today
- C Increase ATRA to speed up differentiation
- D Treat as pneumonia with antibiotics alone
- E Diuretics only, and review again tomorrow

- Q39. Treatment** A fit 66-year-old develops AML 5 years after chemotherapy for breast cancer (intermediate-risk genetics). Which induction is preferred?
- A ATRA with arsenic
 - B 7+3 with gemtuzumab
 - C Low-dose cytarabine
 - D CPX-351
 - E Imatinib with 7+3
- Q40. Diagnosis** A 30-year-old with AML has a RUNX1 variant at about half the reads on NGS, and an aunt had leukaemia. A sibling is the planned donor. What must be done first?
- A Check if the variant is germline; test the sibling
 - B Cancel transplant and give chemotherapy only
 - C Ignore it, since RUNX1 variants are never inherited
 - D Proceed with the sibling as donor without tests
 - E Use the patient's own stem cells (autologous)
- Q41. Treatment** A 35-year-old with NPM1-mutated AML (no FLT3-ITD) is in remission with negative MRD. What is suggested?
- A Oral azacitidine only, without consolidation
 - B Allogeneic transplant in first remission for all
 - C No further treatment after induction
 - D Consolidation; transplant only if MRD persists or rises
 - E ATRA maintenance for two years after induction
- Q42. Treatment** For a 45-year-old with intermediate-risk AML in first remission, what decides between consolidation and transplant?
- A MRD, donor availability and transplant risk
 - B Sex and ABO blood group of the patient
 - C The number of transfusions in induction
 - D The white count at diagnosis, on its own
 - E Whether Auer rods were seen on the film
- Q43. Treatment** Who should be offered oral azacitidine maintenance after consolidation, as in QUAZAR?
- A Only patients after allogeneic transplant
 - B Only patients with APL after arsenic
 - C All patients with favourable-risk AML
 - D Children with high-risk AML after induction
 - E Age 55 or more, intermediate or adverse risk
- Q44. Treatment** A 72-year-old with ECOG 1, Charlson 1 and normal heart, lung, liver and kidney function has AML. Which is correct?
- A Supportive care is the only reasonable option
 - B Age over 70 means lower-intensity therapy only
 - C She is frail by definition at this age
 - D She can be treated as fit for intensive therapy
 - E Transplant is impossible at any age above 70
- Q45. Treatment** An unfit 76-year-old has IDH1-mutated AML. Which combination improved survival in AGILE?
- A Midostaurin with 7+3
 - B Ivosidenib with azacitidine
 - C Quizartinib with decitabine
 - D Gilteritinib with azacitidine
 - E Enasidenib with cytarabine
- Q46. Treatment** A patient on venetoclax with azacitidine is started on posaconazole prophylaxis. What follows?
- A Venetoclax dose is adjusted (azoles raise levels)
 - B Venetoclax is replaced by 7+3 induction
 - C Posaconazole must never be given with it
 - D Azacitidine is stopped while on posaconazole
 - E No change is needed to either drug

- Q47. Treatment** When is the marrow first checked on venetoclax or ivosidenib combinations?
- A Only if the patient feels unwell
 - B Never, because blood counts are enough
 - C Before each daily dose of venetoclax
 - D Early in cycle 1 (day 14–21)
 - E Only after 12 cycles of treatment
- Q48. Treatment** AML relapses 10 months after consolidation; FLT3-ITD is now present. Which drug improved survival over salvage chemotherapy?
- A Imatinib
 - B ATRA
 - C Midostaurin
 - D Ivosidenib
 - E Gilteritinib
- Q49. Treatment** Relapsed AML has an NPM1 mutation. Which class of drug is now FDA-approved for it?
- A BCR::ABL1 inhibitors
 - B BTK inhibitors
 - C Menin inhibitors
 - D JAK2 inhibitors
 - E Anti-CD20 antibodies
- Q50. Treatment** Relapsed AML has an IDH2 mutation. Which targeted drug is used?
- A Gilteritinib
 - B Enasidenib
 - C Gemtuzumab ozogamicin
 - D Ivosidenib
 - E Revumenib
- Q51. Diagnosis** Why must the genetic tests be repeated when AML relapses?
- A Relapse is always a new, unrelated leukaemia
 - B Clones evolve, so the target may have changed
 - C Laboratories require it for billing
 - D The first results expire after 6 months
 - E Genetics never change, but staging does
- Q52. Treatment** A patient with AML develops a new facial nerve palsy; CSF shows blasts. What is the right approach?
- A High-dose systemic chemotherapy during cranial RT
 - B Wait for nerve recovery before any CNS treatment
 - C Intrathecal therapy alone, with RT only if it fails
 - D Craniospinal RT as the first and only choice
 - E Prompt cranial radiotherapy with intrathecal therapy
- Q53. Treatment** A myeloid sarcoma compresses the spinal cord. Which management is right?
- A Hydroxyurea alone, with RT kept for later relapse
 - B Observation until the marrow relapses, then chemotherapy
 - C Surgery alone, with no systemic therapy afterwards
 - D Steroids, urgent MRI, RT and/or decompression, plus systemic therapy
 - E Systemic therapy alone, with no RT or surgery at any stage of care
- Q54. Treatment** On day 5 of neutropenic fever a stable patient is still febrile on piperacillin–tazobactam with negative cultures. What next?
- A Add an antifungal (4–7 days of unexplained fever)
 - B Add vancomycin routinely to the regimen
 - C Change to meropenem because of the fever alone
 - D Stop all antibiotics while cultures are negative
 - E Give a granulocyte transfusion today

- Q55. Treatment** Which antifungal prophylaxis is used during AML induction?
- A Oral nystatin only, throughout induction
 - B Fluconazole, or a mould-active azole in second induction
 - C None until fever appears, then amphotericin for everyone
 - D Amphotericin for every patient from day 1
 - E An echinocandin, but only after transplant
- Q56. Treatment** A patient at high risk of tumour lysis has G6PD deficiency. Which drug must be avoided?
- A Rasburicase
 - B Allopurinol
 - C Calcium gluconate for symptomatic hypocalcaemia
 - D Intravenous saline
 - E Hydroxyurea
- Q57. Treatment** AML is diagnosed at 9 weeks of pregnancy. What does the module advise?
- A Delay all treatment until delivery
 - B Start ATRA only
 - C Low-dose cytarabine throughout pregnancy
 - D Termination of pregnancy, then treatment
 - E Leukapheresis until term
- Q58. Survivorship** A man in remission from NPM1-mutated AML has a rising NPM1 transcript level in blood on two tests. What next?
- A Repeat the test in 2 years
 - B A marrow examination and plans for relapse
 - C Reassurance: blood MRD has no meaning
 - D Stop follow-up, as he is in remission
 - E Start ATRA maintenance at once
- Q59. Treatment** In hyperleukocytosis where induction must be delayed, which measure lowers the white count?
- A High-dose steroids for three days
 - B Red cell transfusion to dilute the blood
 - C Hydroxyurea
 - D Plasma exchange, repeated daily
 - E Leukapheresis alone, without any drug
- Q60. Survivorship** Which advice suits a person living after AML treatment?
- A Report fever only if it lasts a week
 - B Stay active and social, with sensible precautions
 - C Avoid all other people for five years after treatment
 - D Skip all vaccines after a transplant
 - E Never exercise again after treatment

Level 3 · Challenging

- Q61. Treatment** In RATIFY (midostaurin with 7+3 for FLT3-mutated AML), what was median overall survival?
- A 15 vs 10 months
 - B 26 vs 75 months
 - C 9.3 vs 5.6 months
 - D 75 vs 26 months
 - E 32 vs 15 months
- Q62. Treatment** In QuANTUM-First (quizartinib for FLT3-ITD AML), what was median overall survival?
- A 24 vs 8 months
 - B 75 vs 26 months
 - C 15 vs 10 months
 - D 25 vs 15 months
 - E 32 vs 15 months

- Q63. Treatment** In the CPX-351 phase 3 trial (patients aged 60–75 with secondary AML), what was 5-year overall survival?
- A 68% vs 34%
 - B 97% vs 77%
 - C 18% vs 8%
 - D 51% vs 44%
 - E 8% vs 18%
- Q64. Treatment** In VIALE-A, what were median overall survival and complete remission with venetoclax plus azacitidine versus azacitidine alone?
- A OS 10 vs 15 months; CR 18% vs 37%
 - B OS 32 vs 15 months; CR 70% vs 40%
 - C OS 24 vs 8 months; CR 50% vs 10%
 - D OS 15 vs 10 months; CR 37% vs 18%
 - E No survival difference
- Q65. Treatment** In AGILE, what was median overall survival with ivosidenib plus azacitidine in IDH1-mutated AML?
- A 12 vs 11 months
 - B 9.3 vs 5.6 months
 - C 24 vs 8 months
 - D 75 vs 26 months
 - E 15 vs 10 months
- Q66. Treatment** In ADMIRAL (relapsed FLT3-mutated AML), which pair of results is correct for gilteritinib vs salvage chemotherapy?
- A Median OS 9.3 vs 5.6 months
 - B Median OS 32 vs 15 months
 - C Median OS 5.6 vs 9.3 months
 - D No difference in survival
 - E Median OS 24 vs 8 months
- Q67. Treatment** In APLO406, what was 72-month event-free survival with ATRA plus arsenic versus ATRA plus chemotherapy?
- A 77% vs 97%
 - B 68% vs 34%
 - C 50% vs 30%
 - D 97% vs 77%
 - E 99% vs 98%
- Q68. Treatment** In QUAZAR, what was median overall survival with oral azacitidine maintenance versus placebo?
- A 75 vs 26 months
 - B 25 vs 15 months
 - C 48 vs 12 months
 - D 9 vs 6 months
 - E 15 vs 25 months
- Q69. Diagnosis** In the Short 2020 meta-analysis, what was 5-year overall survival with negative versus positive MRD?
- A 34% vs 68%
 - B 50% vs 45%
 - C 18% vs 8%
 - D 90% vs 10%
 - E 68% vs 34%
- Q70. Diagnosis** Under ELN 2024 for venetoclax plus azacitidine, what was median survival in the favourable, intermediate and adverse groups?
- A 5.5, 12.1 and 26.5 months
 - B 12.1, 26.5 and 5.5 months
 - C 36, 24 and 12 months
 - D 75, 26 and 15 months
 - E 26.5, 12.1 and 5.5 months

- Q71. Treatment** In PARADIGM (fit adults), how did azacitidine–venetoclax compare with 7+3?
- A More remissions and transplants, similar survival
 - B Better overall survival by about 20% at three years
 - C Identical remission and transplant rates
 - D Fewer remissions but much less toxicity
 - E More early deaths and fewer transplants
- Q72. Treatment** In relapsed IDH2–mutated AML (Stein 2017), which results are reported for enasidenib?
- A No responses
 - B CR 97%, median OS not reached
 - C CR 19%, median OS 9 months
 - D CR 34%, median OS 9.3 months
 - E CR 70%, median OS 32 months
- Q73. Treatment** What did ziftomenib achieve in its approval study of 112 adults with relapsed NPM1–mutated AML?
- A CR + CRh 75%
 - B CR 5%
 - C No responses
 - D CR + CRh 21%
 - E CR 97%
- Q74. Treatment** What did the Hills 2014 meta–analysis show for gemtuzumab ozogamicin with induction?
- A Benefit only in adverse–risk AML, none in CBF AML
 - B Benefit only in APL, with no gain in other AML
 - C More relapses and deaths with gemtuzumab added
 - D No survival benefit in any genetic risk group
 - E Fewer relapses and better survival, most in CBF AML
- Q75. Treatment** What did Fernandez 2009 show about induction in younger adults?
- A Idarubicin was harmful
 - B Anthracyclines added nothing
 - C Higher daunorubicin exposure improved outcomes
 - D Cytarabine for 3 days was as good as 7
 - E Induction can be omitted with good–risk genetics
- Q76. Diagnosis** At AIIMS, 480 patients aged 8–60 treated with 3+7 and high–dose cytarabine had what induction mortality?
- A 40%
 - B 60%
 - C 5%
 - D 18.4%
 - E 1%
- Q77. Treatment** In 102 patients with APL at SGPGIMS Lucknow (2014–2024), which finding is correct?
- A Induction mortality about 1%, as in clinical trials
 - B All deaths were caused by differentiation syndrome
 - C Induction mortality 24.5%; support limited by shortages
 - D No patient received ATRA during induction
 - E Median age 65, with most patients at low risk
- Q78. Treatment** How do early deaths in APL compare between clinical trials with early ATRA and population registries?
- A About 1% in both
 - B About 29% in trials vs 3.7% in population registries
 - C About 3.7% in trials vs up to 29% in registries
 - D About 50% in both
 - E Registries report fewer deaths than trials do

- Q79. Treatment** At an Indian tertiary cancer centre (Jain 2020), what proportion of AML patients grew multidrug-resistant organisms during induction?
- A 55%
 - B None, with prophylaxis
 - C 15%
 - D 90%
 - E 5%
- Q80. Survivorship** In England (2001–2006), what was 5-year survival from AML at age 70–79?
- A About 13%
 - B About 50%
 - C About 70%
 - D About 3%
 - E About 33%
- Q81. Diagnosis** What did Ganzel 2021 find about CNS involvement at diagnosis of adult AML?
- A Found in about 1–3%; it does not by itself worsen outcome
 - B It always requires craniospinal RT
 - C It doubles the risk of death
 - D Found in about half of patients
 - E It is an indication for routine prophylaxis in all adults
- Q82. Diagnosis** According to TCGA 2013, about how many mutations does an AML genome carry on average?
- A About 1,000, most of them in AML genes
 - B Exactly one driver and no other change
 - C About 13,000 across the genome
 - D None: only translocations drive AML
 - E About 13, about five in AML genes
- Q83. Diagnosis** Why can IDH inhibitors cause differentiation syndrome?
- A They destroy CD33-positive blasts very fast
 - B They lift the block caused by 2-hydroxyglutarate
 - C They inhibit FLT3 signalling in the blasts
 - D They release potassium from leukaemic cells
 - E They deplete fibrinogen and trigger DIC
- Q84. Treatment** About how often does differentiation syndrome occur with different drugs?
- A Only with ATRA in APL, and never with other drugs
 - B Only after allogeneic transplant, whatever the drug
 - C About 3% (APL), up to 20% (IDH/menin), about 25% (FLT3)
 - D About 25% (APL), up to 20% (IDH/menin), about 3% (FLT3)
 - E About 80% with every drug that causes differentiation
- Q85. Diagnosis** Which MRD method suits NPM1-mutated AML, as shown by Ivey 2016?
- A Quantitative PCR
 - B Monthly karyotype
 - C PET-CT
 - D Serum LDH trend
 - E Marrow morphology alone
- Q86. Treatment** How do myeloablative and reduced-intensity conditioning compare for AML transplant?
- A Myeloablative is better for relapse, deaths and survival
 - B Myeloablative: fewer relapses, more deaths; similar survival
 - C They differ only in cost, not in any outcome
 - D Reduced intensity is never used for AML transplant
 - E Reduced intensity gives fewer relapses than myeloablative

Q87. Treatment After an ATO-containing regimen and molecular remission in APL, how often does relapse occur, and what is done?

- A 5–10% (20–30% if high risk); ATRA + arsenic, then auto-HCT
- B About 1%; ATRA maintenance is then given for life
- C About 80%; palliative care is the usual approach
- D Never; no further PCR follow-up is ever needed
- E About 50%; allogeneic transplant in every relapsed case

Q88. Treatment What did the CMC Vellore studies (Mathews 2006, 2010) show in newly diagnosed APL?

- A Chemotherapy alone beats arsenic
- B ATRA is ineffective in Indian patients
- C APL cannot be cured without transplant
- D APL is very rare in India
- E Durable remissions with arsenic alone

Q89. Treatment What is the evidence for leukapheresis in hyperleukocytosis (Oberoi 2014)?

- A It prevents tumour lysis
- B It halves early mortality
- C It replaces induction chemotherapy
- D It is mandatory above 50,000/ μ L
- E No proven benefit on its own

Q90. Diagnosis In what proportion of AML/MDS in young adults may an inherited predisposition be the cause?

- A Under 1%
- B About 50%
- C All cases
- D 10–20%
- E About 80%

Answer key

1-B 2-E 3-E 4-A 5-B 6-D 7-B 8-D 9-C 10-B 11-E 12-C 13-E 14-A 15-A 16-E 17-E 18-D 19-C 20-A 21-E 22-A
23-B 24-B 25-A 26-C 27-C 28-B 29-C 30-C 31-C 32-B 33-B 34-C 35-D 36-E 37-C 38-A 39-D 40-A 41-D
42-A 43-E 44-D 45-B 46-A 47-D 48-E 49-C 50-B 51-B 52-E 53-D 54-A 55-B 56-A 57-D 58-B 59-C
60-B 61-D 62-E 63-C 64-D 65-C 66-A 67-D 68-B 69-E 70-E 71-A 72-C 73-D 74-E 75-C 76-D 77-C 78-C
79-A 80-D 81-A 82-E 83-B 84-D 85-A 86-B 87-A 88-E 89-E 90-D

Explanations

- Q1 · B** AML is the commonest acute leukaemia of adults; median age at diagnosis is 65–68 in Western registries. In children under 10 it is under 10% of acute leukaemias, where ALL dominates.
- Q2 · E** A quarter to two-fifths of patients have fewer than 5,000 white cells (median about 15,000/ μ L). The smear, not the count, makes the diagnosis: blasts are seen in nearly all patients.
- Q3 · E** Auer rods are fused azurophilic granules in myeloblasts. They are pathognomonic of myeloid lineage; bundles of them ("faggot cells") are typical of APL.
- Q4 · A** Myeloid blasts express CD13, CD33, CD117 and MPO (often CD34). Flow also sets the baseline for measuring residual disease (MRD) later.
- Q5 · B** Most AML needs $\geq 20\%$ blasts (also BCR::ABL1-positive AML). Defining lesions (APL, CBF fusions, KMT2A and others) need $\geq 10\%$, and NPM1-mutated AML any count under WHO. Myeloid sarcoma counts as AML at any marrow count.
- Q6 · D** Gum hypertrophy and violaceous skin nodules (leukaemia cutis) suggest monocytic AML. Patients are often sent to the dentist first: a blood count with a smear is the right test.
- Q7 · B** Known risks are previous chemotherapy (alkylating agents, topoisomerase II inhibitors) or radiotherapy, an earlier MDS or MPN, benzene, smoking and obesity. Most AML arises from clonal haematopoiesis that gathers further mutations.
- Q8 · D** Myeloid sarcoma counts as AML at any marrow blast count and is treated as AML. It needs myeloid immunostains, because it is easily called a small round cell tumour or lymphoma.
- Q9 · C** Bleeding with low fibrinogen and granular cells is APL until proved otherwise. ATRA is started on suspicion; confirmation by PCR or FISH comes later.
- Q10 · B** PML::RARA is found in $\geq 95\%$ of APL. It is confirmed by PCR or FISH and is also the marker followed to molecular remission.
- Q11 · E** 7+3 gives complete remission in about three-quarters of fit younger adults. Daunorubicin or idarubicin is the anthracycline.
- Q12 · C** Midostaurin (ITD or TKD) or quizartinib (ITD) is added to induction and continued in consolidation and maintenance. Every fit FLT3-mutated patient should get one.
- Q13 · E** Gemtuzumab ozogamicin targets CD33, which nearly all CBF AML express. It gives fewer relapses and better 5-year survival, with the largest benefit in CBF AML (Hills 2014).
- Q14 · A** In VIALE-A, venetoclax with azacitidine improved survival over azacitidine alone. IDH1-mutated AML can be treated with ivosidenib plus azacitidine.
- Q15 · A** ATRA plus arsenic trioxide gave 72-month event-free survival of about 97% in APL0406. Where arsenic is not available, ATRA with an anthracycline is used.
- Q16 · E** Neutropenic fever is an emergency: cultures, then an antipseudomonal beta-lactam within 60 minutes. Local resistance patterns guide the choice.
- Q17 · E** Red cells raise blood viscosity and can precipitate leukostasis. Cytoreduce first (induction, or hydroxyurea if induction is delayed) and transfuse slowly only if essential.
- Q18 · D** High risk (in AML, WBC $\geq 100,000/\mu$ L): fluids plus rasburicase, which is avoided in G6PD deficiency. Intermediate risk: fluids plus allopurinol.

- Q19 · C** Without consolidation or transplant almost everyone relapses. The choice depends on ELN risk, MRD, age and fitness.
- Q20 · A** Adverse risk: allogeneic HCT in CR1. Favourable risk: consolidation, with transplant only for persistent or rising MRD. Intermediate risk: decided on MRD, donor and transplant risk.
- Q21 · E** Favourable: t(8;21), inv(16)/t(16;16), NPM1 mutated without FLT3-ITD, and in-frame bZIP CEBPA. The others are adverse.
- Q22 · A** TP53-mutated AML (about 8%, more in older and therapy-related cases) responds poorly to all current therapy; trials are preferred.
- Q23 · B** Differentiation syndrome is treated with dexamethasone as soon as it is suspected ("dexamethasone first, questions later"), with cultures, antibiotics, oxygen and diuretics as needed.
- Q24 · B** Late effects include anthracycline heart damage, infertility, chronic graft-versus-host disease and infections after transplant, second cancers, fatigue and the financial toll of treatment.
- Q25 · A** 5-year survival was about 50% under 40, 33% at 40–59, 13% at 60–69 and 3% at 70–79. Results are better with favourable genetics and modern targeted and transplant care.
- Q26 · C** A unilateral aspirate (500-cell count) and biopsy are taken from the posterior iliac crest; the sternum is acceptable for aspiration only. Warn the laboratory: fresh samples are needed for flow, karyotype, FISH and molecular tests.
- Q27 · C** There is no routine CNS prophylaxis in adult AML. Image first if there are focal signs; correct platelets and clotting; in APL defer the LP until the coagulopathy is corrected.
- Q28 · B** Only 29% started standard treatment, and 81% of the rest did not for lack of money. Median age was 40, symptoms had lasted a median of 4 weeks, and patients travelled a median of 580 km.
- Q29 · C** DIC is common in APL and in monocytic AML with a high count. Results guide platelet and cryoprecipitate support and raise the suspicion of APL; recheck often in APL.
- Q30 · C** Fever with pallor, bruises or gum bleeding needs a CBC with a smear the same day. Fatigue and pallor often precede the diagnosis by months.
- Q31 · C** With a defining lesion (APL, CBF fusions, KMT2A, DEK::NUP214, MECOM, CEBPA), ≥10% blasts are enough for AML. t(8;21) occurs in 1–7% of adults (median age 25–30) and is favourable.
- Q32 · B** WHO accepts NPM1-mutated AML at any blast count; ICC 2022 asks for ≥10%. NPM1 without FLT3-ITD is favourable, and NPM1 transcripts can be followed by PCR.
- Q33 · B** ICC calls 10–19% blasts MDS/AML; ≥20% is AML for most cases without a defining lesion.
- Q34 · C** Potassium leaks from cells in a clotted sample (pseudohyperkalaemia). A huge count also lowers glucose and oxygen in the tube; send the blood gas on ice.
- Q35 · D** Abnormal promyelocytes with dense granules and bundles of Auer rods mean APL until proved otherwise. ATRA is started on suspicion; lines, lumbar puncture and heparin are avoided during induction.
- Q36 · E** Platelets and cryoprecipitate keep these targets; no heparin; antifibrinolytics only in selected patients. In Lucknow, 34% got less support than planned because of shortages.
- Q37 · C** A white count >10,000/μL is high risk: ATRA with an anthracycline, or ATRA plus arsenic with hydroxyurea or an anthracycline for a rising count.
- Q38 · A** Differentiation syndrome (≥3 features: fever, hypoxia, weight gain >5 kg, lung opacities or effusions, hypotension, kidney injury) needs dexamethasone at once. Pause ATRA or arsenic only if severe or not better in 24–48 hours.
- Q39 · D** For therapy-related or myelodysplasia-related AML with favourable or intermediate genetics, CPX-351 gave 5-year OS of 18% vs 8% with 7+3 in patients aged 60–75, with more patients reaching transplant.
- Q40 · A** Familial disorders underlie 10–20% of AML/MDS in young adults. Clues: relatives with blood cancers, long-standing cytopenias, a germline-type variant (DDX41, RUNX1, GATA2, biallelic CEBPA). A sibling carrying it should not be the donor.
- Q41 · D** Favourable risk: consolidation (intermediate-dose cytarabine suggested over high-dose for most). Transplant is kept for persistent or rising MRD.

- Q42 · A** Intermediate risk is decided on MRD, donor and transplant risk. MRD-negative patients had 5-year OS of 68% vs 34% in a meta-analysis.
- Q43 · E** QUAZAR showed longer survival (median OS 25 vs 15 months). FLT3-mutated AML gets an FLT3 inhibitor after chemotherapy or after transplant instead.
- Q44 · D** Fitness (ECOG, Charlson or HCT-CI, organ function, cognition, wishes), not age alone, decides intensive versus lower-intensity therapy. Transplant eligibility also depends on organ function, not age.
- Q45 · B** AGILE: overall survival 24 vs 8 months. Venetoclax with azacitidine is the other main option for unfit adults.
- Q46 · A** Azole antifungals raise venetoclax levels, so its dose is adjusted. The marrow is checked early in cycle 1 because responses are fast and counts may need time off venetoclax.
- Q47 · D** Responses are fast and counts may need time off venetoclax, so an early marrow guides the schedule.
- Q48 · E** In ADMIRAL, gilteritinib gave median OS 9.3 vs 5.6 months. Genetics are repeated at relapse because clones evolve; the transplant team is involved at once.
- Q49 · C** Menin inhibitors are approved for NPM1-mutated AML and KMT2A-rearranged acute leukaemia. They cause differentiation syndrome and QT prolongation; Indian availability is to be confirmed.
- Q50 · B** Enasidenib targets IDH2 (CR 19%, median OS 9 months); ivosidenib or olutasidenib target IDH1.
- Q51 · B** Relapse may come from the dominant clone gaining mutations or from a minor clone that survived treatment. A FLT3, IDH or NPM1 result at relapse can open a targeted option.
- Q52 · E** Cranial nerve palsies need RT (for example 18–25 Gy to the brain, field down to C2), started as soon as CNS involvement is suspected, because recovery falls with delay. Cranial RT spares the marrow; avoid high-dose systemic chemotherapy around it.
- Q53 · D** Myeloid sarcoma is AML: systemic treatment is the main therapy. Add RT (about 20 Gy is generally enough) for compression of a vital structure or persistence after recovery from induction.
- Q54 · A** Do not change antibiotics for fever alone in a stable patient. Vancomycin is for line, skin or soft-tissue infection, pneumonia or instability.
- Q55 · B** Fluoroquinolone prophylaxis covers expected ANC <500/ μ L for >7 days; aciclovir for HSV-seropositive patients. In an Indian centre, fungal pneumonia was still diagnosed in 55% during induction.
- Q56 · A** Rasburicase is not given in G6PD deficiency (risk of haemolysis); check G6PD before using it. Allopurinol with fluids is the alternative.
- Q57 · D** In the first trimester, termination followed by treatment is advised. Later in pregnancy, urgent induction is discussed against temporary measures such as leukapheresis.
- Q58 · B** Some experts follow MRD in blood (NPM1, CBF fusions). A falling count or rising MRD needs a marrow, because NPM1 transcripts by PCR predict relapse.
- Q59 · C** Fit patients start induction at once. Leukapheresis has no proven benefit on its own and is used with hydroxyurea only for symptomatic leukostasis when induction cannot start.
- Q60 · B** Encourage activity and normal contact (sensible precautions while counts are low), follow the transplant team's vaccination schedule, and report fever, bleeding and new tiredness promptly.
- Q61 · D** 4-year OS was 51% vs 44%. Midostaurin is used for ITD or TKD mutations; quizartinib for ITD.
- Q62 · E** Quizartinib is added to induction and continued in consolidation and maintenance, like midostaurin.
- Q63 · C** More patients on CPX-351 reached transplant. TP53-mutated therapy-related AML responds poorly to all options; trials are preferred.
- Q64 · D** Venetoclax combinations are standard for unfit adults. In VIALE-C, venetoclax was added to low-dose cytarabine for those unsuitable for an HMA.
- Q65 · C** AGILE compared ivosidenib plus azacitidine with azacitidine alone in unfit adults with IDH1 mutations.
- Q66 · A** Gilteritinib is a bridge to allogeneic transplant, the only cure after relapse.
- Q67 · D** ATRA-arsenic gave fewer relapses and no induction deaths in low/intermediate-risk APL. AML17 tested ATRA-arsenic in all risk groups.
- Q68 · B** It is for patients aged 55 or more with intermediate- or adverse-risk AML in remission after consolidation.

- Q69 · E** MRD negativity roughly doubled 5-year survival across ages, subtypes and methods. NPM1 and CBF fusions are followed by PCR; other AML by multiparameter flow.
- Q70 · E** Favourable (52%): no FLT3-ITD, RAS or TP53 mutation. Intermediate (25%): FLT3-ITD or RAS. Adverse (25%): TP53 mutated.
- Q71 · A** It is acceptable for selected intermediate- or adverse-risk patients; most experts await longer follow-up.
- Q72 · C** Enasidenib, like other IDH inhibitors, can cause differentiation syndrome because it releases the differentiation block.
- Q73 · D** Ziftomenib (approved 13 November 2025) and revumenib are menin inhibitors; both cause differentiation syndrome and QT prolongation.
- Q74 · E** Benefit was seen in favourable and intermediate cytogenetics and was largest in core-binding-factor AML, which is nearly always CD33-positive.
- Q75 · C** 7+3 gives complete remission in about three-quarters of fit younger adults; anthracycline exposure matters.
- Q76 · D** Multidrug-resistant infections drive induction deaths in India; local resistance data should guide empirical antibiotics.
- Q77 · C** Median age was 33, 47% were high risk, and it took a median of 18 days from symptoms to diagnosis. Deaths were mostly from bleeding and infection.
- Q78 · C** The gap between trials and real life reflects delays in starting ATRA and in blood product support. ATRA on suspicion is the single most important step.
- Q79 · A** Fungal pneumonia was also diagnosed in 55% despite prophylaxis. Local resistance data should drive empirical choices.
- Q80 · D** About 50% under 40, 33% at 40–59, 13% at 60–69 and 3% at 70–79. Newer drugs and transplant in older adults have improved this since.
- Q81 · A** MRI and lumbar puncture are done only for neurological symptoms. Leptomeningeal AML without deficits is treated with intrathecal therapy; deficits need RT.
- Q82 · E** Most cases grow out of age-related clonal haematopoiesis (DNMT3A, TET2, ASXL1) that gathers further mutations.
- Q83 · B** IDH1/2 mutations (about 20%) make the oncometabolite 2-hydroxyglutarate. Ivosidenib, olutasidenib and enasidenib release the block, so blasts mature, as with ATRA in APL.
- Q84 · D** Routine steroid prophylaxis is not suggested. Mimics include sepsis, heart failure, pulmonary embolism, alveolar haemorrhage and drug reactions.
- Q85 · A** NPM1 transcripts by PCR predicted relapse. CBF fusions are also followed by PCR; other AML by multiparameter flow.
- Q86 · B** The choice follows age and comorbidity. Transplant in CR2 is myeloablative if fit.
- Q87 · A** No maintenance is needed after an ATO-containing regimen with molecular remission; without ATO, intermittent ATRA maintenance is used. Gemtuzumab treats molecular relapse where available.
- Q88 · E** Today ATRA plus arsenic is standard where both are available; where arsenic is not, ATRA with an anthracycline is used.
- Q89 · E** Leukapheresis is used with hydroxyurea only for symptomatic leukostasis when induction cannot start at once. Hyperleukocytosis occurs in 10–20% of new AML.
- Q90 · D** It is 5–10% in older adults. It matters when a sibling is the planned donor, so germline-type variants (DDX41, RUNX1, GATA2, biallelic CEBPA) need follow-up.