

Acute Lymphoblastic 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.

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drharitha.co.in/professionals/acute-lymphoblastic-leukaemia-mcq

Level 1 · Basics

Q1. Diagnosis In adults with acute lymphoblastic leukaemia (ALL), roughly how are B and T lineage cases divided?

- A About 50% each
- B About 75% B, 25% T
- C About 25% B, 75% T
- D Over 99% B
- E About 90% T

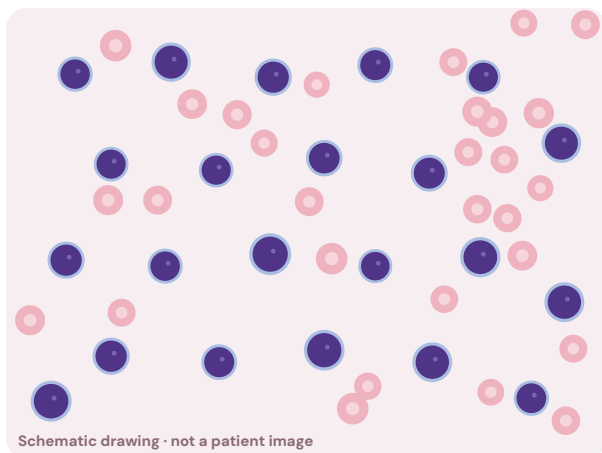
Q2. Diagnosis Which age pattern fits ALL?

- A Mainly people over 80, rarely younger
- B Equally common at every age of life
- C Only adults aged between 30 and 50
- D Only infants under one year of age
- E Mostly children; second peak after 60

Q3. Diagnosis A patient has a mediastinal mass of lymphoblasts and 8% marrow blasts. What is the diagnosis?

- A Acute lymphoblastic leukaemia
- B Classic Hodgkin lymphoma
- C Acute myeloid leukaemia
- D Thymic carcinoma
- E Lymphoblastic lymphoma

Q4. Diagnosis This schematic blood film shows many small-to-medium blasts with scant, granule-free cytoplasm. Which test decides whether they are lymphoid or myeloid?



- A Flow cytometry (immunophenotype)
- B Karyotype alone
- C Serum LDH
- D Antigen-receptor gene rearrangement
- E Morphology alone

Q5. Diagnosis Which pair of findings is typical of lymphoblasts?

- A Surface immunoglobulin and MYC rearrangement
- B TdT positive, myeloperoxidase negative
- C Auer rods and CD117
- D TdT negative, myeloperoxidase positive
- E Both TdT and myeloperoxidase positive

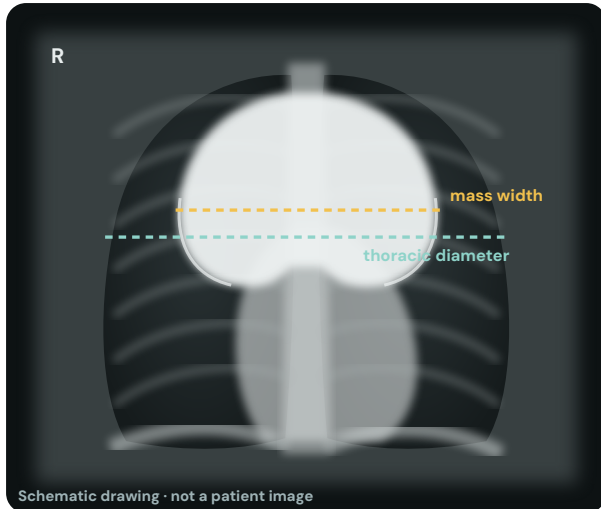
Q6. Diagnosis Which markers define B lineage in ALL?

- A CD13, CD33, CD117 and MPO
- B Cytoplasmic CD3 with CD7 and CD5
- C CD41 and CD61 on megakaryoblasts
- D CD19, cytoplasmic CD79a, CD22
- E CD138 and CD38 on plasma cells

Q7. Diagnosis Which markers define T lineage in ALL?

- A CD19 with CD10 and CD79a
- B Cytoplasmic or surface CD3 with CD7
- C CD13 with CD33 and CD117
- D CD34 with HLA-DR and nothing else
- E CD20 with CD5 and CD23

Q8. Diagnosis A 19-year-old man has cough, breathlessness when lying flat and a swollen face. This is his schematic chest X-ray. What is the key safety step?



- A Start a full course of anti-tuberculosis treatment
- B Bronchoscopy under general anaesthesia today
- C CT-guided biopsy with the patient lying flat
- D Assess airway, SVC and pericardium before sedation
- E Arrange mediastinoscopy before any imaging

Q9. Diagnosis A 7-year-old has a limp, joint pain and a low platelet count. What should you consider?

- A A sprain only
- B Growing pains only
- C Leukaemia rather than arthritis alone
- D Vitamin D deficiency only
- E Juvenile arthritis only, without further tests

Q10. Diagnosis Which constitutional condition is associated with a higher risk of ALL?

- A Cystic fibrosis
- B Down syndrome
- C Turner syndrome
- D Haemophilia A
- E Marfan syndrome

Q11. Diagnosis Why is BCR::ABL1 sent on the first day in a new adult with ALL?

- A It is needed before any blood transfusion
- B It confirms T lineage
- C It decides the CNS grade
- D It predicts allergy to asparaginase
- E It changes induction within days

Q12. Diagnosis About what proportion of adult ALL is Philadelphia-positive?

- A About 5%
- B 75%
- C 25–30%
- D All cases
- E Under 1%

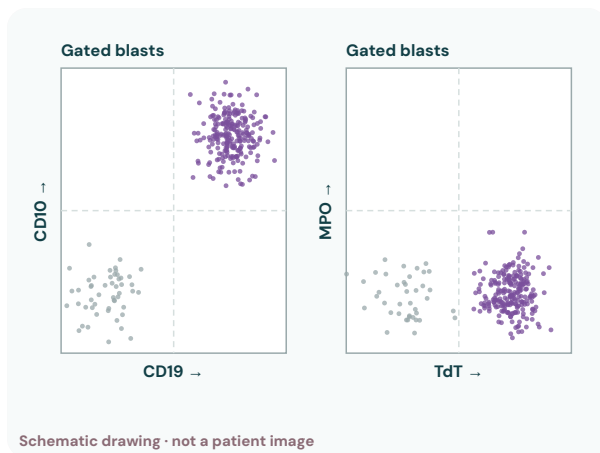
- Q13. Diagnosis** When is the diagnostic lumbar puncture usually done in ALL?
- A Before any blood test is done, on the day of admission
 - B At the end of maintenance, to confirm cure
 - C Only at relapse, never at the time of diagnosis
 - D Only if the patient develops a headache during treatment
 - E Once blood blasts have cleared, with the first intrathecal
- Q14. Diagnosis** How is CNS-3 disease defined?
- A Blasts with ≥ 5 WBC/ μL in the fluid, or a cranial nerve palsy
 - B A raised CSF protein alone, without any lymphoblasts
 - C Lymphoblasts with fewer than 5 WBC/ μL in the spinal fluid
 - D Any headache during induction, whatever the fluid shows
 - E No lymphoblasts at all in the spinal fluid sample
- Q15. Treatment** A 22-year-old man is about to start ALL treatment. Which is offered before treatment?
- A Fertility counselling and sperm banking
 - B Testosterone injections
 - C Nothing, as induction always causes sterility
 - D Orchidectomy
 - E Vasectomy
- Q16. Treatment** Which induction is preferred for a fit 25-year-old with Ph-negative ALL?
- A Imatinib with a steroid alone, without chemotherapy
 - B Venetoclax with azacitidine, as in older adults
 - C Low-dose cytarabine given under the skin
 - D Allogeneic transplant without any induction
 - E A paediatric-inspired, asparaginase-rich regimen
- Q17. Treatment** A 45-year-old has CD20-positive B-ALL (CD20 on 40% of blasts). What is added to induction?
- A Midostaurin
 - B Nelarabine
 - C Gemtuzumab
 - D Imatinib
 - E Rituximab
- Q18. Treatment** How long is a TKI given in Ph-positive ALL?
- A Only after an allogeneic transplant
 - B Only at relapse
 - C Only during induction
 - D From day 1, through every phase
 - E Only if the BCR::ABL1 level starts to rise
- Q19. Treatment** After induction, MRD is 10^{-3} (0.1%) in a fit 35-year-old with Ph-negative B-ALL. What next?
- A Maintenance tablets only
 - B Repeat induction with the same drugs only
 - C Blinatumomab, then allogeneic transplant
 - D Cranial radiotherapy alone
 - E Stop treatment and watch
- Q20. Treatment** What does blinatumomab target?
- A CD19 and CD3
 - B BCR::ABL1 kinase
 - C CD20 alone
 - D CD33 and CD3
 - E CD22 alone

- Q21. Treatment** Inotuzumab ozogamicin is an antibody–drug conjugate against which antigen?
- A CD19
 - B CD30
 - C CD20
 - D CD33
 - E CD22
- Q22. Treatment** How long does ALL maintenance usually last, and what does it contain?
- A ≥ 2 years: daily 6-MP, weekly methotrexate
 - B None: maintenance is not needed after consolidation
 - C One month of high-dose steroids, then nothing
 - D Six months of weekly injections in hospital
 - E Lifelong daily TKI tablets for every patient
- Q23. Treatment** Counts fall sharply on standard 6-mercaptopurine doses. Which test explains this?
- A G6PD activity
 - B TPMT and NUDT15
 - C HLA typing
 - D Antithrombin level
 - E BCR::ABL1 PCR
- Q24. Treatment** How is the tumour lysis risk of ALL classified?
- A High only in T-ALL
 - B Never low: high or intermediate
 - C Low unless the patient is over 60
 - D Unrelated to white count or LDH
 - E Always low
- Q25. Treatment** Which complication is especially linked with asparaginase?
- A Venous thrombosis
 - B Peripheral neuropathy with foot drop
 - C Pulmonary fibrosis on lung function
 - D Hearing loss at high frequencies
 - E Pleural effusion from capillary leak
- Q26. Treatment** Why are azole antifungals avoided around vincristine doses where possible?
- A They trigger tumour lysis during induction
 - B They make vincristine ineffective
 - C They worsen neuropathy and ileus
 - D They cause effusions
 - E They inactivate asparaginase in the blood
- Q27. Treatment** Testicular involvement has not resolved by the end of induction. What is done?
- A Cranial radiotherapy only
 - B Testicular biopsy every month
 - C Radiotherapy 24 Gy to both testes
 - D No further action
 - E Orchidectomy of the involved testis
- Q28. Survivorship** Which late effect is particularly linked with dexamethasone in adolescents?
- A Pulmonary fibrosis
 - B Osteonecrosis
 - C Cataract only in infants
 - D Hearing loss
 - E Kidney stones

- Q29. Survivorship** What is the advice on vaccines during ALL chemotherapy?
- A Vaccines are needed only 10 years after treatment
 - B Only the BCG vaccine, repeated each year
 - C No live vaccines; influenza vaccine for the family
 - D No vaccines at all for anyone in the household
 - E All live vaccines should be given at once to protect
- Q30. Survivorship** In Indian ALL series, how common are non-adherence and abandonment of treatment?
- A About 50%
 - B About 75%
 - C 11–15%
 - D 1%
 - E They do not occur

Level 2 · Applied & innovative

- Q31. Diagnosis** These schematic flow cytometry plots are from marrow blasts. What is the diagnosis?



- A T-lymphoblastic leukaemia
 - B Burkitt leukaemia
 - C B-lymphoblastic leukaemia
 - D Acute myeloid leukaemia
 - E Chronic lymphocytic leukaemia
- Q32. Diagnosis** Blasts are CD19+, cytoplasmic CD79a+, MPO- and also express CD13 and CD33. What is the diagnosis?
- A Mixed-phenotype leukaemia by definition
 - B B-ALL
 - C AML, because CD13 and CD33 are present
 - D CML in chronic phase
 - E T-ALL with aberrant B markers
- Q33. Diagnosis** Leukaemic cells are TdT-negative, carry surface immunoglobulin and have a MYC rearrangement. What is this?
- A Mixed-phenotype leukaemia
 - B Burkitt lymphoma/leukaemia
 - C Early T-cell precursor ALL
 - D Ph-like ALL
 - E Pro-B ALL
- Q34. Diagnosis** A mediastinal mass contains many immature T cells. Which finding favours a type B1 thymoma over T-LBL?
- A A raised LDH with high uric acid
 - B A monoclonal T-cell receptor gene rearrangement
 - C Keratin-positive epithelium, polyclonal thymocytes
 - D A marrow aspirate with 25% lymphoblasts
 - E TdT-positive cells growing in confluent sheets

- Q35. Diagnosis** The diagnostic lumbar puncture is traumatic and blood contains blasts. When is it called CNS-3?
- A Never: a traumatic tap cannot ever be graded
 - B Only if a cranial nerve palsy develops later on
 - C When the CSF glucose is lower than the blood glucose
 - D CSF WBC:RBC ratio $\geq 2\times$ the blood ratio
 - E Whenever any blast is seen
- Q36. Diagnosis** How should the marrow sample for MRD be taken at count recovery?
- A Aspirates pooled from both iliac crests
 - B Peripheral blood only, in every B-ALL
 - C A trephine in formalin, without aspirate
 - D The last 20 mL of the aspirate pull
 - E The first 2–5 mL of the aspirate
- Q37. Diagnosis** Which MRD method can detect one leukaemic cell among a million?
- A Flow cytometry with 6–8 colours
 - B Karyotype
 - C NGS of Ig/TCR rearrangements
 - D Routine microscopy
 - E Expert microscopy
- Q38. Diagnosis** Why should the first diagnostic sample be kept?
- A PCR MRD needs the clone's own Ig/TCR markers
 - B It is needed to repeat the karyotype every month
 - C It confirms the HLA type for the donor search
 - D It is used to grow a personal cancer vaccine
 - E Laboratories must legally keep it for 50 years
- Q39. Diagnosis** Which statement about Ph-like ALL is correct?
- A It is a favourable subtype with excellent results
 - B It always carries the BCR::ABL1 fusion gene
 - C It occurs only in young children, never in adults
 - D BCR::ABL1-like expression without the fusion
 - E It is a subtype of T-ALL defined by NOTCH1
- Q40. Diagnosis** Which feature makes Ph-negative B-ALL high risk under the modified Hoelzer criteria?
- A White count 45,000/ μ L
 - B CD10-positive (common) phenotype
 - C White count 20,000/ μ L
 - D Age 30
 - E Remission reached by week 3
- Q41. Treatment** In E1910, what did adding blinatumomab to consolidation do for adults in MRD-negative remission?
- A Replaced the need for maintenance
 - B Lowered survival because of neurotoxicity
 - C Made no difference
 - D Raised 3-year survival from 68% to 85%
 - E Helped only Ph-positive patients
- Q42. Treatment** A 68-year-old with Ph-positive ALL is not fit for intensive chemotherapy. Which approach fits?
- A A TKI with a steroid or gentle chemotherapy
 - B Hyper-CVAD without a TKI, at reduced intensity
 - C Rituximab alone, given weekly for a month
 - D Supportive care only, as cure is not possible
 - E Allogeneic transplant at once, before any TKI

- Q43. Treatment** In Ph-positive ALL on dasatinib, BCR::ABL1 MRD starts to rise. What is done?
- A Stop all TKIs and watch the counts closely
 - B Add rituximab to the dasatinib and continue
 - C Continue the same TKI
 - D Give cranial radiotherapy as the next step
 - E Mutation test and a TKI switch
- Q44. Treatment** A patient with Ph-positive ALL has a history of pleural effusions and heart failure. Which TKI is best avoided?
- A Ponatinib
 - B Imatinib
 - C None, all are equivalent
 - D Dasatinib
 - E Any TKI must be avoided
- Q45. Treatment** After allogeneic transplant for Ph-positive ALL, how long is the TKI continued?
- A Only if GVHD develops
 - B At least 1 year
 - C It is never used after transplant
 - D Only 1 month
 - E It is stopped on the day of transplant
- Q46. Treatment** A fit 40-year-old has first relapse of Ph-negative B-ALL; blasts are CD19+ and CD22+. Which first salvage became standard?
- A Blinatumomab or inotuzumab, then transplant
 - B A TKI alone, as in Ph-positive disease
 - C The original induction regimen repeated at once
 - D Palliative care only, as cure is not possible
 - E Nelarabine-based chemotherapy as first salvage
- Q47. Treatment** Why is inotuzumab given, if possible, not immediately before an allogeneic transplant?
- A It causes severe graft-versus-host disease
 - B It causes severe neuropathy after transplant
 - C It destroys the donor T cells in the graft
 - D Higher risk of liver veno-occlusive disease
 - E It prevents the donor cells from engrafting
- Q48. Treatment** B-ALL relapses after CD19 CAR-T therapy. Which test is essential before choosing the next treatment?
- A TPMT and NUDT15 testing before any treatment
 - B A serum IgG level to plan immunoglobulin
 - C A repeat lumbar puncture
 - D A chest X-ray to look for a mediastinal mass
 - E Recheck CD19, CD22 and CD20
- Q49. Treatment** Relapsed ALL with a KMT2A rearrangement can be treated with which drug?
- A Imatinib
 - B ATRA
 - C Revumenib
 - D Ivosidenib
 - E Rituximab
- Q50. Treatment** T-ALL relapses in a 30-year-old. Which approach fits?
- A Blinatumomab, then transplant if in remission
 - B Nelarabine-based chemotherapy, then transplant
 - C A TKI alone, continued for at least two years
 - D Inotuzumab, then transplant if in remission
 - E Rituximab with chemotherapy, then maintenance

- Q51. Treatment** Which approach is suggested for an older, unfit adult with T-ALL?
- A Blinatumomab alone
 - B Mini-hyper-CVD with venetoclax
 - C Rituximab-based chemotherapy
 - D Inotuzumab with dexamethasone
 - E Supportive care is the only option
- Q52. Treatment** Which preparation is needed before high-dose methotrexate?
- A Withhold leucovorin until day 7, then start it
 - B Restrict fluids to protect the kidneys from overload
 - C Give a PPI and an NSAID for comfort during it
 - D Leave effusions in place and drain them afterwards
 - E Hydrate, alkalinise, drain effusions, stop PPIs
- Q53. Treatment** What does ISTH guidance suggest during asparaginase treatment in adults?
- A Stop asparaginase in every adult, whatever the risk
 - B No measures, because clots never occur with asparaginase
 - C Platelet transfusions to keep the count above 200,000/ μ L
 - D LMW heparin if platelets $\geq 30,000/\mu$ L; replace low antithrombin
 - E Full-dose warfarin for everyone from the first day of induction
- Q54. Treatment** A cerebral sinus thrombosis develops during asparaginase treatment. What is done?
- A Hold asparaginase; LMW heparin; consider resuming later
 - B Stop all ALL treatment permanently and observe
 - C Continue asparaginase unchanged and add daily aspirin
 - D Give fresh frozen plasma only, with no anticoagulant
 - E Start thrombolysis in every case, whatever the bleeding risk
- Q55. Treatment** In tumour lysis prevention during steroid-based ALL induction, which fluid is favoured?
- A No fluids, to avoid overload
 - B Hypotonic dextrose-saline
 - C Hypertonic saline
 - D Normal saline only, in large volumes
 - E Potassium-rich fluids
- Q56. Treatment** Where should the diagnosis come from in a patient with a large mediastinal mass and a pleural effusion?
- A Blood, marrow or pleural fluid
 - B Open mediastinal biopsy under general anaesthesia
 - C Mediastinoscopy before any imaging is done
 - D A core biopsy, supine and sedated
 - E Thoracotomy
- Q57. Treatment** A 55-year-old with Ph-negative B-ALL is fit. Which induction options fit current guidance for 40–65 years?
- A Imatinib alone, continued for at least two years
 - B Venetoclax with azacitidine, as in older AML
 - C Watchful waiting until symptoms or cytopenias worsen
 - D An asparaginase regimen or hyper-CVAD ($\pm$ rituximab)
 - E Low-dose cytarabine given under the skin monthly
- Q58. Treatment** In GRAALL, which patients benefited from allogeneic transplant in first remission?
- A Only those with MRD below 10^{-5} after induction
 - B MRD $\geq 10^{-3}$ (HR 0.43), not those below
 - C Everyone in first remission, whatever the MRD
 - D Only patients over 70 with high-risk genetics
 - E Nobody: transplant gave no benefit to any group

Q59. Survivorship In remission, a single follow-up MRD test is low-level positive. What is correct?

- A It means the laboratory test must be faulty
- B MRD results are never useful once induction is over
- C Not relapse by itself; act if persistent or rising
- D It needs an immediate transplant without any repeat
- E It always means relapse: start salvage therapy today

Q60. Survivorship After ALL treatment ends, how often are CBC and liver tests done in the first year?

- A Every 5 years
- B Every 1–2 months
- C Every week
- D Only if symptoms appear
- E Once a year

Level 3 · Challenging

Q61. Treatment In CALGB 10403 (295 patients aged 17–39 on a paediatric regimen), what was 3-year survival?

- A 50%
- B 30%
- C 15%
- D 73%
- E 95%

Q62. Treatment Across 23 observational studies of adolescents and young adults, how did paediatric-type regimens compare?

- A No difference in survival or treatment deaths
- B Worse survival, more deaths
- C Better survival only in T-ALL, not in B-ALL
- D Better survival only after allogeneic transplant
- E Better 5-year survival (HR 1.40)

Q63. Treatment In GRAALL-2005 (an asparaginase regimen), what were CR and 5-year survival?

- A CR 70%, 5-year survival 30%
- B CR 92%, 5-year survival 92%
- C CR 92%, 5-year survival 59%
- D CR 99%, 5-year survival 90%
- E CR 50%, 5-year survival 20%

Q64. Treatment In GRAALL-2005/R, what did rituximab add for CD20+ B-ALL?

- A 4-year EFS 85% vs 43%, with more deaths
- B Benefit only in T-ALL, not in B-ALL
- C No benefit in event-free survival
- D 4-year EFS 55% vs 43%
- E Worse survival from infections and toxicity

Q65. Treatment In E1910 (224 adults aged 30–70 in MRD-negative CR), what was the hazard ratio for death with blinatumomab added?

- A 2.00
- B 0.10
- C 0.41
- D 0.95
- E 1.41

Q66. Treatment In BLAST (113 MRD-positive patients), what did blinatumomab achieve?

- A 78% became MRD-negative
- B All patients were cured without any transplant
- C 10% became MRD-negative
- D Responders survived for a shorter time than others
- E MRD levels were unchanged in most patients

- Q67. Treatment** In UKALL XII/ECOG 2993, what did sibling-donor transplant do in standard-risk ALL?
- A No survival change and no extra non-relapse deaths
 - B Fewer non-relapse deaths and fewer relapses as well
 - C Worse survival because of more relapses after transplant
 - D 5-year survival 62% vs 52%; more non-relapse deaths
 - E 5-year survival 90% vs 20%, with no extra deaths
- Q68. Treatment** In a CIBMTR comparison (Seftel 2016), how did paediatric-inspired chemotherapy compare with matched allografts?
- A No difference
 - B 4-year survival 73% vs 45%
 - C Transplant was always better
 - D 4-year survival 20% vs 10%
 - E 4-year survival 45% vs 73%
- Q69. Treatment** In patients over 55 treated with inotuzumab plus dexamethasone (Stelljes 2024), which results were reported?
- A CR in 10%, 3-year survival only 5%
 - B No responses at all in this age group
 - C CR in 85%, 3-year survival 56%, VOD 8%
 - D CR in 50%, 3-year survival only 20%
 - E CR in all 43, 3-year survival 73%
- Q70. Treatment** In COG AALL0434 (children and young adults up to 31), what did nelarabine add for T-ALL?
- A No benefit in DFS or CNS relapse
 - B More CNS relapses with nelarabine added
 - C Better survival in adults over 60 only
 - D DFS 50% vs 30%; CNS relapse unchanged
 - E DFS 88% vs 82%; CNS relapse 1% vs 7%
- Q71. Treatment** In UKALL XII/ECOG 2993, what were CR and 5-year survival in 356 adults with T-ALL, and survival after relapse?
- A CR 94%, 5-year 48%; after relapse 6.5%
 - B CR 50%, 5-year 10%; after relapse 50%
 - C CR 20%, 5-year 5%
 - D CR 99%, 5-year 90%; after relapse 80%
 - E CR 94%, 5-year 94%
- Q72. Treatment** What did a low-cost protocol at CMC Vellore achieve in 113 adults with T-ALL (Abraham 2014)?
- A No long-term survivors
 - B 25% 1-year survival
 - C 51% 5-year survival
 - D 90% 5-year survival
 - E 10% 5-year survival
- Q73. Treatment** In GRAAPH-2005 (268 adults with Ph+ ALL), how did imatinib with vincristine-dexamethasone compare with imatinib plus hyper-CVAD?
- A Better 5-year survival by about 30% overall
 - B More early deaths but better long-term survival
 - C No CRs at all without intensive hyper-CVAD
 - D More CRs and fewer early deaths; equal survival
 - E Fewer CRs and worse 5-year survival overall
- Q74. Treatment** In D-ALBA, what happened after chemotherapy-free dasatinib plus steroid induction when blinatumomab was given?
- A MRD negativity fell after blinatumomab
 - B No patient reached complete remission
 - C Blinatumomab caused the T315I mutation
 - D All patients relapsed within a year
 - E MRD negativity rose (29% → 60%)

- Q75. Treatment** In PhALLCON, how did ponatinib compare with imatinib in Ph-positive ALL?
- A No difference
 - B Only effective without chemotherapy
 - C More MRD-negative CRs (34% vs 17%)
 - D Fewer MRD-negative CRs
 - E Worse survival from infection
- Q76. Treatment** In a retrospective study of 230 MRD-negative Ph+ ALL patients (Ghobadi 2022), how did transplant compare with continued consolidation?
- A Transplant halved survival
 - B Data were not reported
 - C Consolidation was harmful
 - D No survival difference
 - E Transplant doubled survival
- Q77. Treatment** In TOWER (405 adults with relapsed Ph-negative ALL), what was median survival with blinatumomab vs chemotherapy?
- A 4.0 vs 7.7 months
 - B 12 vs 12 months
 - C 7.7 vs 4.0 months
 - D 39 vs 13 months
 - E 24 vs 8 months
- Q78. Treatment** In INO-VATE, which pair of results is correct for inotuzumab vs chemotherapy?
- A CR 50% vs 50%; no VOD
 - B CR 29% vs 81%; VOD 1% vs 9%
 - C CR 81% vs 29%; VOD 9% vs 1%
 - D CR 98% vs 91%; VOD 0%
 - E CR 36% vs 20%; VOD 40%
- Q79. Treatment** In AUGMENT-101 (57 patients with KMT2A-rearranged leukaemia), what did revumenib achieve?
- A Response 63%, CR 23%
 - B No differentiation syndrome in any patient
 - C CR 81%, with MRD negativity in 78%
 - D Response 100%, with CR in every patient
 - E Response 10%, no CRs
- Q80. Treatment** In 118 adults with relapsed T-ALL treated with nelarabine-based chemotherapy (Candoni 2020), what were the results?
- A CR 81%; 2-year survival 23%
 - B CR 5%; no survivors at 1 year
 - C CR 63%; CR 23% with no relapses
 - D CR 36%; 1-year survival 37%
 - E CR 90%; 1-year survival 85%
- Q81. Treatment** Which statement about CD19 CAR-T products for ALL is correct?
- A Obecabtagene: much less severe CRS and ICANS
 - B Brexucabtagene is approved only for young children
 - C CAR-T products for ALL target CD22 alone
 - D Tisagenlecleucel is approved only for adults over 65
 - E NexCAR19 is approved only in the United States
- Q82. Diagnosis** In the Berry 2017 meta-analysis of 39 studies, what was the hazard ratio for survival with MRD negativity in adults?
- A 1.28
 - B 0.05
 - C 2.80
 - D 0.95
 - E 0.28

Q83. Diagnosis In one series of 148 adults, what was 5-year survival with Ph-like ALL compared with other Ph-negative B-ALL?

- A 59% vs 23%
- B 23% vs 59%
- C 80% vs 85%
- D 10% vs 12%
- E 50% vs 50%

Q84. Diagnosis Which statement about adult T-ALL biology is correct?

- A ETP-ALL is the commonest type of adult T-ALL
- B CDKN2A deletion is never found in T-ALL
- C TP53 mutation predicts an excellent response
- D NOTCH1/FBXW7 without RAS/PTEN: longer survival
- E T-ALL is driven mainly by the BCR::ABL1 fusion

Q85. Diagnosis In 1,508 adults with ALL, how common was CNS disease at diagnosis, and how did it affect outcome?

- A 5%; 90% reached remission; survival similar
- B 25%; survival was halved by CNS disease
- C 0%; CNS disease does not occur in adults
- D 5%; none of them survived beyond a year
- E 50%; most did not reach remission at all

Q86. Treatment How common is asparaginase-related thrombosis in adults, and what is its link to central lines?

- A Under 1%, and never related to central lines
- B About 14%; about half catheter-related
- C About 14%, and never related to central lines
- D It occurs only in children, not in adults
- E About 50%, and nearly all of them arterial

Q87. Treatment For CNS relapse of ALL, which radiotherapy is described?

- A Cranial RT 24 Gy with ≥ 6 intrathecal treatments
- B No RT; intrathecal therapy only, twice a week
- C Whole-body RT 2 Gy, then maintenance tablets
- D Craniospinal RT 54 Gy, with no intrathecal therapy
- E Cranial RT 12 Gy alone, with no intrathecal therapy

Q88. Survivorship In one Indian series, how did treatment delays affect relapse-free survival in ALL?

- A It fell from 95% to 90%
- B It rose from 43% to 69%
- C There was no effect
- D It fell to 0%
- E It fell from 69% to 43%

Q89. Survivorship At KGMU Lucknow (Shukla 2025), what proportion of 75 ALL patients on BFM-95 did not complete induction?

- A 75.0%
- B 50.0%
- C 0%
- D 1.0%
- E 10.6%

Q90. Treatment Which statement about older adult regimens for Ph-negative ALL before paediatric-inspired protocols is correct?

- A They gave 90% survival in young adults
- B They are still preferred for patients under 40
- C They were never used in adults with ALL
- D 5-year survival near 30% in young adults
- E They avoided asparaginase toxicity and were better

Answer key

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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 B-ALL is about three-quarters and T-ALL about a quarter of adult ALL. T-ALL peaks in adolescents and young adults, with a 2:1 male excess.

Q2 · E Three-quarters of B-ALL occurs before age 6; there is a second peak after 60. In children it is the commonest cancer, and more than 90% are cured.

Q3 · E Leukaemia and lymphoblastic lymphoma are the same disease: $\geq 20\%$ marrow blasts = ALL (with or without a mass); a mass with $< 20\%$ marrow blasts = LBL. It is treated on the ALL protocol of that lineage.

Q4 · A Immunophenotype decides lineage. Antigen-receptor gene rearrangements are not lineage-specific, and morphology cannot reliably separate lymphoblasts from myeloblasts.

Q5 · B Lymphoblasts are TdT-positive and MPO-negative. Myeloperoxidase or Auer rods point to AML; surface immunoglobulin with MYC rearrangement (TdT-negative) points to Burkitt.

Q6 · D B lineage: CD19, cytoplasmic CD79a and CD22. T lineage: cytoplasmic or surface CD3 with CD7. CD20 is present on more than 10–20% of blasts in about 40% of B-ALL.

Q7 · B T-ALL is defined by CD3 (cytoplasmic or surface) with CD7. It more often has a mediastinal mass, a high white count, CNS and testicular involvement.

Q8 · D An anterior mediastinal mass (T-ALL/T-LBL in 50–75% of T-ALL) can obstruct the airway, SVC or cause tamponade. Image first, and take the diagnosis from the least invasive source: blood, marrow or pleural fluid.

Q9 · C Bone pain, limp or joint pain with odd counts may be leukaemia. Bone pain and large nodes point to ALL rather than AML.

Q10 · B Associations include ionising radiation, Down syndrome and other constitutional disorders, rare familial variants (PAX5, ETV6, TP53) and common inherited polymorphisms.

Q11 · E Ph-positive ALL needs a TKI from day 1 to the end of treatment, so BCR::ABL1 (RT-PCR or FISH) must be known within days.

Q12 · C Ph+ is 25–30% of adults (about half in older adults) and about 5% of children. Ph-like ALL adds up to one-third of adult B-ALL.

Q13 · E Every patient needs a lumbar puncture, generally once blood blasts have cleared, with platelets and clotting corrected. Neurological signs need urgent imaging and LP.

Q14 · A CNS-1: no blasts; CNS-2: blasts with < 5 WBC/ μ L; CNS-3: blasts with ≥ 5 WBC/ μ L or a cranial nerve palsy. CNS-3 needs more frequent intrathecal therapy until the fluid clears.

Q15 · A Fertility counselling and sperm banking are offered; induction does not usually cause sterility in younger patients. Echo, ECG, viral serology, pregnancy test and HLA typing are also done.

Q16 · E Up to about 40, paediatric-inspired regimens cure 60–80%. Older adult-type regimens gave 5-year survival near 30%.

Q17 · E Adding at least 8 doses of rituximab for CD20+ B-ALL in patients under 65 improved 4-year EFS (55% vs 43%) in GRAALL-2005/R.

Q18 · D A TKI is essential but not enough on its own: it is paired with a steroid or chemotherapy and continued through every phase, and for at least a year after transplant.

Q19 · C MRD $\geq 10^{-4}$ after induction is high risk: one or two cycles of blinatumomab (78% became MRD-negative in BLAST) and then allogeneic HCT if eligible.

- Q20 · A** Blinatumomab is a bispecific T-cell engager (CD19×CD3). It can cause cytokine release syndrome and neurotoxicity.
- Q21 · E** CD22 is present on more than 90% of B-ALL. Inotuzumab carries a risk of hepatic veno-occlusive disease, especially just before a transplant.
- Q22 · A** Maintenance is low-intensity treatment at home for at least 2 years, often with vincristine–steroid pulses. Outcomes are better when counts are kept suppressed.
- Q23 · B** Variants in TPMT or NUDT15 reduce thiopurine breakdown and cause marked myelosuppression; the dose is adjusted (CPIC 2019).
- Q24 · B** ALL with WBC $\geq 100,000/\mu\text{L}$ or LDH $\geq 2\times$ the upper limit is high risk (fluids plus rasburicase); all other ALL is intermediate risk (fluids plus allopurinol).
- Q25 · A** Asparaginase lowers antithrombin, protein C/S and fibrinogen: thrombosis occurs in 14% of adults. It also causes allergy, pancreatitis and liver problems.
- Q26 · C** Azoles raise vincristine levels, causing severe neuropathy and ileus. Some Indian centres stop voriconazole for 24 hours before each dose.
- Q27 · C** RT 24 Gy to both testes is given, usually with the first maintenance cycle. The testes are examined at diagnosis in every male.
- Q28 · B** Other late effects: neuropathy (vincristine), cognitive and endocrine effects (especially after cranial RT), cardiomyopathy (anthracyclines), infertility, obesity, abnormal glucose metabolism and second cancers.
- Q29 · C** Pneumocystis prophylaxis is given during therapy; after transplant, revaccination follows the transplant protocol.
- Q30 · C** Maintenance runs for 2–3 years at home, so adherence is treatment. Social workers, counsellors and a written plan help.
- Q31 · C** The blasts are CD19+ CD10+ and TdT+ MPO–: B-lymphoblasts. Burkitt cells are TdT-negative with surface immunoglobulin; AML blasts are MPO-positive; T-ALL lacks CD19.
- Q32 · B** Myeloid markers (CD13, CD33) do not exclude B-ALL; myeloperoxidase does. Mixed-phenotype leukaemia and CML in lymphoid blast phase are differentials.
- Q33 · B** Burkitt cells are mature B cells and are treated differently (see the aggressive B-cell module), not on an ALL protocol.
- Q34 · C** Thymomas contain keratin-positive epithelium and polyclonal thymocytes of all stages. Indolent T-lymphoblastic proliferation is also TdT-positive but non-clonal.
- Q35 · D** This rule separates true CNS disease from contamination. CNS-3 needs more frequent intrathecal therapy until the fluid clears.
- Q36 · E** Later pulls are diluted with blood, which can turn a positive MRD falsely negative. In B-ALL blood is 1–3 logs less sensitive than marrow.
- Q37 · C** Microscopy sees about 1 in 20, expert microscopy 1 in 100, flow 1 in 10,000, RQ-PCR 1 in 100,000 and NGS 1 in 1,000,000. "MRD-negative" needs an assay that reaches 10^{-4} .
- Q38 · A** RQ-PCR for Ig/TCR rearrangements is designed on the patient's own leukaemia clone at diagnosis.
- Q39 · D** Most cases carry kinase-activating changes (ABL-class, JAK2, CRLF2). It is up to one-third of adult B-ALL, high risk, and commoner in people of Hispanic ancestry; trials of ABL/JAK inhibitors are under way.
- Q40 · A** High risk (any one): age over 60, WBC $> 30,000/\mu\text{L}$ (B) or $> 100,000/\mu\text{L}$ (T), t(4;11) or t(1;19), Ph-like signature, pro-B (CD10-negative) blasts, more than 4 weeks to remission, MRD $\geq 10^{-4}$ after induction.
- Q41 · D** Grade ≥ 3 neuropsychiatric events were 23% vs 5%. UpToDate applies this to all patients in remission, whatever the MRD.
- Q42 · A** In older patients, dasatinib with low-intensity chemotherapy gave CR 96% and 5-year survival 36%. Chemotherapy-free dasatinib with a steroid, then blinatumomab, is also possible.
- Q43 · E** Relapses on dasatinib often carry T315I, which ponatinib covers. Falling MRD in a fit patient leads to transplant; rising MRD needs a mutation test and a switch.

- Q44 · D** Dasatinib is favoured for its CNS penetration but avoided with effusions, bleeding or heart failure. Imatinib is generic and cheaper but slower; ponatinib covers T315I at the cost of arterial events.
- Q45 · B** Without transplant, a TKI plus low-intensity maintenance is given for 24–30 months.
- Q46 · A** TOWER and INO-VATE made antibodies the standard first salvage for B-ALL. Allogeneic HCT follows for the fit, best when MRD-negative.
- Q47 · D** In INO-VATE, VOD occurred in 9% vs 1%, and in 10 of 48 transplanted patients. Plan the sequence with the transplant team; ursodeoxycholic acid prophylaxis and defibrotide for severe VOD.
- Q48 · E** Antigen-negative relapses follow antibody and CAR-T therapy. KMT2A-rearranged relapse → revumenib; others → chemotherapy by fitness.
- Q49 · C** In AUGMENT-101, revumenib gave responses in 63% (CR 23%) of 57 patients, with differentiation syndrome in 16%.
- Q50 · B** Inotuzumab and blinatumomab target B-cell antigens (CD22, CD19), so they do not work in T-ALL. After relapse, survival in adult T-ALL is poor without transplant.
- Q51 · B** This is expert opinion; inotuzumab does not work without CD22. For frail patients, vincristine with prednisone or POMP can control the disease for a time.
- Q52 · E** Effusions act as a reservoir and delay clearance. Levels are monitored, leucovorin rescue given, and glucarpidase used within 48–60 hours when levels stay high with kidney injury.
- Q53 · D** Thrombosis occurs in 14% of adults, about half catheter-related. The evidence for prophylaxis is retrospective and mixed.
- Q54 · A** VTE is treated with LMW heparin (anti-Xa monitoring) for at least 6 months; asparaginase is held for cerebral sinus thrombosis, large PE, proximal DVT or arterial clots.
- Q55 · B** Steroids retain sodium, so hypotonic dextrose-saline is favoured. High-risk patients also get rasburicase (not in G6PD deficiency).
- Q56 · A** Biopsy a node or mediastinal mass only when blood, marrow or an effusion cannot give the diagnosis.
- Q57 · D** Asparaginase regimens bring liver, pancreas and clotting problems; hyper-CVAD more neutropenia and admissions.
- Q58 · B** Dhédin 2015. Many also favour HCT for t(4;11), complex karyotype, low hypodiploidy/near-triploidy, TP53 or WBC >70,000/μL.
- Q59 · C** MRD levels fluctuate; reappearance should be confirmed on a second sample. Persistent or rising MRD triggers a donor search and closer monitoring.
- Q60 · B** Then every 3–6 months in the second year and every 6–12 months after that. A final marrow with MRD is done at the end of maintenance.
- Q61 · D** Treatment deaths were 3%; a quarter had raised liver enzymes and 5% thromboses. It showed that adult oncologists can safely deliver a paediatric regimen.
- Q62 · E** Indian AYA series report 40–60% survival on BFM-type protocols.
- Q63 · C** Hyper-CVAD with rituximab gave CR 95% and 3-year survival 50%.
- Q64 · D** At least 8 doses were given to patients under 65. In UKALL14, four doses without CD20 selection did not help.
- Q65 · C** 3-year survival 85% vs 68%; 57% had high-risk features. Grade ≥3 neuropsychiatric events were 23% vs 5%.
- Q66 · A** Blinatumomab is followed by allogeneic HCT in eligible patients.
- Q67 · D** In high-risk disease, fewer relapses (37% vs 63%) were offset by non-relapse mortality (36% vs 14%). A meta-analysis confined the survival gain to patients under 35.
- Q68 · B** This and the meta-analysis explain why transplant in first remission is now guided by MRD and genetic risk.
- Q69 · E** Mini-hyper-CVD plus inotuzumab (median age 68) gave CR 85%, 3-year survival 56%, liver VOD 8% and treatment deaths 12% (Kantarjian 2018).
- Q70 · E** Nelarabine has not improved outcomes in adults; adding it to hyper-CVAD gave no benefit over historical controls.
- Q71 · A** Relapse is the main problem in adult T-ALL, so first-line treatment must be complete.

Q72 · C Indian evidence also includes INWARD AYA data, Tata Memorial AYA on modified BFM-90 and KGMU Lucknow on BFM-95.

Q73 · D Ph+ ALL needs a TKI more than intensive chemotherapy.

Q74 · E Dasatinib plus a steroid gave CR in 98% (LAL1205). Relapses on dasatinib often carry T315I.

Q75 · C With hyper-CVAD, ponatinib gave 3-year EFS 70% (Jabbour 2018), at a cost of arterial events.

Q76 · D Falling MRD and fitness favour transplant; MRD-negative patients may finish consolidation or be transplanted.

Q77 · C Quality of life was better; grade ≥ 3 CRS occurred in 5%.

Q78 · C MRD negativity was 78% vs 28% and 2-year survival 23% vs 10%; 10 of 48 transplanted patients developed VOD.

Q79 · A Menin inhibitors are also approved for NPM1-mutated AML (see the AML module).

Q80 · D Transplant after remission offers the best chance in relapsed T-ALL.

Q81 · A Tisagenlecleucel is used up to 25 years (ELIANA), brexucabtagene in adults (ZUMA-3). NexCAR19 received CDSCO approval on 13 October 2023 for relapsed B-cell lymphoma and leukaemia. Trials differ, so products should not be ranked.

Q82 · E In GMALL 07/2003, 5-year survival was 80% with MRD negativity by week 16 vs 42% without (Gökbuget 2012).

Q83 · B Ph-like ALL is 10–13% of childhood, about 21% of adolescent and up to one-third of adult B-ALL. Targeted drugs are being tested in trials.

Q84 · D About half of adults have this favourable profile (Trinquand 2013). ETP-ALL (5–10%) depends on BCL-2, which is why venetoclax is studied; TP53 mutation (10–15%) predicts resistance.

Q85 · A Without CNS-directed therapy more than half develop CNS leukaemia; with prophylaxis fewer than 20%.

Q86 · B Mort 2025: catheters carry about half of asparaginase thromboses. Two- or three-lumen lines are placed at the start of treatment.

Q87 · A For CNS-3 at diagnosis, cranial RT (typically 18 Gy) can be added in later phases. Some Indian centres still give prophylactic cranial RT (12 Gy) to adults on BFM-type protocols, accepting the late effects.

Q88 · E Abandonment (11–15%) and interruptions are major causes of failure; a written plan for the 2–3 years of treatment and social support help (Ganesan 2021).

Q89 · E Abandonment, resistant infections, cost and limited transplant access shape every decision in Indian ALL care (Ganesan 2021); plan support at diagnosis.

Q90 · D Paediatric-inspired, asparaginase-rich regimens now cure 60–80% up to about 40 years (DFCI: CR 85%).