

Chronic 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.

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drharitha.co.in/professionals/chronic-myeloid-leukaemia-mcq

Level 1 · Basics

Q1. Diagnosis Which genetic change defines chronic myeloid leukaemia (CML)?

- A JAK2 V617F point mutation
- B t(9;22) making BCR::ABL1
- C t(15;17) making PML::RARA
- D t(8;21) making RUNX1::RUNX1T1
- E t(14;18) joining IGH to BCL2

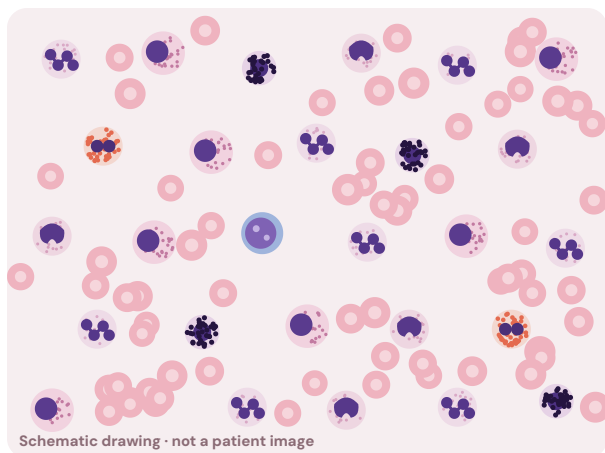
Q2. Diagnosis What does the BCR::ABL1 fusion gene produce?

- A A transcription factor that blocks maturation
- B A growth factor receptor without a kinase
- C An anti-apoptotic protein of the BCL2 family
- D A truncated tumour suppressor protein
- E A tyrosine kinase that is always switched on

Q3. Diagnosis Which is the only established risk factor for CML?

- A Benzene exposure
- B Tobacco smoking
- C A parent with CML
- D Epstein-Barr virus infection
- E Ionising radiation

Q4. Diagnosis This schematic blood film is from a 45-year-old with a white count of 150,000/ μ L and a large spleen. Which description fits it best?



- A Granulocytes at every stage, with basophils
- B Lymphocytes with hair-like cytoplasmic projections
- C Neutrophils with toxic granules and Döhle bodies
- D Small mature lymphocytes and smudge cells
- E Uniform blasts containing Auer rods

Q5. Diagnosis About what proportion of people with CML have no symptoms when it is found?

- A Nearly all
- B Up to half
- C About 9 in 10
- D Fewer than 1 in 20
- E About 1 in 10

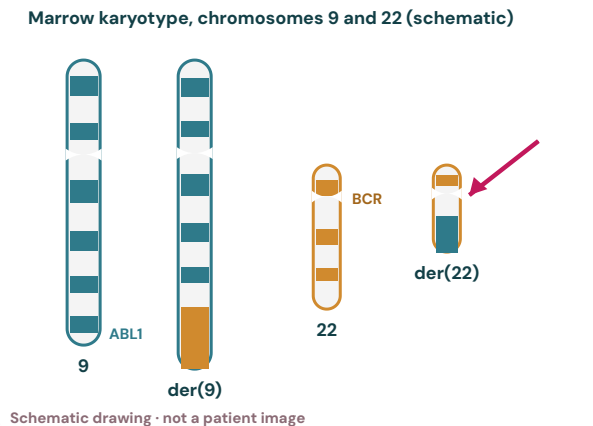
Q6. Diagnosis Which physical sign is most typical of chronic-phase CML?

- A Gum hypertrophy
- B Generalised lymphadenopathy
- C Skin nodules
- D Splenomegaly
- E Jaundice

Q7. Diagnosis A 30-year-old with pneumonia has a white count of 45,000/ μ L. Which finding favours a leukemoid reaction over CML?

- A Basophilia on the film
- B Toxic granules and Döhle bodies in neutrophils
- C More myelocytes than metamyelocytes
- D A spleen palpable 8 cm below the costal margin
- E A platelet count of 700,000/ μ L

Q8. Diagnosis This schematic shows chromosomes 9 and 22 from a marrow karyotype. What is the shortened chromosome marked by the arrow called?



- A A derivative chromosome 7
- B An isochromosome 17q
- C A ring chromosome 22
- D The Philadelphia chromosome
- E A double-minute chromosome

Q9. Diagnosis Which test confirms the diagnosis of CML in a blood sample?

- A Flow cytometry for CD34
- B Serum vitamin B12 level
- C BCR::ABL1 by RT-PCR (or FISH)
- D Leucocyte alkaline phosphatase score
- E JAK2 V617F mutation testing

Q10. Diagnosis BCR::ABL1 is positive in the blood. Why is a bone marrow examination still advised at diagnosis?

- A To exclude tuberculosis of the bone marrow
- B To define the phase and find extra chromosomes
- C To measure iron stores before starting treatment
- D To obtain the platelet count needed for ELTS
- E To confirm that BCR::ABL1 is really present

Q11. Diagnosis Which four items make up the ELTS score?

- A White count, basophils, eosinophils, sex
- B Karyotype, transcript type, JAK2, age
- C Age, haemoglobin, LDH, albumin
- D Spleen, liver, lymph nodes, B symptoms
- E Age, spleen size, blood blasts, platelets

Q12. Diagnosis Why should the spleen be measured and samples sent before hydroxyurea is started?

- A Hydroxyurea causes splenic rupture
- B The spleen grows during hydroxyurea
- C The ELTS score is valid only on untreated values
- D Hydroxyurea prevents a later TKI from working
- E Hydroxyurea makes BCR::ABL1 undetectable

- Q13. Diagnosis** What is a major molecular response (MMR) in CML?
- A BCR::ABL1 of 1% or less on the International Scale
 - B A normal white count and a spleen that is not palpable
 - C No Philadelphia-positive marrow metaphases
 - D BCR::ABL1 of 10% or less on the International Scale
 - E BCR::ABL1 of 0.1% or less on the International Scale
- Q14. Diagnosis** Which set of findings defines a complete haematological response?
- A WBC <10,000/ μ L, platelets <450,000/ μ L, no immature cells, basophils <5%, spleen impalpable
 - B BCR::ABL1 below 10% on the International Scale at 3 months of treatment
 - C Normal haemoglobin and no symptoms, whatever the size of the spleen or the platelet count
 - D No Philadelphia-positive metaphases among 20 or more marrow cells counted
 - E WBC <20,000/ μ L with any platelet count, blasts under 5% and a spleen of any size
- Q15. Treatment** Which statement about the first TKI in chronic-phase CML is correct?
- A Imatinib, a 2G TKI or asciminib give similar long-term survival
 - B Imatinib is no longer recommended for any patient
 - C Ponatinib is the preferred first TKI for most patients
 - D Second-generation TKIs add about 10% to overall survival
 - E Interferon should be added to every first-line TKI
- Q16. Treatment** Which TKI binds the myristoyl pocket of ABL1 rather than the ATP-binding site?
- A Dasatinib
 - B Imatinib
 - C Bosutinib
 - D Nilotinib
 - E Asciminib
- Q17. Treatment** Pleural effusion is a characteristic side effect of which TKI?
- A Nilotinib
 - B Asciminib
 - C Bosutinib
 - D Imatinib
 - E Dasatinib
- Q18. Treatment** For which patient is nilotinib generally best avoided?
- A A 45-year-old with lactose intolerance
 - B A 50-year-old with Crohn disease
 - C A 30-year-old with a past pleural effusion
 - D A 58-year-old with diabetes and a past stroke
 - E A 25-year-old with no other illness
- Q19. Treatment** Which side effect is most typical of bosutinib, mainly in the first weeks?
- A Pleural effusion
 - B Gynaecomastia
 - C Diarrhoea
 - D Periorbital oedema
 - E Peripheral arterial occlusion
- Q20. Treatment** How often should BCR::ABL1 (IS) be measured on a TKI until MMR is reached?
- A At least every 3 months
 - B Only at 12 months
 - C Once a year
 - D Only when the blood count changes
 - E Every week

- Q21. Treatment** Which set of results is "favourable" under ELN 2025 at 3, 6 and 12 months?
- A $\leq 1\%$, $\leq 0.1\%$ and $\leq 0.01\%$
 - B Undetectable at every time point
 - C $\leq 50\%$, $\leq 10\%$ and $\leq 1\%$
 - D $\leq 10\%$, $\leq 10\%$ and $\leq 10\%$
 - E $\leq 10\%$, $\leq 1\%$ and $\leq 0.1\%$
- Q22. Treatment** BCR::ABL1 is rising on imatinib. What is the first thing to check?
- A Missed tablets and interacting drugs
 - B A whole-body PET-CT scan
 - C HLA typing of brothers and sisters
 - D A kinase-domain mutation by NGS
 - E A repeat bone marrow biopsy and karyotype
- Q23. Treatment** A kinase-domain mutation test shows T315I. Which TKIs can still work?
- A Dasatinib or nilotinib
 - B Ponatinib or asciminib
 - C Imatinib at a higher dose
 - D Any second-generation TKI
 - E Bosutinib or nilotinib
- Q24. Treatment** Among carefully selected patients who stop their TKI, about what proportion stay in treatment-free remission?
- A About 20%
 - B 40–50%
 - C About 75%
 - D Nearly all
 - E Under 5%
- Q25. Treatment** A woman with chronic-phase CML becomes pregnant and her counts rise in the first trimester. Which treatment can be used at any stage of pregnancy?
- A Interferon
 - B Imatinib
 - C Nilotinib
 - D Hydroxyurea
 - E Dasatinib
- Q26. Survivorship** A man taking imatinib wants to father a child. What is the advice?
- A Switch to interferon until conception
 - B Stop the TKI for 3 months before trying
 - C Continue the TKI while trying to conceive
 - D Store sperm and avoid natural conception
 - E Stop the TKI and use hydroxyurea instead
- Q27. Treatment** How is myeloid blast phase of CML treated?
- A Interferon plus low-dose cytarabine
 - B Radiotherapy to the spleen and marrow
 - C A TKI with AML-type induction, then transplant
 - D Hydroxyurea and blood transfusions only
 - E A TKI alone, continued for life
- Q28. Survivorship** In Indian studies, up to what share of patients with CML do not take their TKI regularly?
- A About 1 in 20
 - B Up to one-third
 - C About three-quarters
 - D Under 1 in 50
 - E Almost all

Q29. Survivorship With TKI treatment, what is the life expectancy of most patients diagnosed in chronic phase?

- A Shorter than with interferon
- B Normal only after a transplant
- C Close to the general population
- D About 10 years at most
- E About 3 to 5 years from diagnosis

Q30. Diagnosis How does the age at diagnosis in Indian hospital series compare with Western registries?

- A The same (median 66 years)
- B Mostly children under 15
- C About a decade younger (median 32–42 years)
- D About a decade older (median 75 years)
- E Only people over 80

Level 2 · Applied & innovative

Q31. Diagnosis Which finding at diagnosis should make you think of MDS or an advanced phase rather than typical chronic-phase CML?

- A Basophilia on the film
- B A platelet count of 700,000/ μ L
- C A low platelet count
- D Eosinophilia on the film
- E A white count of 300,000/ μ L

Q32. Diagnosis A 40-year-old has a white count of 120,000/ μ L with myelocytes and basophils and a 10 cm spleen. Routine quantitative PCR for p210 and p190 is negative. What next?

- A Start imatinib without further tests
- B FISH for BCR::ABL1 with a marrow karyotype
- C Diagnose a leukemoid reaction and give antibiotics
- D Repeat the same PCR in 3 months
- E Treat as polycythaemia vera with hydroxyurea

Q33. Diagnosis A man treated for chronic-phase CML for 3 years now has lymphoblasts on his blood film. What should you assume, and do?

- A Reactive lymphocytosis after infection: no action
- B Lymphoid blast phase: flow cytometry and lumbar puncture
- C A second cancer (CLL): watch and wait without tests
- D An imatinib side effect: reduce the dose of imatinib
- E A viral reaction: repeat the blood film in 2 weeks

Q34. Diagnosis A marrow at diagnosis shows 14% blasts. How do ICC 2022 and WHO 2022 classify it?

- A Both systems: chronic phase without any risk features
- B Both systems: blast phase, treated like acute leukaemia
- C ICC: accelerated; WHO: chronic with a high-risk feature
- D ICC: blast phase; WHO: accelerated phase
- E Both systems: accelerated phase, with no other label

Q35. Diagnosis What blast percentage defines blast phase in ICC 2022 and WHO 2022, and how did the older ELN criteria differ?

- A 30% or more in all systems
- B 5% or more; the older ELN criteria used 10% or more
- C 50% or more; the older ELN criteria used 30% or more
- D 20% or more; the older ELN criteria used 30% or more
- E 10% or more; the older ELN criteria used 20% or more

Q36. Diagnosis A patient's BCR::ABL1 was 0.08% (IS) in laboratory A. A cheaper laboratory B now reports 0.3%. What is the correct step?

- A Double the TKI dose and recheck in a year
- B Order a kinase-domain mutation test straight away
- C Arrange a bone marrow biopsy and karyotype
- D Declare loss of MMR and switch to another TKI
- E Repeat in the original IS laboratory before acting

Q37. Treatment A 60-year-old with diabetes, a past myocardial infarction and a high ELTS score has chronic-phase CML. Which first-line choice fits best?

- A Nilotinib, because the ELTS score is high
- B Imatinib with interferon added from the start
- C Dasatinib, bosutinib or asciminib, not nilotinib
- D Interferon alone, to avoid TKI toxicity
- E Ponatinib, because the risk score is high

Q38. Treatment Which TKI is best avoided in a patient with chronic lung disease and a previous pleural effusion?

- A Dasatinib
- B Imatinib
- C Asciminib
- D Bosutinib
- E Nilotinib

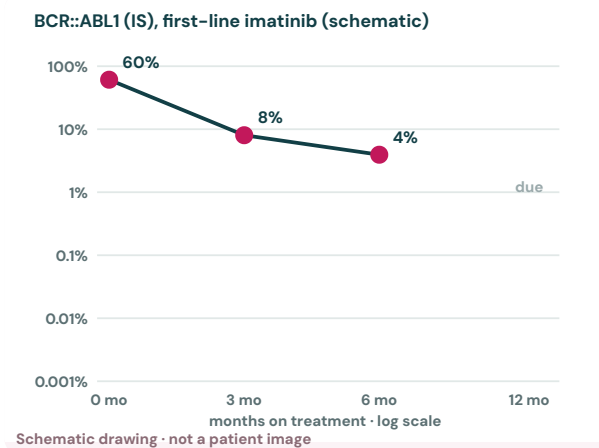
Q39. Treatment A patient on dasatinib is started on omeprazole for dyspepsia. What is the concern?

- A Omeprazole causes pleural effusion
- B Dasatinib levels rise to toxic levels
- C The PCR result becomes falsely low
- D Less dasatinib is absorbed
- E There is no interaction

Q40. Treatment When should an ECG be done after starting nilotinib or dasatinib?

- A At baseline and 1–2 weeks after starting
- B Only before stopping treatment
- C Once a year
- D Only if the patient has palpitations
- E It is not needed with these TKIs

Q41. Treatment This schematic chart shows one patient's BCR::ABL1 (IS) on first-line imatinib. How does ELN 2025 classify the 6-month result?



- A Treatment-free remission can be tried
- B Favourable: no change needed
- C Unfavourable: a switch is preferred
- D Warning: a switch may be needed
- E Major molecular response

- Q42. Treatment** Two weeks after starting imatinib, the platelet count has fallen mildly and the patient is well. What is appropriate?
- A Continue: early cytopenias reflect the leukaemia clearing
 - B Stop imatinib permanently and switch to another TKI
 - C Begin a work-up for urgent allogeneic transplant
 - D Start an erythropoietin-stimulating agent at once
 - E Switch to ponatinib because of primary resistance
- Q43. Treatment** BCR::ABL1 has risen on two tests and the patient takes every tablet with no interacting drugs. What comes next?
- A Adding interferon to the same TKI and continuing
 - B Stopping all treatment and watching the counts
 - C A drug holiday of 3 months, then repeat the PCR
 - D Increasing hydroxyurea alongside the same TKI
 - E Marrow (blasts, karyotype) and a mutation test
- Q44. Treatment** A patient loses response on dasatinib (first line). No mutation is found. Which next step fits current guidance?
- A Switch back to imatinib
 - B Switch to nilotinib
 - C Bosutinib, as it always works after dasatinib
 - D Ponatinib or asciminib, and start a donor search
 - E Continue dasatinib and recheck in a year
- Q45. Treatment** F317L is found in a patient losing response on dasatinib. Which TKI is NOT expected to work against this mutation?
- A Ponatinib
 - B Dasatinib
 - C Asciminib
 - D Bosutinib
 - E Nilotinib
- Q46. Treatment** V299L is found in a patient on bosutinib. Which of these is a suitable next TKI?
- A Nilotinib
 - B Imatinib at a higher dose
 - C Dasatinib
 - D Bosutinib at the same dose
 - E Imatinib
- Q47. Treatment** Four months after stopping a TKI in deep remission, a patient loses MMR. What is the correct response?
- A Wait for a second positive result 3 months later
 - B Start ponatinib
 - C Arrange an urgent allogeneic transplant
 - D Restart the TKI; no mutation test is needed
 - E Order a mutation test before restarting
- Q48. Treatment** After stopping a TKI, when do most molecular relapses occur?
- A After 5 years off treatment
 - B Evenly spread over 10 years
 - C In the second year off treatment
 - D In the first week after stopping
 - E Within the first 6–8 months
- Q49. Treatment** A 29-year-old woman has been in MR4 for 3 years on imatinib (6 years in total) and wants a pregnancy. What does ELN 2025 advise?
- A Continue imatinib throughout the whole pregnancy
 - B Advise against pregnancy at any time in the future
 - C Stop the TKI as for TFR, then plan the pregnancy
 - D Switch to nilotinib and try to conceive straight away
 - E Switch to dasatinib before trying to conceive

Q50. Treatment A woman in MMR (but not MR4) with regular cycles and good access to testing wants a pregnancy. Which option does ELN 2025 accept?

- A Switch to ponatinib before trying to conceive
- B Continue the TKI until the first positive test
- C Undergo allogeneic transplant before conceiving
- D Continue the TKI throughout the whole pregnancy
- E Stop the TKI only after the baby is delivered

Q51. Treatment CML is diagnosed at 10 weeks of pregnancy: white count 60,000/ μ L, low ELTS, no extra chromosomes, no symptoms. What is reasonable?

- A Start dasatinib now, as it is the most potent TKI
- B Watchful waiting; leukapheresis or interferon if needed
- C Termination of the pregnancy in every case
- D Start imatinib now, before the counts rise further
- E Hydroxyurea throughout the rest of the pregnancy

Q52. Treatment Which TKI should be avoided throughout pregnancy, even after week 16?

- A None: all are allowed after week 16
- B Nilotinib
- C Imatinib
- D Interferon (not a TKI)
- E Dasatinib

Q53. Treatment A mother on imatinib after delivery asks about breastfeeding. What is the advice?

- A Breastfeed only on alternate days
- B Breastfeeding is safe on imatinib only
- C Switch to dasatinib to breastfeed
- D No TKI while breastfeeding
- E Breastfeeding is safe on any TKI

Q54. Treatment A fit 40-year-old with CML presents in myeloid blast phase with no prior TKI. What is the best plan?

- A TKI with intensive AML induction, then transplant
- B Hydroxyurea and palliative care from the start
- C Imatinib alone, continued for the rest of life
- D Intensive AML chemotherapy without any TKI
- E Treatment-free remission once induction ends

Q55. Treatment In lymphoid blast phase of CML, which combination is used?

- A Interferon and hydroxyurea, continued indefinitely
- B TKI + Ph+ ALL regimen, lumbar puncture, then transplant
- C A TKI alone, without any chemotherapy or transplant
- D AML-type induction chemotherapy without a TKI
- E Radiotherapy to the spleen and the whole marrow

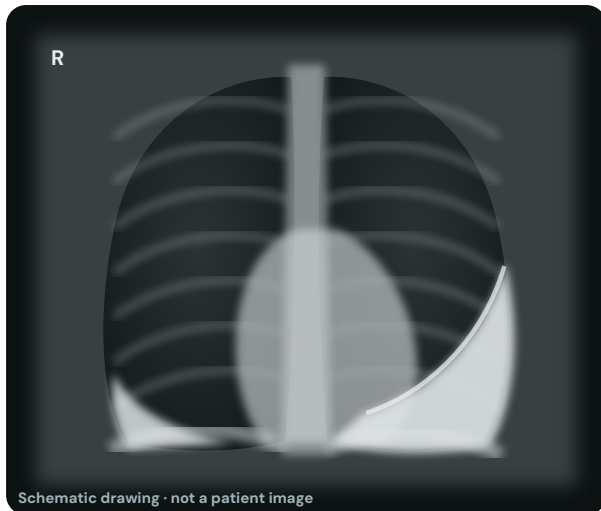
Q56. Treatment In which situation is a lumbar puncture indicated in CML?

- A Lymphoid blast phase or neurological signs
- B Every patient at diagnosis
- C Before every bone marrow biopsy
- D Every patient before stopping a TKI
- E Neurologically well patients in myeloid blast phase

Q57. Treatment A patient presents de novo in accelerated phase. Which statement is correct?

- A Imatinib is the preferred TKI in this phase
- B Transplant is needed before any TKI is given
- C No TKI is effective once accelerated phase is reached
- D A 2G TKI is preferred to imatinib; no TFR attempts
- E TFR can be tried after 2 years of deep response

Q58. Treatment This schematic chest X-ray is from a 55-year-old on dasatinib for 2 years with new breathlessness. What is the most appropriate approach?



- A Switch to imatinib at once without any further assessment
 - B Assess the effusion, exclude other causes, hold or reduce dasatinib
 - C Continue dasatinib unchanged and add a diuretic only
 - D Stop all TKIs permanently and watch the counts
 - E Start anti-tuberculosis treatment empirically for 6 months
- Q59. Survivorship** Which ongoing checks matter most for a patient on long-term nilotinib?
- A Pulmonary function every 3 months
 - B Bone marrow every 6 months
 - C Blood pressure, glucose and lipids
 - D Thyroid scans every year
 - E Hearing and vision
- Q60. Survivorship** A 12-year-old on imatinib for 18 months has slowed growth. Which statement is correct?
- A Always temporary, with full catch-up within 6 months
 - B A recognised TKI effect: watch height, puberty, thyroid and bone
 - C Unrelated to the TKI, so no action or follow-up is needed
 - D A sign that the CML has progressed to blast phase
 - E A reason to stop imatinib at once and permanently

Level 3 · Challenging

- Q61. Treatment** In IRIS, what was the 18-month progression-free survival with imatinib versus interferon plus cytarabine?
- A 92% vs 97%
 - B 80% vs 60%
 - C 75% vs 70%
 - D 97% vs 92%
 - E 99% vs 50%
- Q62. Treatment** In ENESTnd, how many progressions to accelerated or blast phase occurred on imatinib compared with the two nilotinib arms?
- A None in any arm
 - B 12 vs 33 and 40
 - C 9 vs 12 and 33
 - D 100 vs 50 and 25
 - E 33 vs 12 and 9

- Q63. Treatment** In a meta-analysis of 8 trials (2974 patients) comparing 2G TKIs with imatinib, what was the rate of progression?
- A 10% vs 20%, with a survival gain
 - B No difference in progression
 - C 0.8% vs 3.0%, with no survival difference
 - D 0.8% vs 3.0%, with a 10% survival gain
 - E 3.0% vs 0.8%, favouring imatinib
- Q64. Treatment** In ASC4FIRST, what was the MMR rate at 96 weeks with asciminib versus investigator-chosen TKIs?
- A 96% vs 52%
 - B 37.6% vs 15.8%
 - C 52.0% vs 74.1%
 - D 74.1% vs 52.0%
 - E 44% vs 23%
- Q65. Treatment** In ASCEMBL (patients after 2 or more TKIs), what was the MMR rate at 96 weeks?
- A No difference
 - B 60% with asciminib vs 40% with ponatinib
 - C 37.6% with asciminib vs 15.8% with bosutinib
 - D 15.8% with asciminib vs 37.6% with bosutinib
 - E 74.1% with asciminib vs 52.0% with bosutinib
- Q66. Treatment** In PACE (chronic phase after dasatinib or nilotinib, or with T315I), which pair of results is correct for ponatinib?
- A 5-year OS 73%, arterial occlusive events 31%
 - B MMR 90%, arterial occlusive events 1%
 - C 5-year OS 31%, arterial occlusive events 73%
 - D 5-year OS 50%, no vascular events
 - E No responses in T315I
- Q67. Treatment** What was the key design idea of OPTIC?
- A Compare ponatinib with allogeneic transplant in T315I
 - B Use ponatinib first line in low-risk chronic phase
 - C Stop ponatinib after 12 months in major molecular response
 - D Start ponatinib higher, reduce once BCR::ABL1 <1%
 - E Combine ponatinib with pegylated interferon
- Q68. Treatment** In a registry comparison of patients with T315I, what was 48-month survival with ponatinib versus allogeneic transplant?
- A 30% vs 80%
 - B 73% vs 56%
 - C 20% vs 20%
 - D 95% vs 40%
 - E 56% vs 73%
- Q69. Treatment** In EURO-SKI (755 patients), what proportion kept MMR 6 and 12 months after stopping a TKI?
- A 90% and 85%
 - B 46% and 61%
 - C 100% and 95%
 - D 20% and 10%
 - E 61% and 46%
- Q70. Treatment** In DESTINY, what was the treatment-free remission rate after halving the dose for 12 months before stopping, in patients with MR4 or deeper?
- A 25%
 - B 10%
 - C 95%
 - D 45%
 - E 72%

- Q71. Treatment** Which statement about late outcomes after stopping a TKI is correct?
- A Late MMR loss in up to 14%; sudden blast phase $\leq 0.1\%$
 - B Relapse never happens after 12 months off treatment
 - C Once MMR is lost, a second response is unusual
 - D Blast phase occurs in about 10% of those who stop
 - E Most relapses happen more than 5 years after stopping
- Q72. Treatment** In CML Study IV (Lauseker 2023), what was 10-year survival in patients still above 10% BCR::ABL1 at 12 months?
- A Under 5%
 - B About 20%
 - C About 55%
 - D About 80%
 - E About 95%
- Q73. Treatment** Without an early molecular response on imatinib, what did switching to dasatinib achieve in DASCERN?
- A No difference in major molecular response
 - B More progressions to blast phase on dasatinib
 - C Treatment-free remission in most patients
 - D Deeper responses (MMR 77% vs 44%), same survival
 - E A large overall survival gain over imatinib
- Q74. Survivorship** In Marin 2010, what proportion of patients with 90% adherence or more reached MMR at 6 years, compared with those below 90%?
- A 28% vs 95%
 - B 100% vs 80%
 - C 60% vs 55%
 - D 50% vs 45%
 - E 95% vs 28%
- Q75. Diagnosis** In CML Study IV (Saussele 2015), how did 8-year overall survival change across Charlson comorbidity groups?
- A It was the same in all groups
 - B From 80% to 75%
 - C From 94% (Charlson 2) to 46% (7 or more)
 - D From 99% to 95%
 - E From 46% to 94% (rising with comorbidity)
- Q76. Treatment** In 104 patients with myeloid blast phase (Saxena 2021), what was 5-year overall survival with a TKI plus intensive chemotherapy?
- A 0%
 - B 50%
 - C 60%
 - D 30%
 - E 80%
- Q77. Treatment** In lymphoid blast phase treated with hyper-CVAD plus a TKI (Strati 2014), what was median overall survival with and without allogeneic transplant?
- A 9 months vs 93 months
 - B Not reached in either group
 - C 93 months vs 9 months
 - D 60 months vs 48 months
 - E 24 months in both groups

Q78. Treatment Among imatinib-exposed pregnancies with known outcome (Pye 2008), about what proportion had fetal abnormalities?

- A About 50%
- B About 1%
- C About 10%
- D None
- E About 30%

Q79. Treatment In the ELN registry of 87 women diagnosed with CML during pregnancy (Chelysheva 2024), what happened?

- A 76% had a normal childbirth, no excess malformations
- B Most women progressed to blast phase in pregnancy
- C Nearly all women received dasatinib in pregnancy
- D Malformations occurred in about half of the babies
- E Most pregnancies had to be terminated for safety

Q80. Treatment In a Japanese series of 70 pregnancies (Kondo 2025), when did planned pregnancies occur compared with unplanned ones?

- A A median 4.1 vs 10.6 years after diagnosis
- B Both within the first year
- C Only after allogeneic transplant
- D A median 10.6 vs 4.1 years after diagnosis
- E A median 1 vs 10 years after diagnosis

Q81. Diagnosis Which transcripts are expected to resist asciminib?

- A Those lacking ABL1 exon 2 (e13a3, e14a3)
- B Any p190 (e1a2) transcript, whatever the phase
- C The rare p230 (e19a2) transcript in every case
- D The common e13a2 and e14a2 transcripts
- E Any transcript carrying the T315I mutation

Q82. Treatment Which statement about compound kinase-domain mutations is correct?

- A They respond best to imatinib at a standard dose
- B They always respond to bosutinib as second line
- C Sanger sequencing detects them better than NGS
- D They are found routinely at diagnosis in chronic phase
- E Some with T315I resist both ponatinib and asciminib

Q83. Treatment Under the ELN 2025 mutation table, which TKI is NOT recommended for E255K/V?

- A Dasatinib
- B Nilotinib
- C Ponatinib
- D Asciminib
- E None: all four are recommended

Q84. Treatment F359V is found in a patient losing response on nilotinib. Which option does the ELN 2025 table recommend?

- A Continuing nilotinib
- B Stopping all TKIs
- C Asciminib
- D Dasatinib
- E Imatinib at a higher dose

Q85. Treatment A myristoyl-pocket mutation (for example A337V) is found on asciminib. Which statement is correct?

- A ATP-site TKIs may still work
- B Only imatinib remains active
- C A higher asciminib dose overcomes it
- D It always means blast phase
- E No TKI can work any longer

- Q86. Treatment** Which statement on allogeneic transplant in CML reflects ELN 2025?
- A Reduced-intensity conditioning is preferred for every patient
 - B Myeloablative conditioning is preferred where possible
 - C Donor search should wait until ponatinib has failed
 - D TKIs must never be given again after transplant
 - E Transplant of resistant blast phase is advised outside trials
- Q87. Treatment** In de novo accelerated phase treated with dasatinib or nilotinib (Ohanian 2014), what was the complete cytogenetic response rate in 21 patients?
- A About 90%
 - B About 10%
 - C None
 - D About 50%
 - E About 30%
- Q88. Survivorship** In the international registry of 535 children (Milot 2026), what was 3-year PFS by ELTS group (low, intermediate, high)?
- A 99% in all groups
 - B 72.0%, 91.7% and 97.1%
 - C 50%, 40% and 30%
 - D 80%, 60% and 20%
 - E 97.1%, 91.7% and 72.0%
- Q89. Treatment** What did the Tata Memorial series (Parikh 2013; 972 patients, median age 36) show?
- A Interferon gave better results than imatinib
 - B Generic imatinib gave much worse responses
 - C OS was only 40% at 5 years on imatinib
 - D Most patients presented in blast phase
 - E OS 86% at 51 months; generic = innovator
- Q90. Treatment** In heavily pretreated patients (Hughes 2019), what proportion reached MMR at 12 months on asciminib?
- A 90% overall and 80% of those with T315I
 - B 10% overall and none with T315I
 - C 74% overall and 52% with T315I
 - D 48% overall and 28% of those with T315I
 - E 28% overall and 48% of those with T315I

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 The Philadelphia chromosome, t(9;22)(q34;q11.2), joins BCR to ABL1. One fusion gene drives the disease, and it is the target of every tyrosine kinase inhibitor (TKI).

Q2 · E The fusion makes a constitutively active ABL1 tyrosine kinase. TKIs block this kinase; asciminib binds a different site (the myristoyl pocket) from the others (the ATP site).

Q3 · E Ionising radiation is the only known risk factor. There is no familial tendency, and CML is not passed on to children.

Q4 · A Chronic-phase CML shows every stage from myeloblast to neutrophil, more myelocytes than metamyelocytes, basophilia (always) and often eosinophilia, with blasts usually under 2%.

Q5 · B Up to half are diagnosed from a routine blood count. When present, symptoms include fatigue (34%), weight loss (20%), sweats and abdominal fullness (15% each).

Q6 · D The spleen is palpable in about half to three-quarters of patients, causing early satiety or left-sided pain. Nodes, skin or soft-tissue masses suggest a later phase.

Q7 · B Toxic granulation and Döhle bodies point to infection or inflammation; leukemoid reactions are rarely as high as CML and are BCR::ABL1 negative. Basophilia, a myelocyte bulge and a big spleen favour CML.

Q8 · D The reciprocal translocation moves ABL1 from chromosome 9 to chromosome 22, leaving a short der(22): the Philadelphia (Ph) chromosome. More than 90% of patients show it on banding.

Q9 · C CML is confirmed by t(9;22) on banding or FISH and/or BCR::ABL1 by RT-PCR, in blood or marrow. The transcript type and a baseline on the International Scale should be recorded.

Q10 · B Blood PCR confirms CML, but marrow morphology (blasts) and karyotype (extra chromosomes) decide the phase and the risk. A marrow is omitted only when the result would not change treatment.

Q11 · E The EUTOS long-term survival (ELTS) score uses age, spleen size in cm, blood blasts and platelets. It was built on TKI-treated patients and best predicts death from CML; ELN 2025 prefers it to Sokal, Euro and EUTOS.

Q12 · C ELTS needs untreated spleen size, blasts and platelets. Hydroxyurea itself does not affect later milestones, but the risk score is lost if values are recorded after it.

Q13 · E MMR (MR3) is BCR::ABL1 $\leq 0.1\%$ IS; MR4 is $\leq 0.01\%$, MR4.5 $\leq 0.0032\%$ and MR5 $\leq 0.001\%$. Deep molecular response means MR4 or deeper.

Q14 · A Complete haematological response is the first goal of treatment. No Ph-positive metaphases is a complete cytogenetic response; $\leq 10\%$ at 3 months is a molecular milestone.

Q15 · A 2G TKIs and asciminib give faster and deeper responses and fewer transformations than imatinib, but no overall survival gain. Comorbidity, risk score, goals and cost decide the choice.

Q16 · E Asciminib is an allosteric (STAMP) inhibitor. Because it binds a different site, it works against many ATP-site mutations, including T315I, but myristoyl-pocket mutations resist it.

Q17 · E Dasatinib causes pleural and pericardial effusions and, rarely, pulmonary arterial hypertension. It also impairs platelet function and can prolong the QTc.

Q18 · D Nilotinib causes arterial occlusive disease, raised glucose and cholesterol, QTc prolongation and pancreatitis. It is avoided with diabetes, high cardiovascular risk or a previous vascular event. A past effusion argues against dasatinib; bowel disease against bosutinib.

- Q19 · C** Bosutinib commonly causes early diarrhoea and raised liver enzymes; some start low and escalate. It is avoided with bowel, liver or kidney disease.
- Q20 · A** PCR is done at least every 3 months until MMR is achieved and confirmed, then every 3–6 months (UpToDate) or 4–6 months (ELN 2025); more often if values fluctuate or rise.
- Q21 · E** Favourable milestones are BCR::ABL1 $\leq 10\%$ at 3 months, $\leq 1\%$ at 6 months and $\leq 0.1\%$ (MMR) at 12 months. ELN 2025 renamed optimal/warning/failure as favourable/warning/unfavourable.
- Q22 · A** "Resistance" is often missed tablets. Ask kindly and specifically about adherence and new medicines before ordering mutation tests or switching.
- Q23 · B** T315I resists imatinib, dasatinib, nilotinib and bosutinib. Ponatinib or asciminib are used, with a parallel donor search.
- Q24 · B** About 40–50% of eligible patients stay off treatment. Most who relapse lose MMR in the first 6–8 months and regain their response when the TKI is restarted.
- Q25 · A** All TKIs are potentially teratogenic until organogenesis is complete (around week 16). Interferon, including pegylated forms, can be used at any time; imatinib or nilotinib only after week 16; dasatinib is avoided throughout.
- Q26 · C** There is no convincing effect of TKIs on male fertility or on their children: in 428 pregnancies of partners of men on TKIs, 93.5% were live births with 2.5% congenital abnormalities.
- Q27 · C** Myeloid blast phase is treated like AML (intensive induction or, in older patients, a hypomethylating agent $\pm$ venetoclax) with a TKI, followed by allogeneic transplant where possible.
- Q28 · B** Non-adherence affects up to one-third of Indian patients and shortens event-free survival. Adherence is treatment: ask at every visit, involve the family and link to assistance programmes.
- Q29 · C** Life expectancy now approaches that of the general population (Bower 2016), although ELN 2025 notes small residual losses in Swedish data.
- Q30 · C** Indian hospital series report median ages of 32–42, with more symptoms, bigger spleens and more high-risk scores, compared with 55 in Europe and 66 in the US. The Mumbai population registry shows the usual peak at 55–74, so referral patterns explain part of the gap.
- Q31 · C** Low platelets are uncommon in chronic phase. High platelets (over 600,000/ μL in 15–30%), basophilia, eosinophilia and very high white counts are typical of chronic phase.
- Q32 · B** Routine kits detect only p210 and p190; p230 and rare transcripts, and some cryptic fusions, need FISH or a personalised assay. With high suspicion and a negative karyotype, FISH is mandatory.
- Q33 · B** Lymphoblasts in a patient with CML mean lymphoid blast phase until proved otherwise. Lineage by flow cytometry decides treatment (a Ph+ ALL-type regimen with a TKI), and a lumbar puncture is needed.
- Q34 · C** ICC keeps three phases (accelerated 10–19% blasts, basophils $\geq 20\%$, high-risk extra chromosomes). WHO 2022 has only chronic and blast phase and lists blasts 10–19% as a high-risk feature. Both agree such patients need a more potent TKI and closer monitoring.
- Q35 · D** Both modern systems use $\geq 20\%$ blasts (or extramedullary blast proliferation, or bona fide lymphoblasts). Most trials used the older ELN cut-off of $\geq 30\%$, so read trial phase definitions carefully.
- Q36 · E** Results are comparable only on the International Scale in a standardised laboratory: one laboratory, one scale. Never switch on one result; repeat near a threshold within 1–2 months.
- Q37 · C** High ELTS favours a 2G TKI or asciminib; comorbidity then decides. Nilotinib is avoided with diabetes or vascular disease. Interferon added to a TKI is not recommended, and ponatinib is not a first-line drug.
- Q38 · A** Dasatinib causes effusions and pulmonary arterial hypertension, so lung disease, effusions and bleeding disorders argue against it.
- Q39 · D** Acid-suppressing drugs reduce absorption of dasatinib and nilotinib (a dasatinib formulation that tolerates them exists in some markets). Check every new prescription, including azoles, macrolides and QT-prolonging drugs.
- Q40 · A** An ECG for the QTc is done at baseline, 1–2 weeks after starting nilotinib or dasatinib, after dose changes and when new QT-prolonging drugs are added; potassium and magnesium are corrected.
- Q41 · D** At 6 months, $\leq 1\%$ is favourable, $>1\text{--}10\%$ is warning and $>10\%$ is unfavourable. A warning result calls for a repeat, questions about missed doses and interacting drugs, and a decision based on the trend and the patient's goals.

Q42 · A Mild early cytopenias are mostly the leukaemia clearing; continue. Severe ones need a hold or dose reduction. Erythropoietin-stimulating agents are avoided (thrombosis, no benefit).

Q43 · E After confirming the rise and checking adherence and interactions, look for progression (blasts, extra chromosomes) and kinase-domain mutations, then switch TKI. Mutation testing is not done at diagnosis in chronic phase.

Q44 · D After a 2G TKI fails, another 2G TKI rarely works; ponatinib or asciminib are preferred. ELN 2025 advises a donor search from resistance to the first 2G TKI.

Q45 · B In the ELN 2025 mutation table, F317L/V/I/C resists dasatinib; nilotinib, bosutinib, ponatinib and asciminib remain options. These are in-vitro data, read with the patient's history.

Q46 · A V299L resists dasatinib and bosutinib; nilotinib, ponatinib and asciminib are listed as options. Most kinase-domain mutations resist imatinib.

Q47 · D Loss of MMR after stopping is the trigger to restart (one result is enough under ELN 2025). Mutation testing is not needed: 90–95% regain their response on the same TKI.

Q48 · E More than 80% of molecular relapses occur in the first 6–8 months, so PCR is done every 6–8 weeks early on, then every 2 months, then every 3–6 months for life. Late loss of MMR occurs in up to 14% after 2 years.

Q49 · C With MR4 or deeper, women stop as for TFR with close monitoring. Pregnancy should be raised at diagnosis with every woman of child-bearing age; planned pregnancies take time.

Q50 · B With MMR, women may stop and watch, or (with regular cycles and access to tests) continue until the first positive pregnancy test, then stop. Without MMR the response should be deepened first.

Q51 · B With WBC <100,000/ μ L, low or intermediate ELTS and no extra chromosomes, watchful waiting is reasonable; leukapheresis and/or interferon for symptoms or high counts; aspirin and/or LMW heparin for high platelets. No TKI before week 16.

Q52 · E Imatinib and nilotinib cross the placenta poorly and can be used after week 16 if control is lost. Dasatinib reached 75% of maternal levels in the one case measured and is avoided throughout.

Q53 · D ELN 2025 advises no TKI while breastfeeding. The choice between breastfeeding and restarting treatment is planned in advance with the mother.

Q54 · A In 104 patients, 5-year OS was 30% with a TKI plus intensive chemotherapy, 28% with a TKI plus a hypomethylating agent, 13% with a TKI alone and 0% with chemotherapy alone. Imatinib is avoided in blast phase; transplant follows.

Q55 · B Lymphoid blast phase (about one-third of blast phases) is treated like Ph+ ALL (hyper-CVAD best studied; blinatumomab or inotuzumab with a TKI emerging), with CNS assessment and allogeneic transplant.

Q56 · A A lumbar puncture is done in lymphoid blast phase or with neurological signs, not in neurologically well patients with myeloid blast phase. Dasatinib, bosutinib, nilotinib and ponatinib cross the blood-brain barrier.

Q57 · D De novo accelerated phase is treated with a 2G TKI (or ponatinib, or upfront transplant), continued indefinitely; there are no TFR data. BCR::ABL1 <10% at 3 months predicts better survival.

Q58 · B Effusions on dasatinib need a chest X-ray and exclusion of infection, heart failure, kidney disease and progression. Some need thoracentesis, a drain or pleurodesis, with interruption or dose reduction; persistent problems mean a switch.

Q59 · C Cardiovascular risk is highest with ponatinib and nilotinib. Blood pressure, glucose, lipids and symptoms of vascular disease are checked at visits; age, hypertension, diabetes, lipids and smoking add risk whatever the TKI.

Q60 · B In the German CML-PAED II study, median height fell by 0.35 SD at 12 months and 0.76 SD at 24 months. Children need growth, puberty, thyroid and bone surveillance and endocrinology input.

Q61 · D IRIS (1106 patients) established imatinib, with better quality of life. 10-year OS was 84%, with most of the control arm crossing over.

Q62 · E Nilotinib reduced progression but caused more cardiovascular events, with no overall survival gain.

Q63 · C Second-generation TKIs gave faster, deeper responses and fewer transformations (Yun 2016), but overall survival was the same.

- Q64 · D** Asciminib also gave event-free survival of 85.6% vs 70.6% and fewer discontinuations; overall survival is not yet reported. FDA approved it first line; EMA only after ≥ 2 TKIs.
- Q65 · C** Asciminib also caused fewer discontinuations for side effects than bosutinib.
- Q66 · A** PACE gave a major cytogenetic response in 60% and MMR in 40%, but vascular events in 31%. OPTIC then tested response-based dose reduction.
- Q67 · D** Response-based reduction balanced efficacy (44%, 29% and 23% below 1% at 12 months by starting dose) against arterial events (10%, 5% and 3%).
- Q68 · B** Nicolini 2017 favoured ponatinib in chronic phase with T315I. Omacetaxine is no longer manufactured; olverembatinib is approved only in China.
- Q69 · E** LAST reported 77% at 6 and 71% at 12 months, with less fatigue, depression, diarrhoea and sleep disturbance. Similar results followed nilotinib (ENESTfreedom) and dasatinib (DASFREED).
- Q70 · E** De-escalation before stopping also lets a patient who relapses restart at the reduced dose. Overall, 40–50% of eligible patients stay off treatment.
- Q71 · A** Monitoring is the safety net: >80% of relapses come in the first 6–8 months, late loss can occur (Rousselot 2020), and 90–95% regain their response on restarting. A withdrawal syndrome (aches) affects 20–30%.
- Q72 · C** Patients who missed the 3-, 6- or 12-month milestones still had about 80% 10-year survival, but only 55% if BCR::ABL1 was still >10% at 12 months. This is why ELN 2025 bases switching on the trend, adherence, comorbidity and goals.
- Q73 · D** Fit or younger patients seeking TFR may switch for a deeper response; older or comorbid patients may continue with close monitoring.
- Q74 · E** No patient with $\leq 80\%$ adherence reached MMR. Adherence is part of treatment, and in India non-adherence (Ganesan 2011) shortens event-free survival.
- Q75 · C** Comorbidity now predicts survival more than CML itself for many patients, because most now die of other causes. ELTS best predicts death from CML.
- Q76 · D** 5-year OS was 30% with TKI + intensive chemotherapy, 28% with TKI + a hypomethylating agent, 13% with a TKI alone and 0% with chemotherapy alone. MATCHPOINT (ponatinib + FLAG-IDA) bridged two-thirds back to chronic phase.
- Q77 · C** Transplant after remission is the main route to long-term survival in blast phase. Lymphoid blast phase makes up about one-third of blast phases.
- Q78 · C** Of 180 exposed pregnancies, half with known outcome were normal and about 10% had abnormalities. Risk is assumed from implantation (about 15 days after fertilisation) until about week 16, which is why women stop TKIs.
- Q79 · A** Treatment was mostly interferon and, after week 16, imatinib. In advanced phase, treatment cannot wait, and termination should be discussed with the woman and her family.
- Q80 · D** Planned pregnancies (during TFR or on interferon) take time, which is why parenting is discussed from diagnosis.
- Q81 · A** Rare transcripts without ABL1 exon 2 are expected to resist asciminib (ELN 2025). The transcript type also decides how to monitor; e14a2 is associated with more successful TFR.
- Q82 · E** Compound mutations (two in the same molecule) resist most TKIs. NGS finds emerging clones earlier than Sanger sequencing and identifies compound mutations; transplant should be considered.
- Q83 · B** ELN lists dasatinib, ponatinib and asciminib for E255K/V; nilotinib and bosutinib are not recommended. The UpToDate in-vitro table also lists bosutinib, so read the laboratory report with both tables.
- Q84 · D** For F359V/I/C, ELN lists dasatinib and ponatinib; nilotinib, bosutinib and asciminib are not recommended. (UpToDate also lists bosutinib for F359V.)
- Q85 · A** Myristoyl-pocket mutations resist asciminib but not the ATP-site TKIs, which bind elsewhere on the kinase.
- Q86 · B** Reduced intensity is used for older or less fit patients. A donor search starts at resistance to the first 2G TKI, and transplant referral at resistance to ponatinib and/or asciminib. TKIs and donor lymphocytes treat molecular relapse after transplant.

Q87 · A A 2G TKI is preferred to imatinib in de novo accelerated phase, continued indefinitely. In transformed accelerated phase, dasatinib after imatinib gave complete haematological responses in 50% and complete cytogenetic responses in 32%.

Q88 · E 3-year OS was 97.4%. Children have bulky disease, more lymphoid blast crises and slower growth on imatinib, and stopping succeeds less often (28.6% in STOP IMAPED at 6 months).

Q89 · E Generic imatinib and assistance programmes (GIPAP, Max Foundation) made CML treatable for most Indian patients. ELN 2025 puts generic imatinib at as little as US\$200–500 a year.

Q90 · D Responses included some patients after ponatinib. For T315I, asciminib is given at a higher dose than for other mutations.