POST-TEST

Oncology Today with Dr Neil Love: Management of Acute Myeloid Leukemia Not Eligible for Intensive Induction Therapy Edition (Presentation Video)

THE CORRECT ANSWER IS INDICATED WITH YELLOW HIGHLIGHTING.

- 1. The Phase III VIALE-C trial evaluating venetoclax or placebo, each with low-dose cytarabine, for previously untreated acute myeloid leukemia (AML) in patients with comorbidities precluding intensive chemotherapy demonstrated which outcome on the venetoclaxcontaining arm?
 - a. Improved response rates only
 - b. Increased median overall survival (OS) only
 - c. Improvement in transfusion independence only
 - d. Improvement in response rates and transfusion independence only
 - e. Improvement in response rates, median OS and transfusion independence
- 2. Which of the following results best reflects outcomes on the azacitidine/ venetoclax arm of the Phase III VIALE-A trial evaluating azacitidine with either venetoclax or placebo for patients with treatment-naïve AML not eligible for intensive therapy?
 - a. A statistically significant improvement in OS
 - An increase in the proportion of patients achieving complete response

c. Both a and b

d. Neither a nor b

- 3. A subgroup analysis published in *The New England Journal of Medicine* by DiNardo and colleagues from the VIALE-A trial demonstrated increased response rates with venetoclax versus placebo in which of the following patient populations?
 - a. Only patients with poor-risk cytogenetics
 - b. Only patients with secondary AML
 - c. Only patients with FLT3 mutations
 - d. Only patients with IDH1/2 mutations
 - e. Only patients with p53 mutations
 - f. Only patients with poor-risk cytogenetics and/or secondary AML
 - g. Patients with any of the above disease characteristics
- 4. What is the mechanism of action of glasdegib, which was FDA approved in combination with low-dose cytarabine for patients with newly diagnosed AML aged 75 years or older or with comorbidities that preclude intensive induction chemotherapy, an approval based on data from the Phase II BRIGHT AML 1003 trial?
 - a. Bcl-2 inhibition
 - b. Hypomethylation
 - c. Hedgehog signaling pathway inhibition