

Oncology Today with Dr Neil Love: Optimizing the Management of Dedifferentiated Liposarcoma

THE CORRECT ANSWER IS INDICATED WITH YELLOW HIGHLIGHTING.

- 1. Which of the following best explains the mechanistic rationale for inhibiting the MDM2-p53 pathway for dedifferentiated liposarcoma?**
 - a. The MDM2 protein is necessary for tumor angiogenesis in liposarcoma and inhibition results in reduced proliferation
 - b. The MDM2 protein is a negative regulator of p53 and overexpression of MDM2 potentially inactivates wild-type p53**
 - c. The MDM2 protein is directly responsible for repairing double-stranded DNA breaks and inhibition results in tumor cell apoptosis
- 2. MDM2 amplification and alterations are most prominent in which solid tumor?**
 - a. Melanoma
 - b. Gastric cancer
 - c. Liposarcoma**
 - d. Breast cancer
 - e. Bladder cancer
- 3. The Phase III MANTRA study evaluating milademetan versus trabectedin for unresectable and/or metastatic dedifferentiated liposarcoma (DDLPS) reported what efficacy result?**
 - a. A statistically significant improvement in progression-free survival (PFS) with milademetan
 - b. A statistically significant improvement in overall survival (OS) with milademetan
 - c. A statistically significant improvement in both PFS and OS with milademetan
 - d. A statistically significant improvement in neither PFS nor OS with milademetan**
- 4. In a Phase Ib study of brigimadlin for DDLPS, what was the approximate objective response rate?**
 - a. 0%
 - b. 8%
 - c. 19%**
 - d. 34%
 - e. 57%
- 5. The Phase II/III randomized open-label Brightline-1 study will evaluate the efficacy and safety of what pharmaceutical agents for DDLPS?**
 - a. Milademetan versus trabectedin
 - b. Brigimadlin versus doxorubicin**
 - c. MI-773 versus doxorubicin