Exploring Treatment Sequencing Strategies in Chronic Lymphocytic Leukemia

Multiple clinical, disease, and patient factors influence treatment selection and sequencing[1,2]

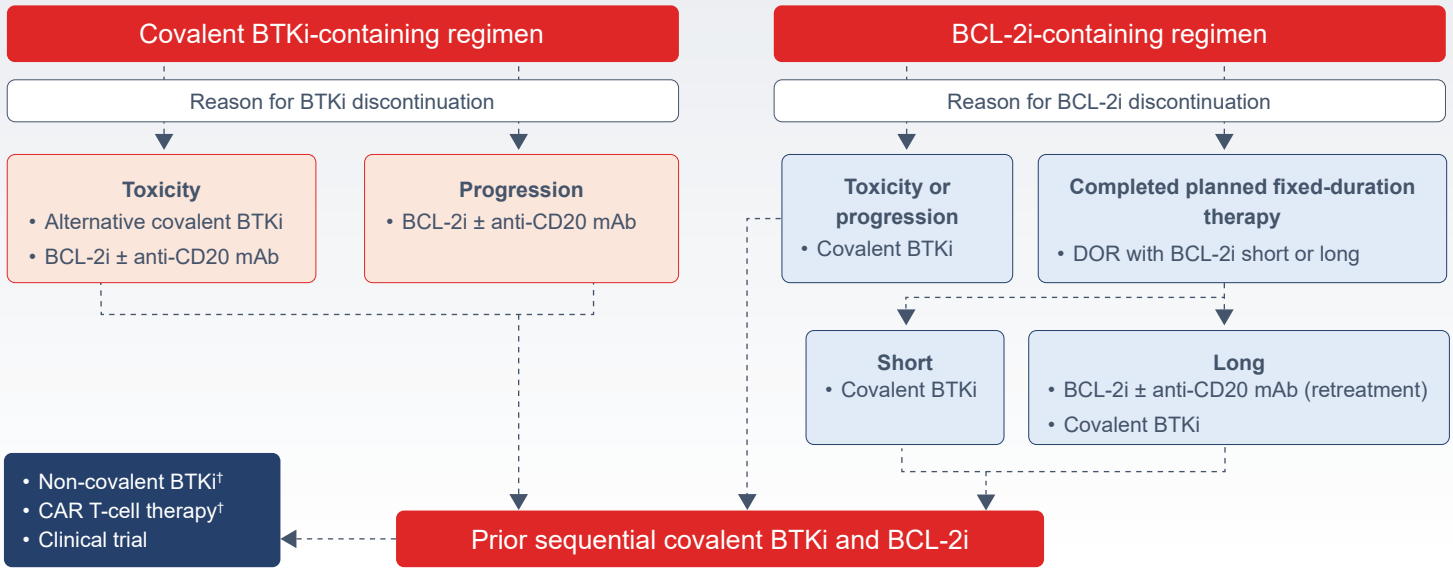
Medicines for the treatment of CLL[3,4]

Laboratory evaluations such as testing for del(17p)/*TP53* mutations and *IGHV* mutational status can help inform treatment decisions and predict likely response to therapy[5,6]

Signs of disease progression and/or treatment intolerance may indicate the need to switch therapies.[3] Switching between drug classes (eg, a BTKi and a BCL-2i) is a common strategy in the 2L setting[3]

Because CLL is a progressive disease, patients will likely require multiple therapies through the course of the disease to continue to achieve a response[1]

Treatment Sequencing Approaches for Previously Treated Patients With CLL[3,4]



Treatment requirements and patient preferences can influence treatment selection[3,4,7]

Patients may perceive different administration routes as more or less convenient[4]

Due to having similar resistance mechanisms, sequencing covalent BTK inhibitors after progression should be avoided. A non-covalent BTKi can be used after progression on a covalent BTKi[3]

Patients may have preferences between continuous oral therapy administered at home vs fixed-duration oral and IV therapy with frequent office visits and increased monitoring[4,7]

*Due to its inferior efficacy when compared to targeted therapies, CIT is appropriate only for select patients.[3,4]
†Optimal sequencing of CAR T-cell therapy and non-covalent BTKi therapy is yet to be determined.[3,4]

1L, first line; 2L, second line; 3L+, third line or greater; BCL-2i, B-cell lymphoma 2 inhibitor; BTKi, Bruton's tyrosine kinase inhibitor; CAR, chimeric antigen receptor; CIT, chemoimmunotherapy; CLL, chronic lymphocytic leukemia; DOR, duration of response; *IGHV*, immunoglobulin heavy chain variable region genes; mAb, monoclonal antibody.

1. Odetola O, Ma S. *Curr Hematol Malig Rep*. 2023;18(5):130-143. **2.** Hallek M, Al-Sawaf O. *Am J Hematol.* 2021;96(12):1679-1705. **3.** Shadman M. *JAMA*. 2023;329(11):918-932. **4.** Jain N. *Lancet*. 2024;404(10453):694-706. **5.** Heerema NA, et al. *Haematologica*. 2021;106(6):1608-1615. **6.** Campo E, et al. *Haematologica*. 2018;103(12):1956-1968. **7.** González-Gascón-Y-Marín I, et al. *Cancers (Basel)*. 2023;15(17):4391.