

3. RELAPSED/REFRACTORY MULTIPLE MYELOMA

## MANAGEMENT OF NEUROLOGICAL TOXICITIES OF PATIENTS TREATED WITH IMMUNOTHERAPIES FOR MULTIPLE MYELOMA: A SINGLE-CENTER REAL WORLD EXPERIENCE

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T cell redirecting therapies (TCRt) have dramatically improved the prognosis of relapsed/refractory multiple myeloma (RRMM) patients (pts), but they are also burdened by novel toxicities, among which neurological ones such as immune cell associated neurotoxicity syndrome (ICANS), headache, and neuropathies, not clearly reported in registration trials, for which real-world data on frequency and management are also lacking. We address this gap by reporting on the recording and management of neurological side effects of TCRt in our institution, where a specific protocol has been established requiring pts to undergo neurological examinations, brain imaging and electroencephalographies before and after chimeric antigen receptor T (CAR T) cell therapy, or a neurological evaluation and an electromyography before bispecific antibody (bsAb) therapy. Between 2022 and 2025, 59 pts have received TCRt outside of clinical trials, be it idecabtagene vicleucel (ide cel) (19), teclistamab (11), talquetamab (17) or elranatamab (12). After a median follow up of 9 months (range 2-39), 32 (54%) of them are still on therapy, with 19 (32%) off therapy due to progressive disease (PD), 6 (10%) to toxicity, and 2 to other reasons. One pt each receiving ide cel, teclistamab and elranatamab developed respectively G1, G1 and G2 ICANS; all instances occurred within the first ten days of therapy and were managed by delaying the following bsAb dose if appli-

cable and initiating dexamethasone in the G2 case, with resolution within three days in all cases. G1 headache was reported by 5 pts (1/19 receiving ide cel, 2/11 teclistamab, 1/17 talquetamab) and treated with painkillers. Finally, 7 pts (5/12 receiving elranatamab, 1/17 talquetamab, 1/11 teclistamab) experienced a G1 or G2 worsening or new onset of peripheral neuropathy, with sensory and/or motor disturbances. One G2 case of sensory neuropathy was managed with clonazepam, due to pain and restless legs syndrome, with no change to bsAb therapy; further three cases of G2 motor neuropathy slowly improved after withdrawing treatment, although only in one case it was possible to restart therapy at a reduced schedule, while the other two pts never resumed treatment. Finally, other two pts with G1 sensory disturbances showed an improvement when the bsAb was either suspended due to PD or reduced in frequency due to a good response. Overall, our centre's experience confirms that TCRt represent a manageable option for RRMM pts, with an overall low incidence of neurological toxicities which nonetheless require prompt management and can result in the need to either reduce the frequency or withdraw treatment, and as such should not be discounted; further, multi-centre studies could be extremely useful in shedding further light on their frequency and risk factors and the best way to reach a prompt diagnosis and an appropriate therapy.