Utilizing UL97 Resistant Codon Specificities to Guide Pharmacotherapy Treatment Decisions in Resistant Cytomegalovirus Infections

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(ANC)>500 was necessary. However, this exacerbated the patient's sickle cell disease causing hip and bone pain. A transition to VGCV 900 mg twice daily for the maintenance phase of treatment was initiated for one month.

Second episode

With the resolution of viral replication, VGCV was decreased to prophylaxis dosing at 900 mg daily to mitigate bone marrow suppression. Unfortunately, this resulted in reoccurrence of CMV viremia. Despite increasing VGCV back to treatment dose 900 mg twice daily, CMV titers continued to rise and the patient was reinitiated on high dose IV GCV. Repeat resistance testing (Sample 2) indicated unchanged UL97 mutation. Due to the antiviral potential of mammalian target of rapamycin inhibitors (mTOR-I), the patient was converted from TAC to everolimus. Despite successful conversion, the patient experienced mTOR-I induced anemia with no improvement in viral loads thus was re-initiated on TAC. Due to low quality of life from the continued need for G-CSF and periodic transfusions due to GCV bone marrow suppression, foscarnet (90 mg/kg IV every 12 hours) was initiated in an attempt to avoid GCV side effects, while acknowledging the risk of nephrotoxicity with foscarnet therapy. The patient completed 28 days of FOS therapy with complete resolution of CMV viremia 5 days into therapy. This was at the cost of serum creatinine increase from a baseline of 1.0 mg/dL to a peak of 2.67 mg/dL and subsequent decline to a new baseline of 1.5 mg/dL and he was to continue VGCV treatment dose indefinitely.

Third episode

The patient became viremic 44 days after viral clearance from the second episode. Repeat resistance panel (Sample 3) results were unchanged. Given the patient's complicated history with ganciclovir and severe renal impairment demonstrated with foscarnet, the decision was made to proceed with compassionate use maribavir (SHP620, Shire Pharmaceuticals). The patient did not qualify for current phase 2 studies due to severe neutropenia, current sickle cell crisis, and acute

di f er in their cellular activation, which lends itself to di f erent levels of

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