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INTENSIFICATION OF A FAILING REGIMEN WITH AZT MAY CAUSE SUSTAINED VIROLOGICAL SUPPRESSION IN THE PRESENCE OF THE K65R MUTATION

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BACKGROUND: The K65R resistance mutation limits the number of NRTI options. However it has the potential to decrease phenotypic AZT-resistance and almost never occurs concurrently with TAMS. Patients with a K65R mutation usually maintain AZT/D4T as options.

METHODS: Three patients experienced virological failure associated with the K65R and other resistance mutations. The three regimens were then intensified with AZT without changing any other drugs, despite genotypic resistance.

RESULTS: None of the patients had a past history of TAMS. Two of the three patients were taking a triple nucleoside regimen. Patient 1 was RTI-naïve and then failed with ABC/ddI/3TC. Patient 2 who was ART-experienced, was never able to attain undetectable viraemia in 4 years of treatment. She failed on a triple regimen of ABC/ddI/TDF. Genotype resistance testing at VF showed K65R, L74V, M184V, and the Y115F mutations to be present in both patients. Patient 3, ART-experienced, started a regimen of TDF, 3TC, and NVP. After 18 months with viral load <50 copies/ml, he experienced a rebound above detectable limits which continued for the next 6 months. Genotype testing showed the K65R, G190S and Y181C mutations. All three patients had K65R but no TAMS. Their failing regimens were intensified with AZT and the other drugs were kept despite genotype resistance mutations to all the drugs in their regimens. All three patients had an immediate viral load reduction to undetectable levels within 4 weeks: Patient 1 dropped more than 2 logs from 5300 copies/ml to <50 copies, patient 2 dropped greater than 3 logs from 48000 to <50 copies, and patient 3 had a 1.4 log reduction from 1130 to <50 copies/ml. Follow up time ranges from 7–16 months. All

three patients have maintained their undetectable levels and all three remain on their regimens.

CONCLUSION: In these patients with the K65R mutation and no TAMS, AZT was added to regimens in which genotypic testing showed resistance to all their drugs. AZT was the only active drug, however, this addition was enough to achieve sustained viral load reductions to below 50 copies/ml.

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17

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