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Predictors of loss to follow up of patients enrolled on anti-retroviral therapy: A retrospective cohort study

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Introduction: There is a growing concern about the increasing rates of Loss to Follow up (LTFU) among people who are on HIV/AIDS treatment programs. It is more common in resource poor settings. However little is known about the time to LTFU and predictors after Anti-retroviral Therapy (ART) initiation in low resource settings including Ethiopia.

Method: Retrospective cohort study was employed among a total of 520 records of patients who were enrolled on antiretroviral therapy in Aksum St. Marry hospital. Baseline patient records were extracted from electronic and paper based medical records database and analyzed using Kaplan Meier survival and Cox proportional hazard model to identify the independent predictors of loss to follow up of patients on ART.

Result: Among 520 patients, 51(9.8%) were loss giving a LTFU rate of 8.2 per 100 person- years. From these LTFU, 21(41%) occurred within the first six months of ART initiation. The independent predictors of LTFU of patient were being smear positive pulmonary TB [Adj.HR (95% CI)=(2.05 (1.02, 4.12)], male gender [Adj. HR (95%CI)=(2.73 (1.31, 5.66)], regiment AZT-3TC-NVP [Adj. HR (95% CI)=(3.47 (1.02,11.83)] and weight ≥60kg [Adj. HR (95% CI)=(3.47 (1.02,11.83)].

Conclusion: Substantial magnitude of loss to follow up has been found among patients on ART which significantly affect the overall outcome of HIV/AIDS program of treatment. The independent predictors identified were TB smear positive, male gender, regiment AZT-3TC-NVP and lower weight. So, continuous and comprehensive follow up is necessary to minimize loss to follow up and optimize treatment outcome of people on ART.

Biography

Kidane Tadesse has completed his MSc from Mekelle University College of Health Sciences. He is member of different college committees related to quality of education. He is a Postgraduate program coordinator. He has published more than 10 papers in reputed journals.

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Signal peptide-binding drug as a selective CD4 receptor down-modulator with anti-HIVactivity

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Human Immunodeficiency Virus (HIV) uses the human CD4 protein (hCD4) as the primary surface receptor for attachment and infection of host cells such as $CD4^{+}$Th-lymphocytes. This hCD4 receptor dependency can be exploited in an antiviral strategy: Removal of the CD4 receptor from the cell surface will prevent HIV infection of target cells and viral replication. In eukaryotic cells, surface expression of most transmembrane proteins is dependent on the presence of a hydrophobic N-terminal signal peptide (SP) on nascent proteins. It facilitate stargeting of the nascent proteins to the Sec61 translocon, a universally conserved protein-conducting channel in the ER-membrane, and subsequent insertion of the chain for translocation. Despite their common function, signal peptides have diverse primary sequences. Thus, drugs that recognize uniquesignal peptide sequences could be exploited to inhibit translocation of selected proteinsin to the ER and their expression at the cell surface. Previously, the small-molecule macrocycle CADA was identified as an antiviral drug with broad spectrum anti-HIV activity. It acts as a highly selective hCD4 expression down-modulator. Here we show that CADA inhibitsh CD4 biogenesis by preventing co-translational translocation of hCD4 to the ER lumen, both in cell culture andin a cell-free *in vitro* translation/translocation system. The activity of CADA maps to the signal peptide of hCD4 which represents the minimal sequence requested for full CADA sensitivity. Importantly, we could show direct binding between this SP and CADA through suface plasmon resonance (SPR). Further more, translocation inhibition by CADA causes the precursor protein to be routed to the cytosol for degradation. These findings demonstrate that a synthetic, cell-permeable small-molecule such as CADA can act as a signal peptide-binding drug to regulate the expression of specific target proteins by selective and reversible inhibition of protein translocation.

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