The activation status can dictate the fate of an HIV-infected CD4+ T cell. Infected cells with a low level of activation remain latent and do not produce virus, while cells with a higher level of activation are more productive and thus likely to transfer more virions to uninfected cells. How the activation status of infected cells affects HIV dynamics under antiretroviral therapy remains unclear. We develop a new mathematical model that structures the population of infected cells continuously according to their activation status. The effectiveness of antiretroviral drugs in blocking cell-to-cell viral transmission is assumed to decrease as the level of activation of infected cells increases because the more virions transferred from infected to uninfected cells, the less effectively the treatment is able to inhibit the transmission. The basic reproduction number of the model is shown to determine the existence and stability of the equilibria. This study also provides a new modeling framework to study the observations, such as the low viral load persistence, extremely slow decay of latently infected cells and transient viral load measurements above the detection limit, in HIV-infected patients during suppressive antiretroviral therapy. (Received September 15, 2020)