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Nucleolin relocalization associated with pre-lethal alterations of T cell morphology: redefining cell death in HIV infection

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Background

To redefine the causal link between cell cycle dysregulation of T lymphocytes and HIV induced cell death.

Methods

Intacellualar concentration of cell cycle regulatory proteins has been measured by western blot; nucleolin (C23) concentration and localization has been established by confocal microscopy; expression of surface proteins and ultrastructural membrane damage have been analyzed by flow cytometry and transmission electron microscopy, respectively.

Results

Here we demonstrate that circulating T lymphocytes, both CD4+ and CD8+, leave lymphoid tissues with diffused regressive lesions such as vacuolization, blebbing, nuclear evanescence and organelle swelling. Equally diffused are biochemical anomalies that accompany the overall disarrangement of cell structure, namely (i) fragmentation and diffusion into the cytoplasm of C23/nucleolin, the principal structural protein of the nucleus (ii) an accumulation of short - lived regulatory proteins (p16, p21 and p53), likely due to the progressive extinction of the ATP - ub - proteasome system and (iii) a decreased expression of membrane proteins.

Discussion

The HIV - induced demise of CD4 -T cells is thought to be a result of the execution of genetically programmed cell death that occurs in lymphoid tissue, where many resident T cells are chronically hyperactivated and primed for apoptosis. The pre-lethal lesions described here recapitulate a series of regressive events that occur in immune cells, when they grow at high mitotic activity in conditions of scarce ATP production.

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