

DETERMINATION OF HFE C282Y MUTATION AND ITS ASSOCIATION WITH THE IRON STATUS AND VIRAL LOAD IN HIV PATIENTS FROM REYNOSA, TAMAULIPAS.

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Background: The HFE protein has a fundamental role in iron homeostasis, the HFE C282Y mutation prevents the specific function of the protein, causing greater intestinal absorption of iron and intracellular accumulation. The HIV virus causes a disease that attacks the cells of the immune system, mainly CD 4 T lymphocytes inducing their destruction and immunosuppression of the patient. Some viruses have the ability to disrupt cellular metabolic processes during their own replication, such is the case of HIV-1, which is involved in alteration of iron metabolism resulting in an overload of iron.

Methods: An exploratory, descriptive, cross-sectional and prolective study was conducted, including 68 patients, ≥ 18 years old, HIV positive, attended at CAPASITS, through informed consent and application of an interview on lifestyle and health, were determined: blood pressure, anthropometric measures, CD₄ T cell count, viral load, and iron status (ferritin, iron and transferrin).

Results: 41 participants were male sex (60%) and 27 (40%) of the female sex, average of 38.22 (± 11.05 SD) years, with a BMI 25.30 (± 4.70 SD). The presence of the C282Y mutation was not detected, only the wild variant (100%) was identified. Patients with viral loads ≥ 40 copies/ml, were ruled out when relating viral load vs. serum ferritin ($r = 0.594$, $r^2 = 0.353$ and $p = 0.004$) with statistical significance, with a ferritin mean of 231.5 (± 216.04 SD).

Conclusions: The C282Y mutation of the HFE gene is not present in the study population, due to its low frequency, so it is not related to iron overload. Because there is a high viral load, serum ferritin levels will also increase, and this may be due to the fact that HIV in addition to interfering with the Iron metabolism induces alterations in the synthesis and regulation of the secretion of this protein.