Neuroendocrine due to hypothalamic dysfunction as a marker of Huntington's disease.

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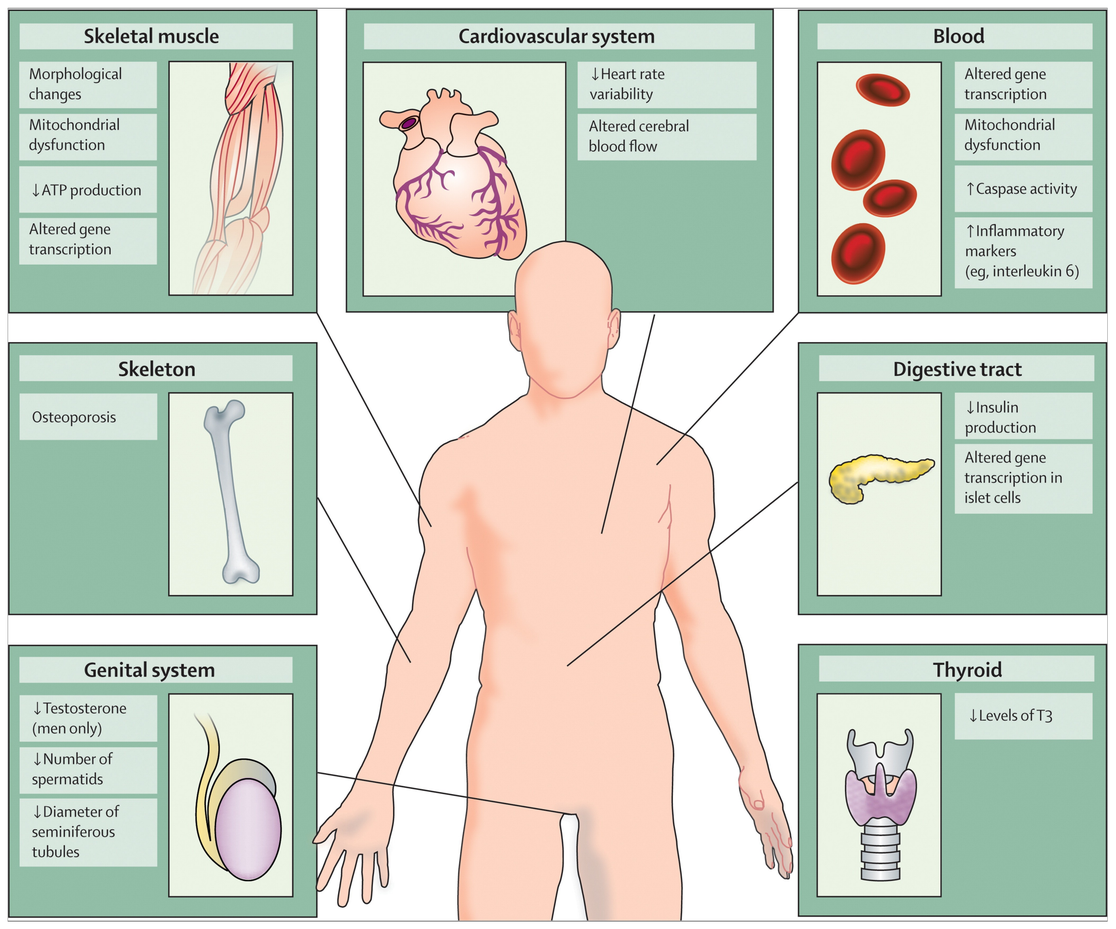
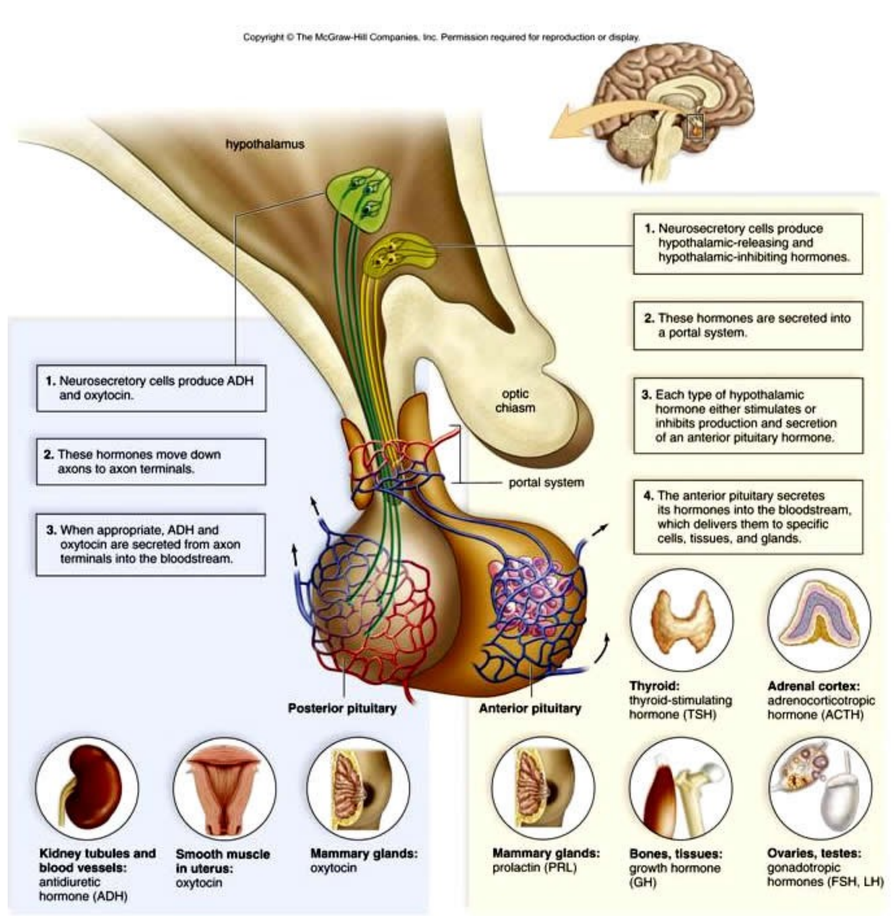
Aim: Huntington's disease (HD) is a hereditary neurodegenerative disorder characterized by cognitive, psychiatric, beavioural and motor disturbances. Neuropathology of HD showed intranuclear and cytoplasmic inclusions of huntingtin aggregates, in striatum and cerebral cortex but also with a widespread pathology in different organs. However, hypothalamic atrophy occurs at early stages of HD with loss of orexin- and somatostatin-containing cell populations. Several symptoms of HD such as sleep disturbances, alterations in circadian rhythm, and weight loss may be due to hypothalamic dysfunction. These features form a substantial contribution to disease burden in HD patients and appear to be accompanied by a number of neuroendocrine and metabolic changes. Aim of the study is to evaluate neuroendocrine changes in a population of HD patients evaluated at the Neurology Clinic "ASST Spedali Civili di Brescia".



Material: We evaluate sierological markers of neuroendocrine changes in 20 HD patients with weight decrease history during the clinical follow up at Neurology Clinic "ASST Spedali Civili di Brescia" in the last 5 years.

Methods: clinical observation of weight changes needs to explore possible endocrine disfunctions in HD patients. These subjects were suggest to underwent blood sample to evaluate thyroid and hypothalamic impairment.



Results: 3 patients showed highest level of sieric PRL; 2 patients was found with thyroid value reductions in fT4; somatomedin (IGF-1) was increased in 4 patients. All patients evaluated, showed a significant and progressive weight loss in almost 2 follow up. Interactions with therapy were considered as possible cause of weight loss but no link was found for these mechanisms. PRL increase was not secondary to neuroleptic treatments. Data of the others patients need to be re-assessed with a second sample because of results near to normal values.

Discussion: Findings of neuroendocrine and metabolic alterations in HD is faraway to be fully understood but the clinical relevance of those diseases need to be clearly defined in a multidimensional way to evaluate and treat HD patients.

Conclusion: These findings indicate that neuroendocrine changes due to hypothalamic atophy in patients suffering from disease may be a pathology marker to be assessed when evaluate HD patients.

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