

Conclusions: The level of Galectin-3 and ST-2 are associated significantly with OSAS severity in comparison with NT-proBNP and could be considered as a sensitive diagnostic and prognostic marker for cardiovascular abnormalities in patients with OSAS and HF with preserved ejection fraction.

Narcolepsy

TYPE 1 NARCOLEPSY IN ANTI-HU ANTIBODIES MEDIATED ENCEPHALITIS: A CASE REPORT

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Introduction: Type 1 Narcolepsy (NT1) is a central hypersomnia due to an autoimmune hypothalamic hypocretin neurons destruction with onset in predisposed subjects carrying the HLA DQB1*06:02 haplotype, mostly during paediatric age. There are few cases of NT1 related to central nervous system (CNS) structural or functional lesions.

Materials and methods: The case at issue reports for the first time a patient with secondary narcolepsy associated with anti-Hu antibodies, in the context of paraneoplastic encephalitis. The patient was an 85-year-old male, previous heavy smoker with many cardiovascular risk factors. His history begun with a two-days-lasting episode of aggression and hallucinations, followed by persistent generalized muscular weakness to the extent that in few months he was confined in a wheelchair. He also had brief (seconds) and frequent episodes of facial twitching and grimaces, ptosis, slurred speech and increase of weakness involving mainly the arms, with preserved consciousness, mainly triggered by emotional or stressful conditions. He also displayed continuous tendency to fall asleep and disrupted nocturnal sleep. Episodes were mislabeled as epileptic seizures and he was started on antiepileptic drugs, without benefit.

Results: Neurological examination revealed subcontinuous fluctuations in muscle tone with ptosis, facial grimaces, muscle sagging of upper limbs. The 24-hour video-polysomnography documented several sleep episodes with frequent sleep onset in REM periods (SOREMPs) during daytime and nighttime sleep with SOREMP and REM sleep behavior disorder. The multiple sleep latency test showed a pathological mean sleep latency with five SOREMPs. Several and subcontinuous focal and generalized cataplectic episodes were video-documented, leading to a diagnosis of NT1 (figure 1). HLA typing was negative for HLA-DR15-DQB1*0602 antigens and dosage of CSF hcrt-1 level was of 146.83 pg/mL (normal > 200 pg/mL). Due to the atypical age of onset and to the "HLA negativity", the patient was therefore investigated for secondary causes of NT1. Serological and biochemical analyses disclosed positivity for anti-neuronal nuclear antibody, type 1, ANNA 1 (anti Hu) and Total body CT and fluorodeoxyglucose positron emission tomography scans showed a nodular hilar-perihilar lung formation, with features characteristic of malignancy (figure 2). Accordingly, the patient received the diagnosis of secondary NT1 related to anti-Hu antibodies and neoplasm of lung.

Conclusions: The atypical age at onset along with the biochemical profile (discrete HLA negativity and the slightly low CSF hcrt-1 levels) may be telltale-signs to suspect a secondary nature of NT1 symptoms and signs and to undergo a prompt and extensive work-up searching for secondary causes. It is likely that Hu-autoantibodies have targeted the lateral hypothalamus, where hypocretinergic neurons are located, along with other structures of the CNS explaining the additional symptoms. Early recognition of paraneoplastic encephalomyelitis and prompt antitumor treatment are indeed pivotal in order to stabilize underlying disease and the related symptoms.

Basic Research

OBSTRUCTIVE SLEEP APNEA AND METABOLIC SYNDROME: IS THERE A CORRELATION?

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Introduction: Obstructive sleep apnea (OSA) and metabolic syndrome (MS) are common disorders with an escalating prevalence and systemic consequences. OSA is defined as recurrent episodes of complete or partial upper airway closure during sleeping. MS is a cluster of factors including obesity, hyperglycemia, hypertension, dyslipidemic.

Materials and methods: The aim of this study was to evaluate the association between OSA and MS.

To this end, we performed a retrospective study of patients who underwent sleep study in Constanta Sleep Disorders Center, between 2010–2016. The patient group consisted of 108 adults (79 male and 29 females). All patients underwent polygraphy, blood sampling and measurement of anthropometric variables. OSA was considered present when AHI > 15. MS was defined according to the National Heart, Lung, and Blood Institute (NHLBI) and the American Heart Association (AHA) guidelines.

Results: Out of a total of 108 patients with OSA, 62 (57.4%) were found to have MS. All of the separate components of metabolic syndrome were common in our patients, but the diagnosis was made based on three (56.4%), four or all criteria (43.5%). Furthermore, all the anthropometric variables were associated with MS. The prevalence of MS among OSA patients increased with increasing AHI. Obesity was a strong link to both, in particular visceral obesity for MS and enlargement of soft tissue structure within and surrounding the airway for OSA.

Conclusions: OSA and MS are significant cardiovascular risk factors that act synergistically. Thus, this positive association between these two pathologies could disclose a role for OSA screening in patient with metabolic abnormalities.

Basic Research

REM-SLEEP DEPRIVATION PREDISPOSES NEUTROPHILIC LUNG INFLAMMATION IN ALLERGIC MICE

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Introduction: Sleep duration exerts an important influence on host immune system homeostasis. Although different studies associated sleep deprivation with systemic inflammatory changes, the effect of sleep duration on the pathology of allergic chronic diseases is poorly understood. Asthma is a complex chronic disease that affects around 235 million people worldwide. Because asthmatic individuals experience disturbed sleep periods due to breathlessness, nighttime coughing, and wheezing, the disease itself impairs sleep quality. In this work, we took advantage of a mouse model to evaluate the influence of sleep deprivation (SD) on allergen-induced pulmonary inflammation.

Materials and methods: Ovalbumin (OVA)-sensitized C57Bl/6, IL-17RA deficient mice (IL-17RA^{-/-}) were exposed to a first set of intranasal OVA challenge under REM sleep deprivation (SD) or healthy sleep (HS) condition followed by a second set of OVA challenge, one week apart. Analysis of cellular profile in the lungs, spleen and lymph nodes (LNs) was performed by flow cytometry. In addition, mice were subjected to corticoid treatment using dexamethasone.

Results: OVA-sensitized SD mice developed a more severe airway inflammation than the HS allergic group. The analysis of lung parenchyma revealed that the inflammation in SD allergic mice was marked by the influx of neutrophils (mainly) and eosinophils and secretion of IL-6, TNF- α , and IL-17, in contrast to the classic eosinophilic inflammation and IL-4 production observed in HS allergic mice. The same cytokine profile was observed in ex vivo culture of cervical lymph nodes cells and splenocytes, indicating that in allergic mice SD polarized the immune responses towards a pro-inflammatory Th17 profile. This idea is supported by the fact that disruption of IL-17 signaling (IL-17RA^{-/-}) prevented airway neutrophilia in SD allergic mice. Furthermore, SD allergic mice became refractory to corticoid treatment, in contrast to HS allergic group.

Conclusions: Collectively, our data show that sleep duration plays an important role in the progression of allergen-induced mild/moderate asthma-like eosinophilic lung inflammation to moderate/severe corticoid refractory neutrophilic manifestation.