### (212) Complex regional pain syndrome due to carpal tunnel syndrome: a case report and review of the literature

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Complex regional pain syndrome (CRPS) type II is an unexpected complication of carpal tunnel syndrome (CTS), with only 3 reported cases. We present a case of CRPS secondary to CTS in the setting of peripheral neuropathy (PN) and vitamin deficiency. A 41 year-old male painter with chronic alcoholism presented with allodynia, dysesthesia, erythema and atrophy of both hands and forearms. Over 3 months, his symptoms progressed from paresthesias in the median nerve distribution of the right hand to allodynia, reduced mobility, and edema of the hand and wrist. He developed similar symptoms in his left hand and wrist one month later. Physical exam revealed allodynia in the median nerve distribution with vasomotor and sudomotor changes of the wrists and hands. Electromyography (EMG) confirmed CTS, but also revealed a diffuse axonal polyneuropathy. Serum thiamine, folate, and vitamin B12 levels were also reduced. Initial treatment with a steroid taper and gabapentin were ineffective. Subsequent treatment with pregabalin, vitamin supplementation, occupational therapy began to alleviate his symptoms. CRPS is more commonly seen after median nerve injury due to trauma or carpal tunnel release, although it may also be seen with untreated CTS. Additional considerations in this patient are his concomitant peripheral neuropathy and vitamin deficiencies. His presenting complaints are unlikely due to the PN alone, as his lower extremities showed no symptoms of CRPS, although it may have predisposed him to develop CTS (i.e. double crush). His improvement after vitamin supplementation may underscore their role in reducing the exaggerated inflammatory component of CRPS, and their role in the treatment of CRPS warrants further study.

## *B06 Headache*

### (213) Migraine, Chiari malformation and cervical muscles

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Chiari 1 (Ch1) malformation is often associated with headaches and/or neck pain. Both Ch1 patients and typical migraineurs may complain of neck and/or trapezius pain. Controversy exists as to the relation of Ch1 and migraine. Exams may reveal areas of neck and trapezius muscle spasm (MusSpsm), tenderness and trigger points (TrPs) in both conditions. Evaluation of 10 Ch1 patients (Group A) with headaches showed: 6 had typical Ch1 posterior headaches and also separate migraine headaches; 1 had only migraine headaches; 7/10 had cervical MusSpsm (5/7 with migraine, 2/3 without migraine). Evaluation of a separate group of 18 patients (Group B) with frequent migraine, neck pain and cervical and/or trapezius MusSpsm on exam revealed: 6 had acute ipsilateral neck pain as part of the migraine episode; 11 noted worsening of a separate chronic neck muscle pain during migraines; in 7, compression of ipsilateral neck muscles intensified a current migraine or reproduced migraine symptoms. Physical therapy and/or TrP injections improved migraine in 14 of 17 treated Group B patients. Of the 6 whose neck pain was part of their migraine episode, 3 experienced simultaneous relief of both headache and neck pain with triptans or beta-blockers. These studies demonstrate that MusSpsm can intensify migraine. Treatment of spasm can improve migraine management. Simultaneous occurrence of neck pain and migraine in 6 patients and simultaneous resolution of both with medication confirms that neck pain can be an integral part of a migraine episode. Migraine and/or MusSpsm can contribute to headaches in Ch1 patients. Treatment of MusSpsm and migraine may improve headache control in Ch1 patients. These types of treatments should be considered before implementing surgical decompression for Ch1 patients with severe headaches. This project was supported by NINDS intramural research funds.

## *B07 Joint and Muscle Pain*

### (214) Risk factors for persistent pain after motor vehicle collision differ between litigants and non-litigants

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Worse pain outcomes are observed among individuals seeking monetary compensation after motor vehicle collision (MVC), but whether the etiology of persistent pain among such litigants differs from non-litigants remains poorly understood. One commonly used method for gaining insights into etiology is to evaluate risk factors for disease development. In this prospective observational study, we compared predictors of persistent pain after MVC among litigants and non-litigants. European Americans ≥18 years of age presenting to the emergency department (ED) within 24 hours of MVC who did not have a fracture or injury requiring hospital admission were enrolled. Baseline ED assessment included an evaluation of participant sociodemographic characteristics, pre-MVC health characteristics, MVC history, and participant cognitions and symptoms in the ED. Six week telephone follow-up evaluation assessed litigation status and neck pain intensity during the past week (0-10 NRS), scores ≥4 were defined as moderate/severe neck pain (MSNP). Candidate predictors of six week MSNP were assessed via logistic regression; significance levels were determined using Bonferroni correction (p=0.00125). Six week follow up was obtained in 849/948 (90%) of enrolled participants, and 148/849 (17%) reported that they had hired a lawyer to sue for compensation ("litigants"). MSNP was reported by 95/148 (64%) of litigants and 199/711 (28%) of non-litigants six weeks after MVC. Female sex, increased ED pain severity, and increased ED somatic symptom burden predicted 6 week MSNP among both litigants and non-litigants. Among litigants, unique predictors of 6 week MSNP included not working full time, not having health insurance, and being a vehicle passenger vs. driver. Among non-litigants, unique predictors of 6 week MSNP included believing that the MVC was someone else's fault and increased participant estimate of time to recovery (assessed at ED evaluation). These findings suggest that the etiology of persistent pain in litigants and non-litigants may differ. Supported NIAMS R01AR056328.

### (215) Sleep-disordered breathing and pain sensitivity in knee osteoarthritis

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Obstructive sleep apnea is a common and underdiagnosed sleep disorder characterized by respiratory disturbance and reduced oxygen saturation of hemoglobin in red blood cells during sleep. Although sleep-disordered breathing is associated with increased pain sensitivity in individuals without chronic pain, preliminary evidence from our group and others suggests that sleep disordered breathing may be associated with reduced pain sensitivity in patients with temporomandibular disorder pain and those with chronic headache. The present study investigated the association of overnight oxygen saturation and pain sensitivity in a sample of patients with knee osteoarthritis who were free of chronic head and jaw pain. Older men (N = 44) and women (N = 93) with knee osteoarthritis underwent one night of ambulatory polysomnography (PSG), including pulse oximetry to measure oxygen saturation. Following PSG, participants engaged in multimodal quantitative sensory testing to evaluate pain sensitivity. Regression models controlling for age, race, sex, body mass index, systolic blood pressure, insomnia severity, and apnea-hypopnea index revealed that lower oxygen saturation predicted significantly lower clinical pain ($p < .01$), higher thermal pain threshold at the forearm ($p < .01$) and patella ($p < .05$), marginally higher pressure pain threshold at the trapezius ($p = .08$), and lower suprathreshold thermal pain sensitivity ($p < .05$). In contrast, lower oxygen saturation predicted greater mechanical temporal summation at the patella and finger (ps < .05). Oxygen saturation was not associated with conditioned pain modulation. The results partially support limited previous evidence for respiratory disturbance-induced hypoalgesia in chronic pain, which may result from altered baroreceptor activity. But opposing effects on mechanical temporal summation suggest that sleep disordered breathing may differentially alter the activity of C versus Aδ fibers. Future studies of chronic musculoskeletal pain should employ a multimodal assessment of sleep disordered breathing to help understand individual differences in pain sensitivity.