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(240) Abdominal cutaneous nerve entrapment syndrome in a 64 year-old woman after undergoing bilateral breast reconstruction with microvascular DIEP flap: a case report

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A 64 year-old Caucasian female with a history of bilateral mastectomy and breast reconstruction with microvascular deep inferior epigastric perforator (DIEP) flap for breast cancer presented to our clinic with chronic abdominal pain. The pain started 2 years subsequent to the breast reconstruction surgery. The patient had gone through an extensive diagnostic workup for her pain complaints, which included a CT scan, ultrasound, and visits to multiple subspecialty providers. The workup was negative prior to her consultation in our clinic. The patient's pain was localized in the left lower quadrant of the abdomen to a point area lateral to the umbilicus. The pain occasionally radiated to the left groin and was characterized as a throbbing, tender, and heavy sensation. Exacerbating factors included standing or sitting for long periods of time, exercise, coughing, and sneezing. Alleviating factors included lying down and bowel movements. On examination, the patient had point tenderness to an area left of the umbilicus on the lateral border of the rectus abdominis muscle. A presumptive diagnosis of abdominal cutaneous nerve entrapment syndrome (ACNES) was established. A diagnostic and therapeutic ultrasound guided-injection of the lateral border of the rectus sheath was performed. This procedure led to immediate and complete relief of the pain, confirming the diagnosis of abdominal cutaneous nerve entrapment. ACNES is a commonly missed cause of chronic abdominal pain. It is a result of the entrapment of the cutaneous nerve exiting lateral to the rectus abdominis muscle. Presentation of ACNES is typically localized sharp pain in the abdominal wall, reproducible with palpation. Patients may demonstrate a positive Carnett's sign. Treatment for ACNES involves injection of local anesthetic to the area of nerve compression. This case is the first report of ACNES as a complication of bilateral breast reconstruction with microvascular DIEP flap surgery.

B21 Urogenital Pain

(241) Segmental hyperalgesia to mechanical stimulus in interstitial cystitis/bladder pain syndrome - evidence of central sensitization

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The objective is to investigate if interstitial cystitis/bladder pain syndrome (IC/BPS) subjects demonstrate mechanical or thermal hyperalgesia, and whether the hyperalgesia is segmental (along the areas of referred pain) or generalized (global). 10 female IC/BPS subjects and 10 age-matched female controls without co-morbid fibromyalgia or narcotic use were recruited for quantitative sensory testing. Pressure pain was measured using a hand-held pressure algometer with a 1 cm^2 flat probe. Heat pain was measured using a 9cm^2 Peltier thermode and the Pathway system. Pressure pain thresholds and heat pain thresholds were measured using ascending stimulation intensity. Suprathreshold pain rating (on a 0-10 VAS) was recorded using a random presentation of a fixed pressure (2 kg, 4 kg) or a fixed temperature (43°C, 45°C). The VAS pain rated by female IC/BPS subjects was significantly higher than the VAS pain rated by female control subjects when a fixed mechanical pressure (2 kg or 4 kg) was applied to the suprapubic (T11) area (p=0.028). There was an up-shift of the stimulus-response curve, which corresponded to the presence of mechanical hyperalgesia in the suprapubic area in IC/BPS. However, the VAS pain rated by IC/BPS subjects was not different from those rated by controls when a fixed pressure was applied at the other body sites (T1 arm, L4 leg, S2-3 sacral). No difference in VAS pain rating was noted when a fixed heat stimulus (35°C or 37°C) was applied to any of the body sites tested (T1, T11, L4, S2). There was no difference in pressure pain thresholds or thermal pain thresholds between IC/BPS and controls. In conclusion, female subjects with IC/BPS without co-morbid fibromyalgia showed segmental hyperalgesia to mechanical pressure stimulation in the suprapubic area (T10-T12). This segmental hyperalgesia (along the areas of referred pain) may be explained in part by spinal central sensitization.

B24 Other

(242) Situational, dispositional and disease-specific catastrophizing in sickle cell disease

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Sickle cell disease is associated with severe episodic and chronic pain, which patients often report to be undertreated. Pain-related catastrophizing is a risk factor for increased pain and poorer psychosocial outcomes among various pain populations. Patients with sickle cell disease have higher dispositional catastrophizing scores compared to other chronic pain populations. However, unlike other pain populations, prior studies have not found a significant relationship between catastrophizing and sickle cell pain after controlling for relevant covariates such as depression. Recent research suggests that pain-related catastrophizing may best be assessed using multiple anchors, including specific events (e.g., laboratory pain stimulation) or contexts (e.g., general clinical pain vs. current clinical pain). Here, we measure dispositional catastrophizing (in response to pain experienced in one's daily life), situational catastrophizing (in response to painful stimuli in the lab), and disease-specific catastrophizing (in response to sickle cell pain) among 84 (58 f) patients with sickle cell disease. Sickle cell disease-specific catastrophizing was moderately correlated with dispositional and situational catastrophizing, whereas situational and dispositional catastrophizing were not correlated. This suggests that patients differentiate their sickle cell pain from other pain, and that pain-related catastrophizing differs across contexts. Importantly, catastrophizing was associated with greater pain when measures corresponded to the type of pain being assessed. Sickle cell disease-specific catastrophizing was associated with sickle cell pain and pain-related interference, dispositional catastrophizing was marginally associated with pain-related interference, and situational catastrophizing was associated with sensitivity to acute experimental stimuli. These findings remain after controlling for relevant covariates (e.g. age, sex, education, and depression). These results suggest that if a disease-specific catastrophizing scale is used - one in which the patient is asked to reference sickle cell pain - catastrophizing is significantly correlated with clinical pain among patients with sickle cell disease.

(243) Aberrant resting state brain connectivity in sickle cell patients with frequent pain

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The objective of present study was to explore if sickle cell disease (SCD) patients with and without history of frequent pain have differences in connectivity in the brain regions implicated in perception and modulation of pain. Eight pain and 4 non-pain SCD patients underwent resting state functional magnetic resonance imaging of the brain. We used a region of interest (ROI) or "seed" based approach using Statistical Parametric Mapping (SPM8). Five minutes of resting fMRI data were collected on a GE 3T scanner using a T2*-W EPI BOLD pulse sequence (repetition time [TR]/echo time [TE] = 2000/30ms, 150 volumes, voxel size=3.75x3.75x3.3). Preprocessing of fMRI data was performed using the SPM8 (Wellcome Department of Cognitive Neurology, London, UK) software package running under MATLAB. Group ROI analyses were performed using the functional connectivity toolbox *Conn v. 13* (Cognitive and Affective Neuroscience Laboratory, Massachusetts Institute of Technology, Cambridge, USA). Results from the whole brain between group intrinsic connectivity analysis showed SCD patients without history of frequent pain had greater connectivity between the right anterior thalamus (seed) to the red nucleus and also to anterior cingulate cortex (FWE $p < 0.05$ cluster level corrected); and the right mid insula cortex (seed) to left periaqueductal gray. The SCD patients with frequent pain showed greater connectivity between the left mid insula cortex (seed) to left cerebellum (FWE $p < 0.05$ cluster level corrected); and the right amygdala (seed) to left inferior frontal gyrus (FWE $p < 0.05$ cluster level corrected).We conclude that SCD patients who do not experience frequent pain may have increased anti-nociception activity due to increased connectivity to the periaqueductal gray. SCD patients with frequent pain may also have aberrant connectivity patterns that may be similar to "central" pain states. Better powered studies with larger samples are needed to confirm this pilot study.