ASSESSMENT OF CENTRAL PAIN PROCESSING AND AUTONOMIC RESPONSES IN WOMEN WITH PROVOKED VESTIBULODYNIA

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INTRODUCTION / AIM

Vulvodynia is a prevalent and highly debilitating health problem affecting up to 16% of women. Provoked vestibulodynia (PVD) is the most common form of vulvodynia and is characterized as a burning and cutting pain sensation localized at the entry of the vagina (vestibule) when a pressure is applied or a vaginal penetration attempted. Despite the serious deleterious repercussions, the pathophysiology of PVD still remains poorly understood. Peripheral pain mechanisms in the vestibule area have been suggested to be implicated in PVD because lower pain thresholds and nerve hyperplasia were found in women with PVD. However, recent studies suggest that the pathophysiology is not limited to the vestibule area but also involves central pain processing alterations. In support of such central alterations, women with PVD have widespread pain suggesting a potential deficit of diffuse inhibitory controls (Conditioned Pain Modulation CPM). Furthermore, the implication of the autonomic nervous system (ANS) was shown to be altered in patients with other pathologies with widespread pain such as fibromyalgia, irritable bowel syndrome and chronic prostatitis and to interfere with the endogenous inhibitory and excitatory pain processing.

Aims: To study and compare the implication of endogenous inhibitory and excitatory mechanisms as well as the reactivity of the autonomic nervous system in women with and without PVD.

METHODS

Thirty women with PVD and 39 asymptomatic controls, paired for age (18-45 years old), participated in this study. Women were included in the PVD group after having their diagnosis confirmed by a gynecologist from our team following a standardised protocol. Endogenous excitatory and inhibitory mechanisms and SNA reactivity were measured during a 2h-assessment session. The evaluation by a trained assessor included an experimental paradigm using a thermal stimulus (Stimulus Test), before and after a cold pressor test (CPT - Conditioning Stimulus - CS). The temperature of the thermode was maintained constant at 50% of the pain tolerance for 2 minutes while the participants rated their pain continuously using a CoVAS (computerized analog scale). The cold pressor test (2 minutes immersion of the forearm in 10°C water) was used to activate the descending inhibitory mechanisms. Excitatory mechanisms were evaluated by analysing the temporal summation during the 2 min of sustained heat with the thermode (central sensitization). The inhibitory mechanisms were measured by comparing the heat pain evaluation before and after the CPT whereas a reduction of pain indicates effective inhibition pathways. The ANS reactivity was studied by measuring heart rate variability and blood pressure.
at baseline (at rest, before the testing procedures) and during the CPT (painful procedure). The comparison of the two values allowed us to evaluate the reactivity of the ANS during the conditioning stimulus. Student t-tests were used to compare the two groups.

RESULTS
A significant difference between women with and without PVD was found for central pain processing. Indeed, healthy women showed a better inhibitory system (mean CPM: 33% ± 29) compare to PVD women (mean CPM: 1% ± 73; t=2.52, p=0.014) when analyzing the 2 minutes stimulations. Moreover, PVD women perceived the 2 minutes cold pressor immersion as more painful (PVD: 86.67% ± 19; HW: 65.64% ± 20.20; t=4.36, p<0.001) and more unpleasant (PVD: 92.5% ± 10.97; HW: 72.05% ± 23.16; t=4.46, p<0.001). Regarding the ANS, results also showed a difference between the two groups. Women with PVD showed a higher hearth rate increase (82.02 ± 10.03) during CPT than healthy women (76.82 ± 8.7) (t=2.27, p=0.027). This difference is supported by the difference in delta hearth rate (baseline HR – CPT HR) (PVD: -6.51 ± 5.71; HW: -2.91 ± 3.72; t=-3.1, p=0.003). Finally, PVD women also showed higher low frequency (sympathetic system) during baseline (PVD: 2510.21 ± 2361.74, HW: 1655.34 ± 1133.51; t=1.99, p=0.05).

DISCUSSION / CONCLUSIONS
Our results suggest an alteration of inhibitory but not excitatory central pain processing and autonomic responses in the pathophysiology of PVD. Moreover, PVD women demonstrated a higher hearth rate at rest as well as a higher increase during CPT. This ANS alteration is in line with findings suggesting a link between ANS, CPM and chronic pain. These results are clinically relevant suggesting that treatments of PVD should focus more on central pain processing rather than local pain.

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