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THE CONNECTION BETWEEN CENTRAL OBESITY AND THE DISTRIBUTION OF SPINAL ANESTHESIA IN FEMALE PATIENTS

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Abstract: Central obesity might influence the spread of spinal anesthesia in female patients. In a study, fifty-seven female patients undergoing spinal anesthesia were divided into either a central obesity group or a non-central obesity group. Following the induction of spinal anesthesia, several parameters were evaluated: maximal sensory blockade (primary outcome), time to maximal sensory block, maximum motor block, time to maximum motor block, time to L2 regression, and time to reach Bromage scale 0. Multiple linear regression analyses revealed that maximal sensory blockade was associated with central obesity (P = .004). Therefore, central obesity is linked to a more extensive spread of spinal anesthesia in female patients.

Key words: Central obesity, Spinal anesthesia, Sensory blockade, Motor block, Female patients

Introduction: Achieving an adequate level of blockade during spinal anesthesia is crucial. The distribution of a drug injected into the cerebrospinal fluid (CSF) is primarily influenced by the baricity of the anesthetic solution, the patient's position, and the dosage. Additionally, factors such as age, sex, intra-abdominal pressure, obesity, and CSF volume have been studied for their impact on the level of spinal blockade. Notably, women might experience a more cephalad spread of spinal anesthesia compared to men, potentially due to lower CSF density. The prevalence of obesity is rising across all age groups, including among older adults. There is a specific group of obese individuals, especially older adults in Asian countries, who have excessive visceral fat relative to their body mass index (BMI). This type of obesity, known as central obesity, is characterized by excess abdominal fat around the stomach and abdomen. It can occur even in individuals with a normal BMI and is accurately measured by waist circumference, which is a reliable indicator of abdominal fat. The relationship between central obesity and the level of spinal blockade remains unclear. Therefore, this study evaluated the connection between central obesity and the spread of spinal anesthesia in non-obese female patients.

Purpose of study: The purpose of the study was to evaluate the relationship between central obesity and the spread of spinal anesthesia in female patients,

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specifically examining how central obesity affects the extent and characteristics of sensory and motor blockade following spinal anesthesia.

Material and methods: Female patients over 60 years of age undergoing elective surgery with spinal anesthesia were included in this study. Exclusions were made for patients with a BMI less than 23 kg/m² or more than 27.5 kg/m², height less than 150 cm or more than 170 cm, or abnormal spinal anatomy. Waist circumference was measured twice at the narrowest point between the lower margin of the last palpable rib and the top of the iliac crest, and the average was used. Based on previous research indicating a waist circumference of 85 cm as the cutoff for central obesity in Korean women, patients were categorized into the central obesity group (CO) if their waist circumference was 85 cm or more, and the non-central obesity group (non-CO) if it was less than 85 cm.

Spinal anesthesia was administered at the L4−L5 intervertebral space in the lateral decubitus position using hyperbaric bupivacaine (12 mg, Marcaine Spinal Heavy 0.5%; AstraZeneca, Södertälje) and 20 μg of fentanyl. Post-procedure, patients were placed in the supine position on the operating table. Arterial pressure and heart rate (HR) were monitored every minute for the first 20 minutes post-anesthesia, then every 5 minutes during surgery. Sensory and motor blockade levels were assessed every 2 minutes for the first 30 minutes after spinal anesthesia induction, then every 30 minutes during surgery. After surgery, sensory and motor blockade levels were evaluated every 5 minutes until regression to L2 and Bromage scale 0. Sensory blockade was assessed using a loss of cold sensation to an alcohol swab, and motor blockade was evaluated using the modified Bromage scale (0 = able to raise legs above the table; 1 = able to flex knees, unable to raise legs; 2 = able to move feet only, unable to flex knees; 3 = no movement in the legs and feet). Block failure was defined as a maximal sensory level below T12 and a Bromage score below 2 at 20 minutes post-spinal block, leading to either another attempt at spinal anesthesia or conversion to general anesthesia, and such patients were excluded from the study. Hypotension, defined as systolic arterial pressure less than 90 mm Hg or mean arterial pressure (MAP) less than 80% of baseline, was treated with intravenous (IV) ephedrine. If more than 40 mg of ephedrine was required, IV phenylephrine was administered. Bradycardia (HR less than 40 beats/min) without hypotension was treated with IV atropine. Fluid management was at the discretion of the anesthesiologist. MAP, HR, ephedrine requirement, occurrence of nausea and vomiting, fluid requirement, and estimated blood loss were recorded for 60 minutes after spinal anesthesia induction. Patients, anesthesiologists, study investigators, and nurses were blinded to group allocation.

The primary outcome measured was maximal sensory blockade. Secondary outcomes included the time to achieve maximal sensory block, maximum motor block, time to achieve maximum motor block, time to L2 regression, and time to Bromage

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scale 0. Data were presented as numbers (%) or medians (interquartile range). Statistical analysis was performed using SPSS version 20 software (IBM Inc, Armonk, NY). Data normality was tested with the Shapiro–Wilk test. Differences in patient characteristics between groups were compared using the Mann-Whitney U test or Fisher exact test with the standardized difference (the difference in means divided by the pooled standard deviation). Differences in maximal sensory block, time to maximal sensory block, time to maximal motor block, time to L2 regression, and time to Bromage scale 0 between groups were tested using the Mann-Whitney U test. Maximal motor blockade differences were analyzed with the Fisher exact test. Univariable linear regression was used to determine the correlation of maximal sensory blockade with variables in Table 1. Multiple linear regression was conducted to assess the relationship between central obesity and maximal sensory blockade, adjusting for confounding variables with a P value less than .05 in univariable linear regression. Secondary outcomes were analyzed using the same methods. Hemodynamic variables over time between groups were analyzed using repeated-measures analysis of variance. A P value less than .05 was considered statistically significant, with P less than .01 considered significant for secondary outcomes (Bonferroni correction).

A power analysis was performed to detect a 2-segment difference with a standard deviation of 2 dermatomes in sensory level between groups, with a significance level of 5% and power of 90%. Consequently, 29 patients per group were enrolled, anticipating a 20% dropout rate.

Results of study: Fifty-eight patients were initially allocated into the two groups, but one patient from the non-CO group was excluded due to a failed lumbar puncture, leaving 57 patients for analysis. No significant differences were observed in patient characteristics and perioperative values between the two groups, except for waist circumference.

The characteristics of spinal anesthesia are detailed in Table 2. The CO group exhibited a significantly higher maximal sensory block compared to the non-CO group (P = .001). Additionally, five patients in the CO group experienced cervical block, while none in the non-CO group did.

Univariable linear regression analysis identified age (P = .037), central obesity (P = .001), and BMI (P < .001) as factors correlated with maximal sensory blockade. Multiple linear regression confirmed that central obesity (P = .004) was significantly related to maximal sensory blockade after adjusting for confounding variables. Post-spinal anesthesia, eight patients in the CO group and one patient in the non-CO group experienced hypotension, with mean ephedrine requirements of 14.5 mg in the CO group and 15 mg in the non-CO group. No bradycardia was observed in either group, and one patient in the CO group experienced nausea and vomiting. Repeated-measures

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analysis of variance showed no significant interaction between group and time on MAP or HR, and no statistical differences were found in MAP and HR between the groups.

This study demonstrates that central obesity is associated with a more extensive spread of spinal anesthesia in female patients. The finding that central obesity leads to a more extensive cephalad spread of spinal anesthesia aligns with Zhou et al.'s study, which found a strong positive correlation between abdominal girth and cephalad spread of spinal anesthesia using plain bupivacaine. A possible mechanism for these results may be related to decreased CSF volume in the central obesity group. Abdominal and epidural pressure increase when patients move from the lateral decubitus to the supine position after spinal drug injection. This positional change can exacerbate the increase in abdominal pressure in patients with greater waist circumference. According to Hogan et al., increased abdominal pressure reduces CSF volume by displacing CSF through inward movement of soft tissue in the intervertebral foramen. Reduced CSF volume, which dilutes local anesthetics, may lead to a more extensive spinal block due to decreased drug dilution. Central obesity can exist in individuals with a normal BMI. This study included patients with a BMI of 23.0−27.5 kg/m², corresponding to the normal to overweight category based on the Asian obesity criteria from the World Health Organization expert consultation. However, it's important to note that CSF volume was not assessed in this study, and the findings are based on the assumed relationship between central obesity and CSF volume. Additionally, this study was conducted in an Asian population, particularly female patients, so the results may need to be extrapolated based on the definition of central obesity for each ethnic group.

Conclusion: In conclusion, central obesity is associated with a more extensive spread of spinal anesthesia in female patients. Larger studies are needed to confirm the effect of central obesity on the spread of spinal anesthesia.

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