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CARDIORESPIRATORY REGULATION IN NON-SEVERE COMMUNITY-ACQUIRED PNEUMONIA ASSOCIATED WITH COMORBID METABOLIC SYNDROME

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Abstract: This study evaluated cardiorespiratory regulation in patients with non-severe community-acquired pneumonia with and without comorbid metabolic syndrome. The study included 35 healthy controls, 14 participants with metabolic syndrome, 62 patients with pneumonia, and 28 patients with pneumonia and metabolic syndrome. Heart rate and respiratory rhythm were synchronously recorded for 10 minutes using electrocardiography and impedance pneumography. Patients with pneumonia and metabolic syndrome demonstrated the greatest reduction in heart rate variability, including SDNN of 15.6 (10.8–20.0) ms and PHF of 22.7 (10.5–64.4) ms² compared with 31.7 (23.1–43.3) ms and 167.0 (52.5–468.7) ms² in pneumonia alone. These findings indicate that metabolic syndrome substantially aggravates cardiorespiratory regulatory dysfunction during non-severe pneumonia.

Keywords: community-acquired pneumonia; metabolic syndrome; cardiorespiratory regulation; heart rate variability; respiratory rhythm variability.

КАРДИОРЕСПИРАТОРНАЯ РЕГУЛЯЦИЯ ПРИ НЕТЯЖЕЛОЙ ВНЕБОЛЬНИЧНОЙ ПНЕВМОНИИ, АССОЦИИРОВАННОЙ С КОМОРБИДНЫМ МЕТАБОЛИЧЕСКИМ СИНДРОМОМ

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Аннотация: В исследовании оценены нарушения кардиореспираторной регуляции у пациентов с нетяжелой внебольничной пневмонией при наличии

и отсутствии коморбидного метаболического синдрома. В исследование включены 35 здоровых лиц, 14 пациентов с метаболическим синдромом, 62 больных внебольничной пневмонией и 28 пациентов с сочетанием пневмонии и метаболического синдрома. Синхронную регистрацию электрокардиограммы и импедансной пневмограммы проводили в течение 10 минут с последующей оценкой вариабельности сердечного и дыхательного ритмов. Наиболее выраженное снижение вариабельности сердечного ритма отмечено при сочетании пневмонии и метаболического синдрома: SDNN составила 15,6 (10,8–20,0) мс, а PHF — 22,7 (10,5–64,4) мс² против 31,7 (23,1–43,3) мс и 167,0 (52,5–468,7) мс² при пневмонии без метаболического синдрома. Полученные результаты свидетельствуют о существенном усугублении нарушений кардиореспираторной регуляции при сочетании нетяжелой пневмонии с метаболическим синдромом.

Ключевые слова: внебольничная пневмония; метаболический синдром; кардиореспираторная регуляция; вариабельность сердечного ритма; вариабельность дыхательного ритма.

Introduction: Community-acquired pneumonia is an acute infectious disease capable of producing systemic disturbances beyond the respiratory tract. Even when the course is non-severe, inflammatory activation, fever, and altered respiratory mechanics may influence cardiovascular regulation. Metabolic syndrome represents an additional burden characterized by abdominal obesity, elevated blood pressure, impaired glucose metabolism, and lipid abnormalities. Its coexistence with pneumonia may intensify autonomic imbalance and reduce adaptive capacity of the cardiovascular and respiratory systems. Heart rate variability provides a sensitive indicator of autonomic regulation, while respiratory rhythm variability reflects the stability of spontaneous breathing control. Simultaneous assessment of both systems can therefore provide information about cardiorespiratory interaction that is not obtained from routine vital signs alone. The study compared patients with non-severe community-acquired pneumonia with and without metabolic syndrome. Healthy individuals and subjects with metabolic

syndrome without disease were also evaluated. Particular attention was given to SDNN, CVNN, RMSSD, and spectral components of heart rate variability. Respiratory variability was assessed through the duration and dispersion of respiratory cycles. The investigation determined whether metabolic syndrome produces an additional reduction in physiological variability during pneumonia and whether these abnormalities persist after treatment. Clarifying this relationship may improve recognition of patients with increased regulatory vulnerability and provide a basis for closer cardiovascular and respiratory observation during recovery.

Aim of the study: To evaluate the additional contribution of comorbid metabolic syndrome to cardiorespiratory regulatory disturbances in patients with non-severe community-acquired pneumonia.

Materials and methods. The investigation used a prospective, controlled, parallel-group design. Four groups were studied: 35 clinically healthy individuals, 14 participants with metabolic syndrome, 62 patients with non-severe community-acquired pneumonia, and 28 patients with pneumonia accompanied by metabolic syndrome. Patients with pneumonia were assessed twice, at admission and at the end of treatment, baseline and follow-up assessments, whereas controls underwent one examination. Pneumonia severity was classified according to the SMRT-CO score. Metabolic syndrome was diagnosed using criteria that included abdominal obesity, together with at least two additional abnormalities involving blood pressure, triglycerides, HDL cholesterol, or glucose metabolism. Clinical measurements included body mass index, waist circumference, respiratory rate, blood pressure, heart rate, temperature, and oxygen saturation. Laboratory assessment included routine inflammatory and metabolic markers, specifically leukocyte count, erythrocyte sedimentation rate, C-reactive protein, glucose, HDL cholesterol, and triglycerides. Cardiorespiratory regulation was evaluated during a standardized 10-minute simultaneous recording of electrocardiography and impedance pneumography. Heart rate variability was characterized by SDNN, CVNN, RMSSD, low-frequency power, high-frequency power, LF%, HF%, and

LF/HF. Respiratory rhythm variability was determined from Ttot intervals using TtotSD and TtotCV. Statistical analysis used PSPP with median and interquartile range under comparable examination conditions. Quantitative variables were compared with the Mann–Whitney test, proportions with the chi-square test across groups, and $p<0.05$ was considered statistically significant.

Results. The groups were comparable in age and sex. Participants with metabolic syndrome had greater body mass index, waist circumference, and systolic blood pressure. In the metabolic syndrome groups, fasting glucose was 6.8 (5.3; 8.1) mmol/L compared with 4.5 (4.1; 5.1) mmol/L in healthy controls, $p<0.01$, while HDL cholesterol was lower, 1.3 (1.1; 1.6) versus 1.9 (1.7; 2.2) mmol/L, $p=0.04$. Pneumonia severity did not differ significantly according to SMRT-CO, $p=0.18$. Patients with pneumonia and metabolic syndrome had higher leukocyte counts, 10.7 (8.4; 11.7) $\times 10^9$/L versus 7.7 (6.1; 11.3) $\times 10^9$/L, $p=0.02$, higher neutrophil proportions, 78 (71; 84)% versus 70 (58; 79)%, $p=0.04$, and ESR, 25 (10; 46) versus 10 (5; 25) mm/h, $p=0.01$. Heart rate variability was most impaired in the pneumonia plus metabolic syndrome group. SDNN was 15.6 (10.8; 20.0) ms compared with 31.7 (23.1; 43.3) ms in pneumonia without metabolic syndrome, $p<0.001$. CVNN was 2.2 (1.7; 2.8)% versus 3.9 (2.7; 5.0)%, $p<0.001$; PLF was 33.9 (15.3; 81.5) versus 150.8 (73.6; 272.5) ms², $p<0.001$; and PHF was 22.7 (10.5; 64.4) versus 167.0 (52.5; 468.7) ms², $p<0.001$. By the end of treatment, SDNN increased to 22.4 (14.7; 33.9) ms in the comorbid group but remained lower than 40.7 (29.5; 62.6) ms in pneumonia alone, $p<0.001$. Respiratory rhythm variability was reduced, with the lowest TtotSD and TtotCV values observed in patients with combined disease. These abnormalities remained evident at treatment completion.

Conclusions: Comorbid metabolic syndrome was associated with substantially greater impairment of cardiorespiratory regulation in patients with non-severe community-acquired pneumonia. The most pronounced abnormalities concerned heart rate variability, with markedly lower SDNN, CVNN, PLF, and PHF values than in pneumonia without metabolic syndrome. Although SDNN increased during

treatment, it remained significantly reduced in the comorbid group, indicating incomplete functional recovery. Respiratory rhythm variability was likewise lowest when pneumonia and metabolic syndrome coexisted. These findings suggest that metabolic syndrome creates an unfavorable regulatory background during pneumonia and supports closer cardiovascular and respiratory monitoring during treatment and recovery, particularly in stable patients after discharge.

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