Original paper

Ventilator-associated pneumonia in critically ill patients with subarachnoid haemorrhage: single-centre experience

Zapalenie płuc związane z wentylacją mechaniczną u krytycznie chorych pacjentów w przebiegu krwawienia podpajęczynówkowego: badanie jednoośrodkowe

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Medical Studies/Studia Medyczne 2023; 39 (4): 334–341
DOI: https://doi.org/10.5114/ms.2023.134084

Key words: embolisation, intracranial aneurysm, subarachnoid haemorrhage, ventilator-associated pneumonia.

Słowa kluczowe: embolizacja, tętniak wewnątrzczaszkowy, krwotok podpajęczynówkowy, zapalenie płuc związane z respiratorem.

Abstract

Introduction: The need for prolonged mechanical ventilation (MV) in patients with subarachnoid haemorrhage (SAH) increases the risk of developing ventilation-associated pneumonia (VAP).
Aim of the research: To assess the prevalence of VAP and to determine its aetiological factors.
Material and methods: The study group consisted of 58 critically ill patients treated between 01.2019 and 10.2021. Demographic and clinical data were collected, including the method of collecting material from the respiratory tract, and results of laboratory and microbiological tests.
Results: More than 97% of patients were intubated on the day of admission to the ICU. The median duration of MV was 8 days (IQR: 3–19). On admission, 47 microbiological samples from the respiratory tract (39 endotracheal aspirates, 8 bronchopulmonary lavage) were collected, 26 of which (55%) were physiological flora. In the following days, VAP was diagnosed in 9/47 patients (19%). The median time from admission to diagnosis was 3.5 days (IQR: 3–4.5). Multi-drug-resistant species were found in 3 patients (K. pneumoniae ESBL+). In-hospital mortality among patients with VAP was 62%. None of the patients with SAH and VAP was liberated from ventilator.
Conclusions: In patients with SAH, early-onset VAP is frequent, and its aetiology is unrelated with bacterial colonization found on ICU admission. Even in EO-VAP, multidrug-resistant (MDR) bacteria must be suspected, mostly causing hospital-acquired pneumonia. Prognosis of patients with SAH who develop VAP is poor.

Streszczenie

Wprowadzenie: Potrzeba przedłużonej wentylacji mechanicznej u pacjentów z krwotokiem podpajęczynówkowym (SAH) zwiększa ryzyko rozwoju zapalenia płuc związanego z wentylacją mechaniczną (VAP).
Cel pracy: Ocena częstości występowania VAP i określenie jego czynników etiologicznych.
Materiał i metody: Grupa badana składała się z 58 krytycznie chorych pacjentów leczonych od stycznia 2019 do października 2021 roku. Zebrano dane demograficzne i kliniczne, w tym sposób pobrania materiału z dróg oddechowych, wyniki badań laboratoryjnych i mikrobiologicznych.
Wyniki: Ponad 97% pacjentów zostało zaintubowanych w dniu przyjęcia na OIT. Mediana czasu trwania wentylacji mechanicznej wynosiła 8 dni (IQR: 3–19). Przy przyjęciu pobrano 47 próbek mikrobiologicznych z dróg oddechowych (39 aspiratów dotchawiczych, 8 popłuczyn oskrzelowo-płucnych), z których 26 (55%) stanowiło florę fizjologiczną. W kolejnych dniach VAP zdiagnozowano u 9 spośród 47 pacjentów (19%). Mediana czasu od przyjęcia do rozpoznania wynosiła 3,5 dnia (IQR: 3–4,5). Gatunki wielolekooporne stwierdzono u 3 pacjentów (*K. pneumoniae* ESBL+). Śmiertelność wewnątrzszpitalna wśród pacjentów z VAP kształtowała się na poziomie 62%. Żaden z pacjentów z SAH i VAP nie został odłączony od respiratora.
Wnioski: U pacjentów z SAH często występuje VAP o wczesnym początku (EO-VAP), a jego etiologia nie jest związana z kolonizacją bakteryjną stwierdzoną przy przyjęciu na OIT. Nawet w przypadku EO-VAP należy podejrzewać bakterie wielolekooporne (MDR), które najczęściej powodują szpitalne zapalenie płuc. Rokowanie u pacjentów z SAH, u których rozwinie się VAP, jest złe.

Introduction

The need for prolonged mechanical ventilation (MV) in patients with subarachnoid haemorrhage (SAH) increases the risk of ventilator-associated pneumonia (VAP) [1]. It is estimated that VAP develops in 20% to as many as 75% of patients with SAH [2]. Pathophysiologically, systemic infection leads to an increase in the percentage of T regulatory cells and immature neutrophils, which pass into the cerebrospinal fluid and exacerbate intracranial inflammation. Deterioration of oxygenation index, carbon dioxide retention, and concurrent fever promote secondary brain damage, worse neurological outcomes, and increased mortality [3].

In the brain injury patient population, day 7 of hospitalisation is the cut-off point for differentiation between early-onset (EO-VAP) and late-onset (LO-VAP) VAP [4]. Earlier tracheal colonisation and microaspiration of gastrointestinal contents into the lower respiratory tract due to rapid onset of unconsciousness have been reported as risk factors for EO-VAP, in which usually non-multidrug-resistant strains predominate [5]. In contrast, the aetiology of LO-VAP is more likely to involve multidrug-resistant pathogens, necessitating the use of broad-spectrum antibiotic therapy [4].

Aim of the research

The aim of this study is to assess the prevalence of VAP, identify its aetiology, and investigate the role of bacterial colonisation of the respiratory tract in critically ill patients with SAH.

Material and methods

Study design

A single-centre, retrospective observational study was conducted in the intensive care unit (ICU) of a Polish university hospital with the highest level of reference for the treatment of acute neurological conditions. Selected demographic and clinical data of all consecutive patients with SAH diagnosed by brain computed tomography (CT), regardless of aetiology, hospitalised between 1.2019 and 9.2021, were analysed. The inclusion criterion for the study was a microbiological examination from the lower respiratory tract on the day of ICU admission. All patients were intubated using endotracheal tubes without subglottic secretion drainage.

Due to the non-interventional nature of the study, the local Bioethics Committee waived the need for informed consent from patients to participate in the study (PCN/CBN/0052/KB/116/22).

Clinical data

Data were collected including the following: SAH aetiology, severity of symptoms according to the Hunt-Hess scale, severity of bleeding on CT scan according to the Fisher scale, baseline neurological status on admission assessed by the Glasgow Coma Scale (GCS), time from hospital admission to endotracheal intubation (or the fact of intubation before hospital admission), duration of invasive MV, and ICU mortality. Laboratory data (arterial blood gas analysis, whole blood count analysis) were retrieved from medical records (AMMS software; Asseco, Poland).

The results of the available microbiological tests (i.e. cultures of blood, urine, rectal swab, and upper or lower respiratory tract material) were analysed. The decision on the type of microbiological examination from the respiratory tract was at the discretion of the attending physician and was not determined by local management protocols. Endotracheal aspirate (ETA) was collected with a Tracheal Suction Set (Primed Halberstadt, Germany) or bronchoalveolar lavage (BAL) with a disposable aScope bronchofiberoscope (Ambu, Ballerup, Denmark). The diagnosis of VAP required meeting the criteria defined by the Centers for Disease Control and Prevention, which are shown in Table 1 [6]. Threshold values for cultured samples were adapted: $\geq 10^4$ CFU/ml for BAL and $\geq 10^5$ CFU/m for ETA [6].

According to CDC recommendations, the diagnosis of physiological flora in lower respiratory tract material did not meet the definition of VAP [6]. Blood was collected under aseptic conditions from a minimum of 2 sites simultaneously, in accordance with national recommendations. On admission to the ICU, a rectal swab was routinely taken for microbiological screening for multidrug-resistant pathogens, using Equimed transport medium (Deltalab, S.L., Spain). Urine culture was performed when a urinary tract infection was found on urinalysis, ordered routinely for each patient. The interval between microbiological tests was determined by the attending physician based on the patient's clinical condition and microbiological results.

Statistical analysis

Statistical analysis was performed using the procedures available in the licensed MedCalc Statistical Software version 18.2.1 (MedCalc Software bvba, Ostend, Belgium; http://www.medcalc.org; 2018). Quantitative variables were presented as median and interquartile range (IQR, interquartile range) to unify reporting for normally distributed and skewed data. Qualitative variables were presented as absolute values and percentage. The difference between quantitative variables was assessed using analysis of variance (or Student's *t*-test for 2 groups) or the Kruskal-Wallis test (or Mann-Whitney *U*-test for 2 groups). For qualitative variables, the χ^2 test or Fisher's exact test (for group sizes of $N \leq 30$) was used. All tests were 2-sided. The correlation was assessed using Spearman

Table 1. Criteria of VAP diagnosis according to Centers for Disease Control and Prevention

Imaging test evidence	Signs/symptoms	Laboratory
Two or more serial chest imaging test results with at least one of the following: New and persistent or Progressive and persistent • Infiltrate • Consolidation • Cavitation	For ANY PATIENT, at least one of the following: • Fever (> 38.0°C or > 100.4°F) • Leukopenia (≤ 4000 WBC/mm^3) or leukocytosis (≥ 12,000 WBC/mm^3) • For adults ≥ 70 years old, altered mental status with no other recognized cause And at least two of the following: • New onset of purulent sputum or change in character of sputum, or increased respiratory secretions, or increased suctioning requirements • New onset or worsening cough, or dyspnoea, or tachypnoea • Rales or bronchial breath sounds • Worsening gas exchange (for example, O_2 desaturations (for example, $PaO_2/FiO_2 \leq 240$)], increased oxygen requirements, or increased ventilator demand)	At least one of the following: • Organism identified from blood • Organism identified from pleural fluid • Positive quantitative culture or corresponding semi-quantitative culture result from minimally contaminated LRT specimen (specifically, BAL, protected specimen brushing, or endotracheal aspirate) • ≥ 5% BAL-obtained cells contain intracellular bacteria on direct microscopic exam (for example, Gram's stain) • Positive quantitative culture or corresponding semi-quantitative culture result of lung tissue

rank correlation coefficient (R). Statistical association for dichotomous variables was assessed by odds ratio (OR) analysis with 95% confidence intervals (CI).

The criterion for statistical significance was $p < 0.05$.

Results

Fifty-eight patients with a median age of 52 (IQR: 47–62) years constituted the study group. Most patients were characterised by SAH severity category 4 on the Fisher scale (85%) and category 4+ on the Hunt-Hess scale (64%). Baseline data are presented in Table 2.

All patients were admitted to the ICU immediately after the procedure, from the emergency department, neurosurgery department, or radiology department. None of them was transferred from another department or hospital. None of the patients had a noticeable pulmonary infection before index hospitalisation. On admission to the ICU, 47 respiratory microbiological samples were collected (39 ETAs and 8 BALs), of which bacterial growth was present in 21 (54%) ETAs and 6 (75%) BALs. Sterile samples or physiological flora were obtained in 18 (46%) ETAs and 2 (25%) BALs. The cultured pathogens in ETA are shown in Table 3, and in BAL in Table 4.

During the ICU stay, 31 bronchoscopies were performed, during which microbiological material was collected for culture. The results of these BALs are shown in Table 5. Physiological florae were obtained in 4 (12%) samples.

Finally, VAP was diagnosed in 9/47 patients (19%). The detailed characteristics of these patients are shown in Table 6. The median time from admission to diagnosis of VAP was 3.5 (IQR: 3–4.5) days. There was no statistically significant difference in the incidence of VAP between men and women (9% vs. 7%, $p = 0.98$; OR = 0.98, 95% CI: 0.23–4.1). There was no correlation between neurological status in GCS scores and time to onset of VAP ($R = 0.13$; $p = 0.7$). There was no significant statistical difference between the age of patients and time to development of VAP. The median age for patients with VAP was 50 (IQR: 39–51) years, while for patients without VAP it was 52 (IQR 47–62) years ($p = 0.5$). There was no association between VAP incidence and SAH method of treatment (or no treatment), or between SAH treatment and mortality ($p > 0.05$ for all). Patients who developed VAP had higher ICU (OR = 1.69, 95% CI: 0.38–7.57) as well as in-hospital mortality (OR = 2.29, 95% CI: 0.43–12.24). None of the patients with VAP was liberated from ventilator.

Discussion

In this observational study in a population of patients with SAH, EO-VAP was diagnosed in 19% of cases, usually on day 4 of mechanical ventilation. Multidrug-resistant (MDR) bacteria were responsible for 33% of EO-VAP. Patients with VAP had a poor prognosis.

Pneumonia is one of the most common complications in neurocritical care and may concern more than a half of ventilated patients. Brain injury-induced immunosuppression syndrome is usually considered the common mechanism through which patients with critical central nervous system conditions become susceptible to different kinds of infection, including pneumonia [7]. Apoptosis and inflammation play a significant role in the brain-lung interactions leading to

Table 2. Basic demographic and clinical data

Variable	Value
Men, *n* (%)	32 (55)
Age [years]	52 [47–62]
Glasgow Coma Scale [points]	6 [3–13]
Fisher scale, *n* (%):	
1	1 (2)
2	4 (7)
3	4 (7)
4	49 (85)
Hunt-Hess scale, *n* (%):	
1	4 (7)
2	5 (9)
3	12 (21)
4	9 (16)
5	28 (48)
Cause of subarachnoid haemorrhage, *n* (%):	
Ruptured aneurysm	49 (85)
Arteriovenous malformation	3 (5)
Trauma	6 (10)
Location of aneurysm, *n* (%):	
Anterior communicating artery	17 (29)
Middle cerebral artery	11 (19)
Internal carotid artery	9 (16)
Vertebral artery	5 (9)
Basilar artery	5 (9)
Anterior cerebral artery	2 (3)
Surgical treatment of aneurysm, *n* (%)	8 (17)
Endovascular treatment of aneurysm, *n* (%)	21 (45)
History of hypertension, *n* (%)	35 (60)
Intubation on the day of ICU admission, *n* (%)	56 (97)
Duration of mechanical ventilation [days]	8 [3–18]
In-hospital successful liberation from ventilation, *n* (%)	18 (31)
ICU mortality, *n* (%)	31 (53)
In-hospital mortality, *n* (%)	36 (62)

Table 3. Results of ETAs taken on admission

Isolated pathogen	Number of cases (%)
Physiological flora	14 (31)
Staphylococcus aureus	8 (18)
Escherichia coli	5 (11)
Streptococcus pneumoniae	4 (9)
Haemophilus influenzae	4 (9)
Klebsiella pneumoniae -of which 1 KPC(+) strain	2 (4)
Klebsiella oxytoca	2 (4)
Acinetobacter baumannii	1 (2)
Candida tropicalis	1 (2)
Citrobacter freundi	1 (2)
Enterobacter cloacae ESBL(+)	1 (2)
Serratia marcescens	1 (2)
Neisseria meningitidis	1 (2)
No growth (sterile samples)	4 samples

100% refers to the number of pathogens cultured, not the samples taken.

Table 4. Results of BAL taken on admission

Isolated pathogen	Number of cases (%)
MSSA	2 (22)
Streptococcus pneumoniae	2 (22)
Acinetobacter baumannii	1 (11)
Enterobacter cloacae ESBL(+)	1 (11)
Haemophilus influenzae	1 (11)
Klebsiella pneumoniae KPC(+)	1 (11)
Escherichia coli	1 (11)
No growth (sterile samples)	2 samples

100% refers to the number of pathogens cultured, not the samples taken.

pulmonary damage of different cells. Aspiration due to delayed intubation and microaspiration post-intubation are the most common mechanical reasons for infection. Inappropriate ventilator settings may accelerate or exaggerate this complication [8]. There are no typical risk factors of VAP in SAH, but male gender, older age, tracheobronchitis, the use of therapeutic hypothermia, treatment with mannitol, and high doses of enteral nutrition were linked with VAP occurrence in neurocritically ill patients [9–11].

There are many preventive strategies that should be implemented to avoid VAP in patients, and their effectiveness has been proven in clinical settings [12, 13]. These include, but are not limited to, the use of prophylactic methods of ventilation, the use of subglottic suction tubes, cuff pressure control, semi-supine position, oral and hand hygiene, appropriate sedation and analgesia, early liberation from mechanical ventilation by spontaneous breathing trials, control and prevention delirium, early rehabilitation and mobilization, prevention of ICU-acquired weakness, prudent ulcer prevention, and early volume-graded

Table 5. Results of BAL taken during bronchoscopy during ICU stay

Isolated pathogen	Number of cases (%)
MSSA	6 (18)
Klebsiella pneumoniae	6 (18) of which: – 3 ESBL(+) strains – 1 KPC(+) strain
Candida albicans	3 (9)
Enterobacter cloacae	3 (9) of which: – 1 ESBL(+) strain
Haemophilus influenzae	3 (9)
Escherichia coli	2 (6)
MRCNS	2 (6)
Klebsiella oxytoca	1 (3)
Acinetobacter baumannii	1 (3) multidrug-resistant
Pseudomonas aeruginosa	1 (3)
Streptococcus pneumoniae	1 (3)

enteral nutrition. All these methods should be considered in patients with SAH, to prevent VAP.

The higher incidence of pneumonia in the SAH patient population is favoured by sudden disturbances of consciousness [6]. Dysphagia, as a focal neurological symptom in SAH, can affect up to 75% of patients [14]. With delayed airway protection by endotracheal intubation, the risk of pathogen aspiration into the lower airway increases, but intubation and bronchoscopy alone also carry a risk of pathogen transmission from the upper airway [15]. Sirvent *et al.* showed that tracheal colonisation in patients with traumatic brain injury on the first day may be a risk factor for subsequent pneumonia [16]. In our study, the pathogen present in the lower airway immediately after intubation subsequently identified as a possible aetiological agent of EO-VAP concerned only in 3/9 patients. At the same time, in all 3 cases, it was not the only pathogen identified by screening. This calls into question whether it is justified (medically and economically) to perform routine lower airway screening in this population of patients admitted to the ICU. Of the remaining 6 cases of EO-VAP, in 3 patients the aetiological agent was an MDR pathogen (*K. pneumoniae* ESBL+) acquired during hospitalisation in the ICU, because no patient with EO-VAP had previously been colonised with MDR pathogens. In comparison, in another single-centre, 5-year retrospective study involving 194 SAH patients, a diagnosis of VAP was made in 49% of patients, 42% of whom were EO-VAP. The main pathogen of EO-VAP was methicillin-sensitive *Staphylococcus aureus* (MSSA) (34.9%), while *Enterobacteriaceae* strains accounted for only 11%. Risk factors for EO-VAP were male sex, early use of mannitol, and delayed achievement of full enteral feeding [17]. A similar prevalence and a similar aetiology of EO-VAP were reported in a study by Bronchard *et al.* [18] conducted several years earlier, suggesting that the epidemiology of EO-VAP remains similar. The persistent rate of MDR infections is of concern with regard to the need for decisions about the use of empirical, broad-spectrum antibiotic therapy. Clinical management requires consideration of MDR infection risk factors and up-to-date microbiological mapping data from each ward. These vary between centres, making data comparison difficult. Practices on how to collect material from the lower respiratory tract also vary. BAL remains the reference method, but guidelines allow the use of ETA, mainly due to its very high negative predictive value for VAP [19]. In our study, 8/9 patients with VAP had BAL collected.

Differentiating the causes of impaired gas exchange in SAH requires consideration of pulmonary oedematous changes of neurogenic origin (neurogenic pulmonary oedema – NPE) resulting from sudden, intense activation of the sympathetic nervous system with subsequent alveolar damage [20]. In some patients, catecholamine output causes cardiac damage with subsequent cardiogenic pulmonary oedema [20]. Although macroscopically, airway aspiration was not reported in any of our patients during intubation, it cannot be excluded that some of the EO-VAP was a clinical manifestation of previous microaspiration. Lung injury is also favoured by inappropriate mechanical ventilation [21], which was not analysed in our study. All the above conditions can coexist in a single patient [17], so the diagnosis of VAP requires correlation of laboratory, radiological, and microbiological parameters and a thorough physical examination [22]. Classical criteria for VAP require radiological identification of new pulmonary lesions [6]. Lung computed tomography (CT) has the best sensitivity and specificity, but its repeated use exposes the patient to risks associated with higher radiation dose and the need for transport outside the ICU. Bedsides, chest radiography (CXR) has unacceptable sensitivity and specificity in detecting inflammatory lesions and can be abnormal as early as ICU admission in up to one-third of patients [23]. In a study by Samanta *et al.* [24], the diagnostic accuracy of lung ultrasonography (LUS) in detecting inflammatory consolidations was significantly superior to bedside CXR. Standardly determined procalcitonin with a cut-off point of > 1 ng/dl did not predict VAP more accurately than LUS [23]. The combination of LUS and echocardiography data allows us to differentiate the aetiology of pulmonary oedema and diagnose neurogenic myocardial damage [23].

Finally, one must understand that there is a growing body of evidence that the brain-lung crosstalk significantly exceeds the above-described effects and must be considered in diagnostics and treatment [25]. Acute brain injury initiates a cascade of consequences

Table 6. Characteristics of patients on the day of diagnosis of VAP

Patient	WBC [10^9/l]	Oxygenation index	Presence of purulent discharge in the bronchi	New developments in chest X-ray/CT	Result of rectal swab on admission	ETA/BAL result on admission	Pathogen causing VAP (ETA/BAL)	Day of VAP diagnosis	In-hospital death
1	13.1	143	+	+	NG	NG	*Klebsiella* pneumoniae ESBL(+) (BAL)	4	+
2	12.3	170	+	+	NG	*Citrobacter freundii* *Streptococcus pneumoniae*	*Klebsiella pneumoniae* *Streptococcus Pneumoniae* (BAL)	3	+
3	12.4	165	+	+	NG	PF	*Klebsiella pneumoniae* ESBL(+) (BAL)	4	–
4	20.9	164	+	+	NG	NG	*Pseudomonas aeruginosa* (BAL)	6	+
5	17.0	123	+	+	NG	PF	MRCNS (BAL)	4	–
6	15.2	148	+	+	NG	*Escherichia coli* *Haemophilus influenzae*	*Haemophilus influenzae* (ETA)	4	+
7	15.4	158	+	+	NG	PF	*Klebsiella pneumoniae* ESBL(+) (BAL)	4	–
8	19.8	137	+	+	NG	*Klebsiella pneumoniae* *Haemophilus influenzae* *Streptococcus pneumoniae*	*Streptococcus pneumoniae* (BAL)	4	+
9	12.1	194	+	+	NG	NG	*Streptococcus pneumoniae* (BAL)	3	+

NG – no growth, PF – physiological flora, ESBL – extended-spectrum β-lactamases, MRCNS – methicillin-resistant coagulase-negative staphylococci, ETA – endotracheal aspirate, BAL – bronchoalveolar lavage.

that can directly cause lung damage, and this can contribute to poor neurological outcomes, particularly in the early phases after severe brain trauma [26, 27].

Our study has several limitations. Firstly, it is a single-centre retrospective analysis and has all the drawbacks typical of this type of study. Due to its design and the small study group, external validity (generalizability) is limited, and further studies are needed to investigate the time of onset and origins of ventilator-associated lower respiratory tract infections in different acute neurological conditions. It also relates to patients with diabetes (only 5 subjects in our cohort) or those who are immunocompromised (none of them was recruited in our study). Secondly, the diagnosis of VAP belonged solely to the attending physician and was not subject to committee verification. Thirdly, there was no standard procedure in the ward for the performance of BF and the collection of material from the lower respiratory tract, either in terms of the timing of sampling or the method of collection (ETA or BAL), which is why we had to exclude 11 patients from our study, but we hope that this study will help to establish such a procedure. The results of the study might have changed if the samples were taken in the post-admission days. Probably the first colonization starts with intubation and becomes evident after a few days, with a significant role of co-infections. Recently, an interesting discussion has been commenced regarding differences in definitions of VAP and tracheobronchitis. It seems to be a never-ending debate: when to start and how to monitor antibiotic treatment in colonized patients. Finally, our study group comprised more severe cases of SAH. Mild cases of SAH (i.e. those without serious disturbances in consciousness and its sequelae) were treated in neurological units of our hospital. Therefore, we were unable to check the association between radiological and other features of SAH within our cohort.

Conclusions

In our study, VAP was diagnosed in a significant proportion of patients with severe subarachnoid haemorrhage (SAH), and the mortality of those who developed pneumonia was 2-fold higher compared with subjects without such a complication. Patients with EO-VAP often showed no previous colonisation with MDR pathogens, suggesting that the cause of infection was bacteria acquired during hospitalisation. Therefore, it seems medically debatable to screen from the lower respiratory tract just post-ICU admission to identify potential VAP pathogens. On the other hand, this finding underscores the need for vigilant monitoring and preventive measures against VAP in unconscious SAH patients with acute respiratory failure.

Acknowledgments

Financial support and sponsorship: Statutory research grant of the Medical University of Silesia, PCN-1-211/N/9/K.

Conflict of interest

The authors declare no conflict of interest.

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