DOI: 10.32471/rheumatology.2707-6970.19634

S.I. Smiyan
B.O. Koshak

Ivan Horbachevsky Ternopil National Medical University of the Ministry of Health of Ukraine

Key words: *ankylosing spondylitis, spondylarthritis, pulmonary hypertension, systemic inflammation, endothelial dysfunction, echocardiography.*

THE HIDDEN COMPLICATION: PULMONARY HYPERTENSION IN PATIENTS WITH ANKYLOSING SPONDYLITIS

Ankylosing spondylitis (AS) is a chronic inflammatory disease predominantly affecting the axial skeleton. While pulmonary complications in AS are often underrecognized, emerging evidence suggests that pulmonary hypertension (PH) may represent a significant comorbidity in this population. ***Objective****. This study aimed to investigate the association between AS and the risk of developing PH, with a particular focus on underlying mechanisms such as endothelial dysfunction, systemic inflammation, and respiratory impairment.* ***Methods.*** *A total of 127 AS patients and 50 healthy controls were evaluated through clinical assessments, laboratory analyses, and echocardiographic screening for PH. Echocardiography was used as a non-invasive tool to assess PH, in accordance with the diagnostic criteria set by the European Society of Cardiology (ESC) and the European Respiratory Society (ERS) guidelines, serving as an alternative to more invasive confirmatory measurements. Endothelial function was assessed via endothelium-dependent vasodilation (EDVD) and circulating endothelial cells (CEC). Pulmonary function tests were conducted using spirometry.* ***Results.*** *PH was presumed in 15% of AS patients and was not detected in any of the control group participants. AS patients with suspected PH showed significantly higher markers of systemic inflammation (CRP, ESR) and disease activity (BASDAI), longer disease duration, lower BMI, and impaired endothelial function. EDVD was markedly reduced and CEC levels elevated in patients with suspected PH. Spirometry revealed restrictive patterns in all AS patients, though not significantly different between PH-positive and PH-negative groups. The analysis of correlation relationships demonstrated the dependence of pulmonary artery pressure on disease duration, its activity and functional impairments, as well as endothelial condition.* ***Conclusions.*** *Echocardiography, as a non-invasive and safe method, is a valuable tool for detecting potential signs of PH and monitoring cardiovascular status in AS patients. This study highlights a substantial prevalence of probable PH in AS patients, primarily linked to systemic inflammation and endothelial dysfunction rather than mechanical respiratory limitations. Routine vascular and cardiopulmonary screening in AS management may facilitate early detection of PH and help improve long-term patient outcomes. However, further diagnostic confirmation through right heart catheterization is recommended to more precisely diagnose PH in these patients.*

Ankylosing spondylitis (AS) is a chronic systemic inflammatory disorder primarily targeting the axial skeleton, including the sacroiliac, intervertebral, and costovertebral joints. It is classified within the group of seronegative spondyloarthritides (SpA) [1–4].

SpA comprises a heterogeneous group of diseases characterized by inflammation of joints and adjacent tissues, displaying a diverse range of clinical manifestations. While joint involvement remains the hallmark of these disorders, systemic and extra-articular features are increasingly recognized. Among these manifestations, pulmonary hypertension (PH) has gained recognition as a formidable complication in SpA patients [4–5].

PH is defined by elevated arterial pressure in the pulmonary circulation, leading to increased vascular resistance and potential right ventricular dysfunction or failure [6]. This condition presents significant diagnostic and therapeutic challenges, particularly in SpA, where its occurrence is often overlooked due to the predominant focus on joint-related symptoms.

The pathogenesis of PH in SpA patients involves a multifactorial interplay of systemic inflammation, immune dysregulation, and structural changes. Chronic

inflammation inherent in spondyloarthropathies contributes to widespread endothelial dysfunction and vascular remodeling. Immune system activation leads to the deposition of immune complexes and inflammatory mediators in the pulmonary vasculature, exacerbating vascular resistance [6–8].

Additionally, mechanical factors related to the musculoskeletal manifestations of SpA significantly influence pulmonary function. Restricted chest mobility, resulting from ankylosis of the thoracolumbar and costovertebral joints, limits chest wall expansion. Although diaphragmatic excursion compensates to some extent, this mechanism often proves insufficient, resulting in pulmonary hypoventilation and subsequent hypoxemia. This hypoxemia further impairs endothelial function and exacerbates vascular stress, creating a favorable environment for the development of PH [7–10].

Moreover, prolonged systemic inflammation and cardiovascular stress in SpA contribute to structural and functional alterations in both the heart and pulmonary vessels. Over time, these changes culminate in the onset and progression of PH, which can significantly impact patient outcomes if left unaddressed [7].

In recent years, advances in immunobiological therapies have shifted the focus toward better understanding and managing autoimmune diseases, including joint and spinal disorders [11]. The growing recognition of comorbidities such as PH has underscored the need for a more holistic approach to SpA management. However, despite increasing interest in the systemic implications of SpA, research addressing the link between AS and PH remains sparse [12–13]. Current studies largely focus on other rheumatic diseases, such as rheumatoid arthritis (RA) and systemic scleroderma (SSD) [7, 14–16]. Evidence specific to AS and its association with PH is limited, highlighting a critical gap in understanding the prevalence, mechanisms, and clinical impact of this comorbidity [8].

Future research must aim to elucidate the pathophysiological mechanisms underlying PH in SpA and establish effective screening and management strategies to improve outcomes for affected individuals.

The aim of this study is to investigate the association between AS and the risk of developing PH, delving into the mechanisms and factors contributing to this complication within the spectrum of spondyloarthropathies.

METHODS

In total, the study included one hundred twenty-seven patients diagnosed with AS using the New York Modified Criteria [17–18]. All participants met the ASAS (Assessment of SpondyloArthritis International Society) 2010 diagnostic criteria for spondyloarthritis (SpA) [5]. The examinations were performed at the Department of Rheumatology, Ternopil Regional Hospital (Ternopil, Ukraine), during the period of 2017–2024. Participants had to be at least 18 years old and have a documented disease history of at least 5 years. Exclusion criteria encompassed individuals over 60 years old and those with chronic conditions independently contributing to PH development, including diabetes, respiratory disorders (COPD, ILD), ischemic heart disease (IHD), significant congenital or acquired heart defects, circulatory insufficiency, and severe liver impairment (active hepatitis, cirrhosis). Moreover, a control group consisting of fifty clinically healthy participants aged between 25 and 35 years was included in the study. Written consent was obtained from all participants in accordance with the Declaration of Helsinki, and the study received approval from the local Ethical-Scientific Committee of Ternopil National Medical University.

All enrolled participants who provided consent underwent a thorough examination, including a detailed physical assessment and BMI measurement. Diagnostic procedures involved comprehensive biochemical analyses of blood parameters to characterize the course of the underlying disease and comorbidities.

Other examinations included ultrasound of the carotid intima-media complex, assessment of endothelium-dependent vasodilation (EDVD) in response to reactive hyperaemia. Echocardiography was performed to assess cardiac function, including the measurement of pulmonary artery pressure.

According to the European Society of Cardiology/European Respiratory Society (ESC-ERS) guidelines, PH is defined by a mean pulmonary arterial pressure (mPAP) of ⩾20 mmHg at rest [19].

PH was assessed and diagnosed through transthoracic echocardiography, a widely recognized non-invasive technique recommended for the initial evaluation of suspected PH cases [19–21]. While right heart catheterization (RHC) remains the gold standard for confirming PH, it was not utilized in this study due to its invasive nature and ethical constraints.

Echocardiographic assessment included estimating systolic pulmonary artery pressure (sPAP) using tricuspid regurgitation velocity (TRV) and the modified Bernoulli equation ($sPAP = 4 \cdot v2 + mean\ RAP$ [right atrial pressure]).

PH was diagnosed in patients presenting with the following echocardiographic features [19]: TRV ⩾2.8 m/s, indicating elevated sPAP.

Evidence of right ventricular overload or dysfunction supporting the presence of PH.

Based on these echocardiographic findings, all AS patients were divided into two groups: those with probable PH and those without echocardiographic signs of PH. This classification was made acknowledging the limitations of echocardiography in definitively diagnosing PH, as right heart catheterization was not performed.

This echocardiographic approach provided a practical and ethically acceptable method for identifying patients at risk of PH within the study cohort.

Statistical analysis was performed using standard methods in Statistica 12.0 software (StatSoft, Tulsa OK, USA).

RESULTS

Pulmonary hypertension (PH) was presumed in 15.0% of patients during the undertaken examination and was not detected in any of the control group participants. The mean systolic pulmonary artery pressure

(sPAP) in patients with ankylosing spondylitis (AS) was 21.7±0.9 mm Hg. In the control group, which consisted of healthy individuals, the mean sPAP was 18.9±1.1 mm Hg. To investigate potential risk factors for PH, an analysis of the clinical and laboratory characteristics of AS patients was performed (Table 1).

Table 1

Clinical and laboratory characteristics of patients stratified by the presence of probable PH (M±m)

Parameter	PH (+) (n=19)	PH (-) (n=108)	p-value
Mean age, years	39.8±1.17	36.6±1.35	p=0.041
Disease duration, years	15.7±0.92	11.8±0.41	p<0.001
Body mass index (BMI), kg/m²	17.8±0.47	21.3±0.88	p<0.001
Chest expansion, cm	1.5±0.08	3.6±0.14	p<0.001
Schober's test, cm	1.8±0.19	4.1±0.37	p<0.001
BASDAI	6.37±0.12	5.42±0.41	p=0.028
BASFI	7.13±0.24	6.22±0.21	p<0.001
Smoking, n (%)	6 (22.2%)	18 (21.7%)	–
Carotid intima-media thickness (CIMT), mm	1.02±0.02	0.95±0.01	p<0.001
Total cholesterol (TC), mmol/L	5.14±0.15	4.71±0.08	p=0.02
High-density lipoprotein cholesterol (HDL-C), mmol/L	0.92±0.02	1.13±0.03	p=0.018
Low-density lipoprotein cholesterol (LDL-C), mmol/L	3.18±0.07	2.86±0.05	p=0.004
Triglycerides (TG), mmol/L	1.51±0.14	1.33±0.18	p=0.4
Atherogenic index (AI)	4.61±0.15	3.98±0.09	p<0.001
Arterial hypertension (AH), n (%)	7 (25.9%)	17 (20.5%)	–
C-reactive protein (CRP), mg/L	16.88±0.67	12.51±0.47	p<0.001
Erythrocyte sedimentation rate (ESR), mm/h	29.67±1.26	22.56±0.76	p<0.001
Circulating endothelial cells (CEC), ×10⁴/L	6.4±0.18	5.2±0.24	p<0.001
Endothelium-dependent vasodilation (EDVD), %	7.14±0.21	8.95±0.18	p<0.001
Endothelium-independent vasodilation (EIVD), %	26.4±0.29	21.8±0.24	p<0.001
Note: p-value represents statistical significance between PH (+) and PH (-) groups.			

The obtained data indicate that PH in AS patients is associated with inflammatory markers (CRP, ESR), disease duration and activity (BASDAI), and functional status (BASFI). Additionally, PH patients exhibited a slightly higher prevalence of arterial hypertension (AH), reinforcing the interconnected nature of pathophysiological processes within the cardiovascular system.

Smoking was identified as an important factor in PH development; however, the prevalence of smokers was similar between patients with and without echocardiographic indications of probable PH (22.2% *vs* 21.7%).

Literature suggests that an increased BMI is linked to PH development. However, in this study, PH was associated with a lower BMI. This inverse relationship is consistent with findings in long-term AS, where chronic disease progression is often accompanied by weight loss.

To explore the potential role of endothelial dysfunction in the development of PH among AS patients, endothelial function parameters and markers of endothelial injury were analyzed in both groups—those with and without echocardiographic signs suggestive of PH. The results showed that EDVD was significantly lower in patients with probable PH compared to those without. EDVD is a key indicator of endothelial function, primarily reflecting the ability to produce and release nitric oxide (NO). In addition to reduced EDVD, AS patients with probable PH exhibited significantly higher circulating endothelial cell (CEC) levels (p<0.001), a marker of endothelial damage. These findings further highlight the complex interplay of pathogenic factors potentially contributing to PH development.

Significant correlations were found between sPAP and the following parameters: Disease duration (r=0.71, p<0.001), functional impairment (BASFI) (r=0.64, p<0.001), markers of endothelium dysfunction (CEC level (r=0.51, p<0.001), EDVD (r=–0.49, p<0.001)), Lower BMI (r=–0.47, p<0.001), disease activity (BADAI) (r=0.42, p<0.05). These findings emphasize the role of endothelial dysfunction in the pathogenesis of PH in AS patients.

To evaluate the impact of reduced respiratory movement volume on the development of PH in AS patients, we analyzed pulmonary function parameters based on spirometry results in both groups (Table 2).

Table 2

Pulmonary Function Parameters Based on Spirometry (M ± m)

Parameter	PH (+) (n=27)	PH (-) (n=83)	p-value
Vital capacity (VC), %	71.3±15.4	73.9±13.2	0.524
Forced vital capacity (FVC), %	74.2±12.1	77.1±10.2	0.341
Peak expiratory flow (PEF), %	66.1±8.8	69.1±9.1	0.412
Forced expiratory volume in 1 second (FEV1), %	78.1±14.2	81.1±12.9	0.342
Tiffeneau index	1.1±2.2	1.09±2.4	0.388
Gensler index	1.05±0.12	1.04±0.09	0.404
Note: VC – vital capacity, FVC – forced vital capacity, PEF – peak expiratory flow, FEV1 – forced expiratory volume in 1 second.			

The obtained data clearly demonstrate the presence of restrictive respiratory impairment in the examined patients. a decrease in vital capacity parameters was observed, with FVC values exceeding VC, along with reduced PEF, normal FEV1, and increased Tiffeneau and Gensler indices. However, the severity of these respiratory function abnormalities did not significantly differ between PH-positive and PH-negative patients. These findings suggest that although respiratory dysfunction and alveolar hypoventilation are important, they are not the primary risk factors for PH development in AS patients.

DISCUSSION

The identification of probable PH in 15.0% of AS patients in our study suggests that this complication may be more prevalent than previously assumed. The available literature on PH in AS is sparse, with most studies focusing on other rheumatic diseases such as SSD and RA, where PH has been recognized as a major co-

morbidity [7, 14–16, 22–24]. For instance, PH prevalence in systemic sclerosis is reported to range between 8–12% when diagnosed using RHC [16, 25–26], with echocardiographic screening detecting even higher rates. The higher prevalence observed in our cohort may be partially explained by the use of echocardiographic criteria, which, while widely accepted for screening, tend to overestimate PH prevalence compared to RHC. Nevertheless, our findings indicate that AS should be considered among the inflammatory diseases predisposing patients to pulmonary vascular complications.

One of the most striking aspects of our findings is the strong association between PH and systemic inflammation, as evidenced by significantly elevated CRP and ESR levels in PH-positive patients. This supports the hypothesis that chronic inflammation plays a pivotal role in pulmonary vascular remodeling, a phenomenon well-documented in other inflammatory conditions [24–25].

Studies on RA and SSD have demonstrated that inflammatory cytokines such as TNF-α, IL-6, and IL-1β contribute to endothelial dysfunction, promoting vasoconstriction and vascular remodeling [14, 15]. While specific mechanistic studies in AS remain limited, our results align with findings from studies on other spondyloarthropathies, which suggest that systemic inflammation exerts widespread vascular effects beyond joint pathology [2, 4, 8].

Another intriguing aspect of our study is the inverse relationship between BMI and PH. Unlike in the general population, where obesity is a known risk factor for PH due to increased metabolic and cardiovascular strain, AS patients with PH in our study had significantly lower BMI values. Similar findings have been reported in advanced RA, where disease-related cachexia leads to muscle wasting and systemic metabolic alterations that may predispose to vascular complications [27–28]. This observation suggests that, in chronic inflammatory diseases, PH may not follow traditional metabolic risk patterns but instead be driven by long-standing inflammation, endothelial injury, and progressive structural impairment.

While restrictive lung disease is a well-recognized feature of AS [29], our study found no significant differences in spirometric parameters between PH-positive and PH-negative patients. Our data suggest that, in AS, pulmonary restriction alone may not be the primary mechanism leading to PH. Instead, systemic inflammation and endothelial dysfunction appear to play a more dominant role, as evidenced by the strong correlations between PH and markers of endothelial injury, including CEC and EDVD.

Endothelial dysfunction is increasingly recognized as a key mechanism in PH across various disease contexts [30–31]. Studies on pulmonary arterial hypertension in autoimmune diseases highlight the critical role of nitric oxide (NO) dysregulation [32] and endothelial injury in driving vascular resistance [33]. The significantly lower EDVD values in our PH-positive AS patients suggest that endothelial nitric oxide bioavailability is impaired, which aligns with previous research indicating reduced NO-mediated vasodilation in inflammatory rheumatic diseases [32, 34]. Elevated CEC levels further corroborate this, as CECs serve as a biomarker of ongoing endothelial injury and vascular remodeling [22].

Taken together, our results support the hypothesis that PH in AS is primarily driven by systemic inflammatory and endothelial pathways rather than by mechanical respiratory impairment [14, 35–36].

The clinical implications of our findings are substantial, as PH can lead to significant morbidity and mortality if unrecognized and untreated. Our study suggests that AS patients, particularly those with high disease activity and long disease duration, may have slightly elevated echocardiographic parameters, indicating a probable risk for pulmonary hypertension, and could benefit from systematic screening for pulmonary vascular involvement.

Current AS management guidelines primarily focus on musculoskeletal and cardiovascular risks [5], with limited recommendations regarding pulmonary complications, yet the observed associations between PH, endothelial dysfunction, and disease activity highlight the need for an expanded clinical approach that incorporates vascular assessments into routine AS care.

Although right heart catheterization remains the gold standard for confirming the diagnosis of pulmonary hypertension (PH), recent studies, including those by Nikolaos P.E. Kadoglou (2024), have shown a high correlation between echocardiographic findings and invasive pulmonary pressure measurements [37]. These results suggest that echocardiography can be an effective method for both diagnosing and prognosticating PH, offering a practical and less invasive approach to detecting pulmonary hypertension in clinical practice. Specifically, the use of echocardiography to assess systolic pulmonary artery pressure and right ventricular function allows for effective identification of patients at higher risk for developing PH, underscoring its potential as an important diagnostic tool.

Further research is needed to confirm our findings and explore potential therapeutic strategies targeting vascular dysfunction in AS. The impact of biologic disease-modifying antirheumatic drugs (bDMARDs) on PH development remains unclear. Some studies suggest that TNF inhibitors and IL-17 blockers may exert protective vascular effects by reducing systemic inflammation and improving endothelial function [11], while others indicate that long-term vascular outcomes in AS remain suboptimal despite adequate joint disease control. Future prospective studies incorporating serial endothelial function assessments and long-term PH follow-up in AS patients receiving different treatment regimens are warranted.

In conclusion, our study provides strong evidence linking PH to systemic inflammation and ED in AS. These findings call for increased awareness of pulmonary vascular complications in AS patients, particularly those with high disease activity or long disease duration. Routine screening for PH, alongside comprehensive vascular assessments, could significantly improve patient outcomes by enabling earlier detection and intervention.

REFERENCES

1. van Gaalen F.A., Rudwaleit M. (2023) Challenges in the diagnosis of axial spondyloarthritis. Best Practice & Research: Clinical Rheumatology, 37(3): 101871.

2. Duba A.S., Mathew S.D. (2018) The seronegative spondyloarthropathies. Primary Care, 45(2): 271–287.

3. Dougados M., Baeten D. (2011) Spondyloarthritis. Lancet, 377(9783): 2127–2137.

4. Walsh J.A., Magrey M. (2021) Clinical manifestations and diagnosis of axial spondyloarthritis. Journal of Clinical Rheumatology, 27(8): e547-e560.

5. Burgos-Vargas R., Dougados M., Hermann K.G. et al. (2009) The Assessment of SpondyloArthritis international Society (ASAS) handbook: a guide to assess spondyloarthritis. Annals of the Rheumatic Diseases, 68(2): 1–44.

6. Hassoun P.M. (2021) Pulmonary arterial hypertension. New England Journal of Medicine, 385: 2361–2376.

7. Cansu D., Korkmaz C. (2023) Pulmonary hypertension in connective tissue diseases: Epidemiology, pathogenesis, and treatment. Clinical Rheumatology, 42(10): 2601–2610.

8. Сміян С.І., Кошак Б.О., Білуха А.В. (2024) Поза суглобами: легенева гіпертензія при спондилоартропатіях (Огляд літератури). Сімейна Медицина. Європейські практики, 1: 89–95.

9. Distler O., Pignone A. (2006) Pulmonary arterial hypertension and rheumatic diseases–from diagnosis to treatment. Rheumatology, 45(4): 21–25.

10. Bousseau S., Sobrano Fais R., Gu S. (2023) Pathophysiology and new advances in pulmonary hypertension. BMJ Medicine, 2: e000137.

11. Danve A., Deodhar A. (2022) Treatment of axial spondyloarthritis: An update. Nature Reviews Rheumatology, 18(4): 205–216.

12. Yang T.Y., Chen Y.H., Siao W.Z., Jong G.P. (2022) Case report: a rare manifestation of pulmonary arterial hypertension in ankylosing spondylitis. Journal of Personalized Medicine, 13(1): 62.

13. Hung Y.M., Cheng C.C., Wann S.R., Lin S.L. (2015) Ankylosing spondylitis associated with pulmonary arterial hypertension. Internal Medicine, 54: 431–434.

14. Montani D., Henry J., O'Connell C. et al. (2018) Association between rheumatoid arthritis and pulmonary hypertension: Data from the French pulmonary hypertension registry. Respiration, 95(4): 244–250.

15. Stefanantoni K., Sciarra I., Vasile M. et al. (2015) Elevated serum levels of macrophage migration inhibitory factor and stem cell growth factor β in patients with idiopathic and systemic sclerosis-associated pulmonary arterial hypertension. Reumatismo, 66(4): 270–276.

16. Lechartier B., Humbert M. (2021) Pulmonary arterial hypertension in systemic sclerosis. Presse Medicale, 50(1): 104062.

17. Raychaudhuri S.P., Deodhar A. (2014) The classification and diagnostic criteria of ankylosing spondylitis. Journal of Autoimmunity, 48–49: 128–133.

18. van der Linden S., Valkenburg H. A., Cats A. (1984) Evaluation of diagnostic criteria for ankylosing spondylitis. a proposal for modification of the New York criteria. Arthritis & Rheumatology, 27(4): 361–368.

19. Humbert M., Kovacs G., Hoeper M.M. et al. (2022) 2022 ESC/ERS guidelines for the diagnosis and treatment of pulmonary hypertension. European Heart Journal, 43(38): 3618–3731.

20. Meinel K., Koestenberger M., Sallmon H. et al. (2020) Echocardiography for the assessment of pulmonary hypertension and congenital heart disease in the young. Diagnostics (Basel), 11(1): 49.

21. Koestenberger M., Grangl G., Avian A. et al. (2017) Normal reference values and z scores of the pulmonary artery acceleration time in children and its importance for the assessment of pulmonary hypertension. Circulation: Cardiovascular Imaging, 10: e005336.

22. Evans C.E., Cober N.D., Dai Z. et al. (2021) Endothelial cells in the pathogenesis of pulmonary arterial hypertension. European Respiratory Journal, 58(3): 2003957.

23. Haensel M., Wojciak-Stothard B. (2023) The role of endothelial cells in pulmonary hypertension: Old concepts and new science. Current Opinion in Physiology, 34: 100667.

24. Knarborg M., Hyldgaard C., Bendstrup E. et al. (2023) Comorbidity and mortality in systemic sclerosis and matched controls: Impact of interstitial lung disease. a population-based cohort study based on health registry data. Chronic Respiratory Disease, 20: 14799731231195041.

25. Rubio-Rivas M., Homs N.A., Cuartero D., Corbella X. (2021) The prevalence and incidence rate of pulmonary arterial hypertension in systemic sclerosis: Systematic review and meta-analysis. Autoimmunity Reviews, 20(1): 102713.

26. Giuggioli D., Bruni C., Cacciapaglia F. et al. (2021) Pulmonary arterial hypertension: Guidelines and unmet clinical needs. Reumatismo, 72(4): 228–246.

27. Santo R.C.E., Fernandes K.Z., Lora P.S. et al. (2018) Prevalence of rheumatoid cachexia in rheumatoid arthritis: a systematic review and meta-analysis. Journal of Cachexia, Sarcopenia and Muscle, 9(5): 816–825.

28. Masuko K. (2014) Rheumatoid cachexia revisited: a metabolic co-morbidity in rheumatoid arthritis. Frontiers in Nutrition, 1: 20.

29. Dhahri R., Mejri I., Ghram A. et al. (2023) Assessment tools for pulmonary involvement in patients with ankylosing spondylitis: Is diaphragmatic ultrasonography correlated to spirometry? Journal of Multidisciplinary Healthcare, 16: 51–61.

30. Bian J.S., Chen J., Zhang J. et al. (2024) ErbB3 governs endothelial dysfunction in hypoxia-induced pulmonary hypertension. Circulation, 150(19): 1533–1553. doi.org/10.1161/CIRCULATIONAHA.123.067005.

31. Boucly A., Gerges C., Savale L. et al. (2023) Pulmonary arterial hypertension. Presse Medicale, 52(3): 104168.

32. Mandras S., Kovacs G., Olschewski H. et al. (2021) Combination therapy in pulmonary arterial hypertension–Targeting the nitric oxide and prostacyclin pathways. Journal of Cardiovascular Pharmacology and Therapeutics, 26(5): 453–462.

33. Lázár Z., Mészáros M., Bikov A. (2020) The nitric oxide pathway in pulmonary arterial hypertension: Pathomechanism, biomarkers, and drug targets. Current Medicinal Chemistry, 27(42): 7168–7188.

34. Ruopp N.F., Cockrill B.A. (2022) Diagnosis and treatment of pulmonary arterial hypertension: a review. JAMA, 327(14): 1379–1391.

35. Mathai S.C. (2022) Pulmonary hypertension associated with connective tissue disease. Cardiology Clinics, 40(1): 29–43.

36. Fayed H., Coghlan J.G. (2019) Pulmonary hypertension associated with connective tissue disease. Seminars in Respiratory and Critical Care Medicine, 40(2): 173–183.

37. Kadoglou N.P.E., Khattab E., Velidakis N. et al. (2024). The role of echocardiography in the diagnosis and prognosis of pulmonary hypertension. Journal of Personalized Medicine, 14(5): 474.

ЛЕГЕНЕВА ГІПЕРТЕНЗІЯ ПРИ АНКІЛОЗИВНОМУ СПОНДИЛОАРТРИТІ: ПРИХОВАНА ПРОБЛЕМА

С.І. Сміян, Б.О. Кошак

Тернопільський національний медичний університет ім. І. Я. Горбачевського МОЗ України

Резюме. *Анкілозуючий спондилоартрит (АС) — це хронічне запальне захворювання, яке переважно уражує осьовий скелет. У той час як легеневі ускладнення при АС часто недооцінюються, нові дані свідчать про те, що легенева гіпертензія (ЛГ) може бути значущою супутньою патологією серед цієї категорії пацієнтів.* ***Метою*** *дослідження було оцінити зв'язок між АС та ризиком розвитку ЛГ, з особливим акцентом на механізми ендотеліальної дисфункції, системного запалення та порушення функції зовнішнього дихання.* ***Методи:*** *обстежено 127 пацієнтів з АС, що перебували на лікуванні в ревматологічно-*

му відділенні Тернопільської обласної лікарні у 2017–2024 рр., та 50 здорових осіб (контрольна група). Усім пацієнтам проведені діагностичні обстеження, що характеризують перебіг захворювання. Ехокардіографію використовували як неінвазивний інструмент для оцінки ЛГ відповідно до діагностичних критеріїв, встановлених Європейським товариством кардіологів (European Society of Cardiology — ESC) і рекомендаціями Європейського респіраторного товариства (European Respiratory Society — ERS), слугуючи альтернативою більш інвазивним дослідженням. Функцію ендотелію оцінювали за допомогою ендотелій-залежної вазодилатації (ЕЗВД) і циркулюючих ендотеліальних клітин (ЦЕК). Дослідження функції зовнішнього дихання проводили за допомогою спірометрії. ***Результати.*** *Ознаки ймовірної ЛГ виявлені у 15% обстежуваних з АС та не виявлені у жодної особи з групи контролю. У пацієнтів з АС виявлено значно вищі маркери системного запалення (С-реактивний білок, швидкість осідання еритроцитів) та активності захворювання (Bath Ankylosing Spondylitis Disease Assessment — BASDAI), більшу тривалість захворювання, нижчий індекс маси тіла та порушення функції ендотелію. ЕЗВД була помітно знижена, а рівні ЦЕК підвищені у пацієнтів із підозрою на ЛГ. Спірометрія підтвердила рестриктивні зміни у функції зовнішнього дихання, але без значної різниці між пацієнтами з ймовірною ЛГ та без неї. Аналіз кореляційних зв'язків продемонстрував залежність тиску в легеневій артерії від тривалості недуги, її активності та функціональних порушень, стану ендотелію.* ***Висновки.*** *Це дослідження підкреслює значну поширеність ймовірної ЛГ у пацієнтів з АС, пов'язану насамперед із системним запаленням та ендотеліальною дисфункцією, а не з функціональними порушеннями зовнішнього дихання. Рутинний кардіопульмональний скринінг при лікуванні АС може полегшити раннє виявлення ЛГ і допомогти покращити віддалені результати для пацієнтів. Ехокардіографія як неінвазивний та безпечний метод, є цінним інструментом для виявлення потенційних ознак ЛГ та моніторингу серцево-судинного статусу у пацієнтів із АС. Однак для більш точної діагностики ЛГ у цих пацієнтів рекомендується подальше діагностичне підтвердження шляхом катетеризації правих відділів серця.*

Ключові слова: *анкілозивний спондилоартрит, серонегативна спондилоартритопатія, легенева гіпертензія, системне запалення, ендотеліальна дисфункція, ехокардіографія.*

Відомості про авторів

Сміян Світлана Іванівна — докторка медичних наук, завідувачка кафедри, професорка, кафедра внутрішньої медицини № 2, Тернопільський національний медичний університет ім. І.Я. Горбачевського МОЗ України.
ORCID ID: 0000-0001-5543-9895

Кошак Богдан Олександрович — кандидат медичних наук, доцент, кафедра внутрішньої медицини № 2, Тернопільський національний медичний університет ім. І.Я. Горбачевського МОЗ України.
ORCID ID: 0000-0002-8470-4421

Надійшла до редакції/Received: 25.03.2025
Прийнято до друку/Accepted: 31.03.2025