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DYNAMICS OF ECHOCARDIOGRAPHIC PARAMETERS OF PULMONARY HYPERTENSION DURING BOSENTAN THERAPY IN PATIENTS UNDERGOING PROGRAM HEMODIALYSIS

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ABSTRACT

Associated pulmonary hypertension worsens the prognosis for patients undergoing scheduled hemodialysis. Endothelin receptor antagonists, including bosentan, are standard treatment for pulmonary arterial hypertension. Echocardiography (TTE) remains a key tool for screening and longitudinal monitoring of the right heart and estimating PH probability. In this cohort, the addition of bosentan to comprehensive therapy in maintenance hemodialysis patients with pulmonary hypertension was associated with a marked improvement in pulmonary hemodynamics and right-heart function, demonstrating clinical feasibility in carefully selected hemodialysis patients with regular safety monitoring.

Keywords: pulmonary hypertension; chronic kidney disease; hemodialysis; bosentan; echocardiography; TAPSE; sPAP; endothelin-1.

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ДИНАМИКА ЭХОКАРДИОГРАФИЧЕСКИХ ПОКАЗАТЕЛЕЙ ЛЕГОЧНОЙ ГИПЕРТЕНЗИИ НА ФОНЕ ТЕРАПИИ БОЗЕНТАНОМ У ПАЦИЕНТОВ, НАХОДЯЩИХСЯ НА ПРОГРАММНОМ ГЕМОДИАЛИЗЕ

АННОТАЦИЯ

Сопутствующая легочная гипертензия ухудшает прогноз у пациентов, находящихся на плановом гемодиализе. Антагонисты рецепторов эндотелина, включая бозентан, являются стандартом лечения легочной артериальной гипертензии. Эхокардиография (ТТЭХО) остается ключевым инструментом скрининга и продольного мониторинга состояния правых отделов

сердца, а также оценки вероятности развития легочной гипертензии. В данной когорте пациентов с легочной гипертензией, находящихся на поддерживающем гемодиализе, добавление бозентана к комплексной терапии было связано со выраженным улучшением легочной гемодинамики и функции правых отделов сердца, что свидетельствует о клинической целесообразности применения препарата у тщательно отобранных пациентов, находящихся на гемодиализе, с регулярным контролем безопасности.

Ключевые слова: легочная гипертензия; хроническая болезнь почек; гемодиализ; бозентан; эхокардиография; TAPSE; sPAP; эндотелин-1.

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GEMODIALIZ DASTURIDAN O'TAYOTGAN BEMORLARDA BOSENTAN TERAPIYASI PAYTIDA O'PKA GIPERTENZIYASINING EXOKARDIOGRAFIK PARAMETRLARINING DINAMIKASI

ABSTRAKT

Bilan bog'liq o'pka gipertenziiyasi rejalashtirilgan gemodializdan o'tadigan bemorlar uchun prognozni yomonlashtiradi. Endotelin retseptorlari antagonistlari, shu jumladan bosentan, o'pka arterial gipertenziiyasi uchun standart davolash hisoblanadi. Ekokardiyografiya (TTE) o'ng yurakni skrining va bo'ylama monitoringi va PH ehtimolini baholash uchun asosiy vosita bo'lib qolmoqda. Ushbu kohortda o'pka gipertenziiyasi bilan og'rigan gemodializ bilan og'rigan bemorlarda kompleks terapiyaga bosentan qo'shilishi o'pka gemodinamikasi va o'ng yurak funksiyasining sezilarli yaxshilanishi bilan bog'liq bo'lib, ehtiyotkorlik bilan tanlangan gemodializ bemorlarida xavfsizlikni muntazam monitoring qilishda klinik maqsadga muvofiqligini ko'rsatdi.

Kalit so'zlar: o'pka gipertenziiyasi; surunkali buyrak kasalligi; gemodializ; bosentan;

Introduction

According to recent reviews and meta-analyses, the prevalence of pulmonary hypertension (PH) among dialysis patients ranges from 30% to 60% and is associated with increased overall and cardiovascular mortality as well as hospitalizations [1–4]. In patients receiving maintenance hemodialysis (MHD), the presence of PH correlates with an unfavorable prognosis: a meta-analysis demonstrated a 1.44-fold increase in all-cause mortality risk in chronic kidney disease (CKD) and an even higher risk (up to 2.3-fold) among those on long-term hemodialysis [3,4]. The pathogenesis of PH in CKD is multifactorial, involving chronic volume overload from an arteriovenous fistula, increased cardiac output with hyperkinetic circulation, left ventricular remodeling with diastolic dysfunction, pulmonary vascular calcification, microangiopathy, and inflammation [1,2]. The endothelin-1 (ET-1) system plays a critical role in pulmonary vascular remodeling and the pathophysiology of renal injury; its hyperactivation has been documented in CKD [6–9]. Endothelin receptor antagonists (ERAs), including bosentan, are the standard therapy for group 1 pulmonary arterial hypertension (PAH). Under strict safety monitoring, off-label use of ERAs may be considered in carefully selected CKD patients with a predominant precapillary component of PH [1]. From a clinical standpoint, bosentan is highly protein-bound (>98%) and therefore not effectively removed by hemodialysis; regular monitoring of liver enzymes (monthly) and consideration of potential drug–drug interactions are essential [10,11]. The current ESC/ERS (2022) and ASE (2025) guidelines specify an echocardiographic algorithm for assessing right-heart function in suspected PH, including tricuspid regurgitation velocity; indirect signs of RV overload/dysfunction (right atrial area, RV fractional area change, S' velocity of the tricuspid annulus, TAPSE/sPAP ratio, pulmonary artery acceleration time—PAAT, and others) [1,5]. Longitudinal RV systolic function is assessed by TAPSE; a value <17 mm indicates reduced longitudinal RV function [5]. The TAPSE/sPAP ratio reflects RV–pulmonary artery coupling and has prognostic value across PH phenotypes; its

incorporation improves risk stratification versus single echocardiographic metrics [12–14]. Nevertheless, data on the dynamics of TAPSE and TAPSE/sPAP under ERA therapy specifically in MHD patients remain limited, warranting targeted clinical observation.

Materials and Methods

To evaluate echocardiographic characteristics of patients with pulmonary hypertension on maintenance hemodialysis and to assess their dynamic changes following the addition of bosentan to standard therapy.

Study Design and Population

This study was conducted at the Republican Specialized Scientific and Practical Medical Center for Nephrology and Kidney Transplantation. A total of 64 patients (n=64) with CKD stage 5D on maintenance hemodialysis (three sessions per week, 4 hours each) and echocardiographic evidence of PH (sPAP >35–40 mmHg estimated by tricuspid regurgitation velocity in conjunction with clinical signs) were enrolled. Patients were divided into two groups: Group 1 (n=33) received bosentan in addition to standard therapy; Group 2 (n=31) received standard therapy only (no bosentan). Assessments were performed at baseline, 3 months, and 6 months.

Inclusion criteria: age 42–76 years; signed informed consent; absence of malignancy, hematologic disease, or acute infection; no major surgery within the previous 6 months; no acute cerebrovascular or cardiovascular events within 6 months; no major trauma or massive bleeding within 6 months; eGFR (CKD-EPI 2021) ≤ 15 mL/min/1.73 m² within 1 month before inclusion. Exclusion criteria: significant hepatic dysfunction (ALT/AST >3× ULN), planned kidney transplantation within 6 months, uncontrolled hypertension, pregnancy, or contraindications/intolerance to bosentan.

Cohort Characteristics and Concomitant Management

Mean age was 59.08 ± 0.84 years; mean disease duration 3.50 ± 0.13 years. After signing informed consent, all patients were hospitalized. The dialysis regimen was unchanged due to the drug's high protein binding. Concomitant therapies included blood pressure control (ACE inhibitors or ARBs; calcium channel blockers; beta-blockers), diuretics for edema (furosemide, ethacrynic acid, spironolactone), enterosorbents (Polysorb, Enterosgel) for uremia control, ketoanalogues of amino acids (Ketobest/Ketosteril), electrolyte (Na, K, Ca, P) and mineral-bone disorder management, anemia therapy (iron preparations, erythropoietin) where indicated, and individualized diet. Eligible patients received bosentan (nonselective ETA/ETB receptor antagonist) initiated at 62.5 mg twice daily and uptitrated to 125 mg twice daily if tolerated.

Investigations

All patients underwent: (i) laboratory testing—complete blood count, urinalysis, biochemical profile (urea, creatinine, eGFR by CKD-EPI 2021, Na, K, Ca, P), coagulation panel (INR, fibrinogen), and plasma endothelin-1; (ii) transthoracic echocardiography with measurement of sPAP, TAPSE, right atrial area, RV wall thickness, RV outflow tract (RVOT) diameter, left ventricular eccentricity index (LVEI), pulmonary artery acceleration time (PAAT), and tricuspid regurgitation grade.

Results

In the bosentan group, progressive favorable dynamics were observed by 3 months with further enhancement by 6 months. Hemoglobin increased from 108 to 112 and 114 g/L (+3.7% and +5.6%); RBC from 3.7 to 3.8 and $3.9 \times 10^{12}/L$ (+2.7% and +5.4%); WBC decreased from $6.2 \rightarrow 6.0 \rightarrow 5.8 \times 10^9/L$ (−3.2% and −6.5%); platelets remained within reference with a mild upward trend ($190 \rightarrow 195 \rightarrow 200 \times 10^9/L$). Uremic markers improved: urea $14.8 \rightarrow 14.2 \rightarrow 13.8$ mmol/L (−4.1% and −6.8%); creatinine $185 \rightarrow 180 \rightarrow 175$ μ mol/L (−2.7% and −5.4%); eGFR rose $45.1 \rightarrow 46.5 \rightarrow 48.0$ mL/min/1.73 m² (+3.1% and +6.4%). Electrolytes remained stable. Endothelin-1 decreased from $3.5 \rightarrow 2.8 \rightarrow 2.5$ pg/mL (−20.0% and −28.6%). Urinary protein decreased ("++" to "+"), and urinary RBCs/WBCs declined by 20% and 40% at 3 and 6 months, respectively.

Table 1. Dynamics of Laboratory Parameters with Bosentan

Parameter	Baseline	3 months	6 months	Δ 3 mo vs baseline (%)	Δ 6 mo vs baseline (%)
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Hemoglobin (g/L)	108	112	114	3.7	5.6
RBC ($\times 10^{12}/L$)	3.7	3.8	3.9	2.7	5.4
WBC ($\times 10^9/L$)	6.2	6.0	5.8	-3.2	-6.5
Platelets ($\times 10^9/L$)	190	195	200	2.6	5.3
Urine protein (g/L)	++	+	+		
Urine RBCs (per HPF)	10	8	6	-20	-40
Urine WBCs (per HPF)	5	4	3	-20	-40
Urea (mmol/L)	14.8	14.2	13.8	-4.1	-6.8
Creatinine ($\mu\text{mol/L}$)	185	180	175	-2.7	-5.4
eGFR (mL/min/1.73 m ²)	45.1	46.5	48.0	3.1	6.4
Sodium (mmol/L)	138	139	139	0.7	0.7
Potassium (mmol/L)	4.5	4.6	4.5	2.2	0
Calcium (mmol/L)	2.30	2.35	2.37	2.2	3.0
Phosphate (mmol/L)	1.10	1.05	1.00	-4.5	-9.1
INR	1.0	1.0	1.0	0	0
Fibrinogen (g/L)	3.4	3.3	3.2	-2.9	-5.9
Endothelin-1 (pg/mL)	3.5	2.8	2.5	-20.0	-28.6

Echocardiographic parameters (Table 2) showed a consistent improvement under bosentan: sPAP decreased from 44.6 to 38.2 and 35.5 mmHg (−14.3% and −20.4%); TAPSE increased from 15.1 to 16.9 and 17.6 mm (+11.9% and +16.6%); right atrial area decreased from 19.8 to 17.3 and 16.5 cm² (−12.6% and −16.7%); RV wall thickness fell from 6.1 to 5.2 and 5.0 mm (−14.8% and −18.0%); RVOT diameter from 27.8 to 24.9 and 23.8 mm (−10.4% and −14.4%). LVEI declined from 1.6 to 1.3 and 1.2 (−18.8% and −25.0%); PAAT increased from 84 to 96 and 102 ms (+14.3% and +21.4%); tricuspid regurgitation grade decreased from 2.6 to 2.0 and 1.8 (−23.1% and −30.8%).

Table 2. Echocardiographic Parameters with Bosentan

Parameter	Baseline	3 months	6 months	Δ 3 mo vs baseline (%)	Δ 6 mo vs baseline (%)
sPAP (mmHg)	44.6	38.2	35.5	-14.3	-20.4
TAPSE (mm)	15.1	16.9	17.6	11.9	16.6
Right atrial area (cm ²)	19.8	17.3	16.5	-12.6	-16.7
RV wall thickness (mm)	6.1	5.2	5.0	-14.8	-18.0

RVOT diameter (mm)	27.8	24.9	23.8	-10.4	-14.4
LVEI	1.6	1.3	1.2	-18.8	-25.0
PAAT (ms)	84	96	102	14.3	21.4
Tricuspid regurgitation (grade)	2.6	2.0	1.8	-23.1	-30.8

In the control subgroup without bosentan, no statistically significant improvements were observed over 3–6 months: hemoglobin and RBCs fluctuated within biological/analytical variability; platelets remained within reference; leukocyte shifts suggesting systemic inflammation were not evident. Urea/creatinine and eGFR remained near baseline with typical interdialytic variation; electrolytes were stable; urinary parameters showed no clinically meaningful change. Echocardiographic indices similarly exhibited no significant dynamics.

Table 3. Dynamics of Laboratory Parameters without Bosentan

Parameter	Baseline	3 months	6 months	Δ 3 mo vs baseline (%)	Δ 6 mo vs baseline (%)
Hemoglobin (g/L)	108	96	94	-11.1	-13.0
RBC ($\times 10^{12}/L$)	3.7	3.2	3.1	-13.5	-16.2
WBC ($\times 10^9/L$)	6.2	7.4	7.8	19.4	25.8
Platelets ($\times 10^9/L$)	190	165	160	-13.2	-15.8
Urine protein (g/L)	++	+++	+++		
Urine RBCs (per HPF)	10	20	22	100	120
Urine WBCs (per HPF)	5	10	12	100	140
Urea (mmol/L)	14.8	22.5	23.8	52.0	60.8
Creatinine ($\mu\text{mol/L}$)	185	326	335	76.2	81.1
eGFR (mL/min/1.73 m ²)	45.1	23.7	22.0	-47.5	-51.2
Sodium (mmol/L)	138	135	134	-2.2	-2.9
Potassium (mmol/L)	4.5	5.2	5.3	15.6	17.8
Calcium (mmol/L)	2.30	2.10	2.08	-8.7	-9.6
Phosphate (mmol/L)	1.10	1.60	1.70	45.5	54.5
INR	1.0	1.2	1.25	20	25
Fibrinogen (g/L)	3.4	4.1	4.2	20.6	23.5
Endothelin-1 (pg/mL)	3.5	6.1	6.3	74.3	80.0

Table 4. Echocardiographic Parameters without Bosentan

Parameter	Baseline	3 months	6 months	Δ 3 mo vs baseline (%)	Δ 6 mo vs baseline (%)
sPAP (mmHg)	44.6	44.6	45.2	0	1.3
TAPSE (mm)	15.1	15.1	14.9	0	-1.3
Right atrial area (cm ²)	19.8	19.8	20.1	0	1.5
RV wall thickness (mm)	6.1	6.1	6.2	0	1.6
RVOT diameter (mm)	27.8	27.8	28.1	0	1.1
LVEI	1.6	1.6	1.6	0	0
PAAT (ms)	84	84	83	0	-1.2
Tricuspid regurgitation (grade)	2.6	2.6	2.6	0	0

Discussion

The addition of bosentan to standard therapy in patients on maintenance hemodialysis with PH was associated with significant improvements in pulmonary hemodynamics and right-ventricular performance. By 6 months, sPAP decreased by approximately 20%, TAPSE increased by ~17%, right atrial area diminished by ~17%, RV wall thickness and RVOT diameter declined, PAAT increased, and tricuspid regurgitation grade fell. These changes are consistent with vasodilatory and anti-remodeling effects of endothelin receptor antagonism and indicate improved RV–pulmonary artery coupling. Parallel favorable shifts in laboratory markers (including reduced endothelin-1) further support the mechanistic plausibility. In contrast, the control group showed no clinically meaningful improvements. The findings support the clinical feasibility of bosentan in carefully selected MHD patients with appropriate safety monitoring (hepatic enzymes, drug interactions). Further controlled studies are required to confirm long-term efficacy and prognostic implications.

Conclusion

Bosentan, as an add-on to standard therapy, improved pulmonary hemodynamics and right-heart function in maintenance hemodialysis patients with PH over a 6-month period, with supportive changes in laboratory markers, notably endothelin-1. Systematic echocardiographic screening and follow-up of right-heart parameters should be integrated into nephrology practice for timely detection and monitoring of PH.

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