# Massive right ventricular thrombus formation in a fatal course of hypereosinophilic syndrome: A complex diagnostic approach and interventional management

Maria Binkiewicz-Orluk[1], Agnieszka Wsół[2], Marcin Konopka[3], Edyta Kostarska-Srokosz[1], Wojciech Król[3], Joanna Petryka-Mazurkiewicz[4], Mariusz Kuśmierczyk[5], Wojciech Braksator[3], Marek Kuch[1]

[1]Department of Cardiology, Hypertension and Internal Disease, Medical University of Warsaw, Poland, Mazovia Brodno Hospital, Warszawa, Poland
[2]Department of Experimental and Clinical Physiology, Laboratory of Center for Preclinical Research, Medical University of Warsaw, Warszawa, Poland
[3]Department of Sports Cardiology and Noninvasive Cardiovascular Imaging, Medical University of Warsaw, Warszawa, Poland
[4]Magnetic Resonance Unit, Department of Radiology, National Institute of Cardiology, Warszawa, Poland
[5]Department of Cardiac Surgery, Medical University of Warsaw, Warszawa, Poland

**Correspondence to:**
Maria Binkiewicz-Orluk, MD, Mazovia Brodno Hospital, Kondratowicza 8, 03–242 Warszawa, Poland, phone: +48 663 357 333, e-mail: maria.binkiewicz@gmail.com



DOI: 10.33963/KP.a2022.0241

**Received:**
July 24, 2022

**Accepted:**
September 22, 2022

**Early publication date:**
October 26, 2022

Hypereosinophilia is quite a frequent finding in clinical practice. Common causes of hypereosinophilia include infectious, allergic, neoplastic, and hematological disorders. Another less frequent cause is hypereosinophilic syndrome (HES), characterized by eosinophilic infiltration and damage of various target organs [1]. Therefore, a diagnostic approach in hypereosinophilia should specifically focus on a constellation of signs and symptoms presented by the patient. Heart involvement in HES is present in 58% of cases, and it is potentially life-threatening [2]. The present report aimed to describe a case of heart failure due to large thrombus formation in the right ventricle (RV) and to discuss the diagnostic approach and interventional management in this fatal course of HES.

A 64-year-old woman with a history of arterial hypertension, allergy to multiple drugs, recurrent angioedema episodes, and eosinophilia under observation in the earlier 12 months was admitted to the hospital due to acute dyspnea with concomitant Quincke's edema. In the previous 3–4 months, she experienced progressive exertional dyspnea. Rheumatological, pulmonological, or parasitic backgrounds of hypereosinophilia were excluded.

On admission, the patient presented dyspnea (predominantly platypnea) with desaturation to 85%. Laboratory tests demonstrated marked eosinophilia $2.07 \times 10^9$/l (14%), elevated N-terminal pro-B-type natriuretic peptide (7160.1 pg/ml), D-dimer concentration, and mild respiratory alkalosis with hypoxemia and hypocapnia. Computed tomography pulmonary angiogram excluded pulmonary embolism and revealed a lack of post-contrast enhancement in the RV (Figure 1D), corresponding with the presence of a large mass on transthoracic echocardiography (Figure 1A–C). Bone marrow aspiration and trepanobiopsy ruled out a hematologic cause of hypereosinophilia. Cardiac magnetic resonance confirmed its thrombotic character and revealed global subendocardial fibrosis of the RV (Figure 1E, F). The clinical picture was typical of HES.

After steroid therapy, a decrease in the eosinophilic count was obtained. The 38-day-long anticoagulant treatment did not decrease the size of the thrombus, and heart failure symptoms were still present. The possible treatment pathways, including thrombus aspiration methods [3], were discussed with the multidisciplinary team. Considering the size of the thrombus, ineffectiveness of anticoagulants, and high risk of RV wall damage, the cardiac surgery method to evacuate the thrombus was recommended.

The operation was initially successful. However, in the postoperative period, mechanical ventilation could not be discontinued. The mechanism of the above was unclear, but a rapid increase in right ventricular outflow after surgery, resulting in pulmonary edema and damage of pulmonary capillaries should

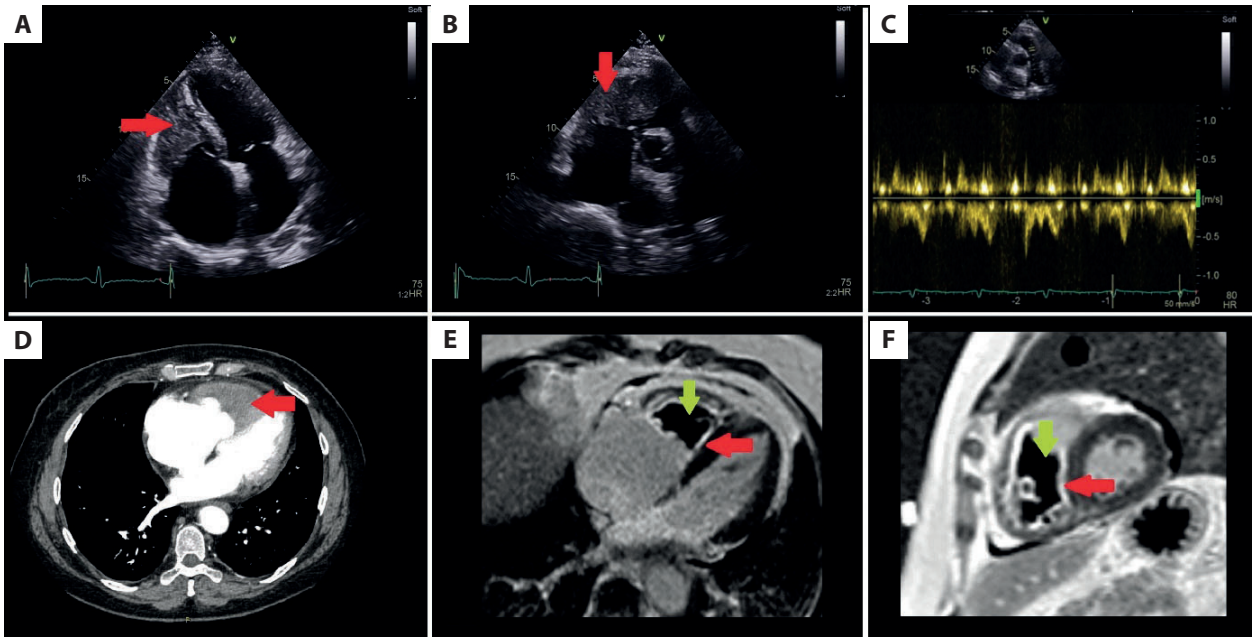


**Figure 1. A–C.** Transthoracic echocardiography: **A.** Apical 4-chamber view. **B.** Parasternal short axis view: the presence of a large mass filling almost completely the right ventricular cavity and right ventricular outflow tract (the red arrows). **C.** Parasternal short axis, the pulsed wave Doppler technique: abnormal spectrum of the pulmonary flow with atypical notching. **D.** Computed tomography angiogram: a lack of post-contrast enhancement in the right ventricle. **E, F.** Cardiac magnetic resonance: **E.** 4-chamber view. **F.** Short axis mid-ventricular view. Late gadolinium enhancement. A giant heterogenic mass filling the right ventricular cavity (the green arrows). The global subendocardial fibrosis of the right ventricle (the red arrows)

be taken into consideration. The patient developed pneumonia and sepsis, which resulted in death.

The presence of intracardiac thrombi was described in other rare systemic diseases [4]. However, there are limited data on the presence and management of intra-cardiac thrombotic complications in the course of HES. Zhang et al. [5] reviewed 477 articles about hypereosinophilia and found 33 cases describing this phenomenon. Intra-cardiac thrombi occurred rarely (about 7%), definitely more often in the left or both ventricles (91%), and they could lead to peripheral embolism. The pharmacological treatment included steroid and anticoagulant therapy, hydroxyurea, and imatinib. Three patients underwent surgery procedures: one died due to septic shock and two were alive, but their prognosis was poor. The overall mortality rate of the group with intracardiac thrombus was high (27.3%).

This case raises important issues: (1) the diagnosis of HES requires multimodality imaging and management engaging a multidisciplinary team; (2) in cases of hypereosinophilia and symptoms suggesting cardiac involvement urgent diagnostic and therapeutic strategy should be applied; (3) regardless of the implemented procedure, the presence of a giant intra-cardiac thrombus in the course of HES significantly worsens the prognosis.

## *Article information*

**Conflict of interest:** None declared.

**Funding:** None.



## REFERENCES

1. Sheikh J, Weller PF. Clinical overview of hypereosinophilic syndromes. Immunol Allergy Clin North Am. 2007; 27(3): 333–355, doi: 10.1016/j.iac.2007.07.007, indexed in Pubmed: 17868853.
2. Pereira NL, Grogan M, Dec GW, et al. Spectrum of restrictive and infiltrative cardiomyopathies: part 1 of a 2-part series. J Am Coll Cardiol. 2018; 71(10): 1130–1148, doi: 10.1016/j.jacc.2018.01.016, indexed in Pubmed: 29519355.
3. Puślecki M, Stefaniak S, Katarzyński S, et al. AngioVac: The first in Poland percutaneous solid thrombus aspiration from the right atrium. Kardiol Pol. 2022; 80(1): 103–104, doi: 10.33963/KP.a2021.0128, indexed in Pubmed: 34643258.
4. Gościniak P, Baumert B, Rajewska-Tabor J, et al. Massive atrial thrombus in sinus rhythm cardiac amyloidosis is not a wild goose chase? Kardiol Pol. 2022; 80(4): 507–508, doi: 10.33963/KP.a2022.0073, indexed in Pubmed: 35546784.
5. Zhang Q, Si D, Zhang Z, et al. Loeffler endocarditis with intracardiac thrombus: case report and literature review. BMC Cardiovasc Disord. 2021; 21(1): 615, doi: 10.1186/s12872-021-02443-2, indexed in Pubmed: 34961478.