European Heart Journal - Case Reports (2024) **8**, ytae415
https://doi.org/10.1093/ehjcr/ytae415

**CASE REPORT**
*Adult congenital heart disease*

# Percutaneous patent foramen ovale closure for hypoxaemia at the age of 73: a case report showing immediate benefit

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*Received 1 December 2023; revised 30 April 2024; accepted 5 August 2024; online publish-ahead-of-print 16 August 2024*

**Background** Approximately 25% of the general population has a patent foramen ovale (PFO) that remains asymptomatic in the vast majority. Right-to-left shunt (RLS)-mediated hypoxaemia is a rare associated condition of PFO.

**Case summary** This report describes a case of percutaneous PFO closure for hypoxaemia in a 73-year-old patient showing immediate clinical benefit. She experienced progressive dyspnoea on exertion requiring oxygen therapy. SaO2 was 87% at rest without oxygen therapy, which increased to 98% after percutaneous PFO closure.

**Discussion** Most PFOs remain clinically insignificant but RLS-mediated hypoxaemia is a rare phenomenon that can occur even at advanced age. Percutaneous PFO closure is a safe and effective therapy option that provides immediate improvement of hypoxaemia.

**Keywords** Patent foramen ovale • Hypoxaemia • Right-to-left shunt • Percutaneous closure • Case report

**ESC curriculum** 9.7 Adult congenital heart disease • 2.1 Imaging modalities

## Learning points

- To recognize right-to-left shunt-mediated hypoxaemia as a rare complication of patent foramen ovale (PFO).
- To demonstrate that percutaneous PFO closure can provide rapid resolution of hypoxaemia even in patients with advanced age.

## Introduction

The foramen ovale plays a crucial part in the foetal blood circulation. After birth, the pulmonary vascular resistance drops and the left atrial pressure rises leading to fusion of the opposing septa in ~75% of the population. In the remaining 25%, fusion of the septa is incomplete resulting in a patent foramen ovale (PFO).[1] The vast majority remains asymptomatic but there are several associated conditions including stroke, migraine, decompression illness, and platypnoea–orthodeoxia syndrome.[1] A less familiar related condition is hypoxaemia based on arterial deoxygenation due to a raise in right-sided cardiac pressure that may create a right-to-left shunt (RLS) through the PFO. We present a case of percutaneous PFO closure for hypoxaemia in a 73-year-old patient showing immediate clinical benefit.

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Handling Editor: Filippo Puricelli
Peer-reviewers: Christoph Sinning; Melonie Johns
Compliance Editor: Nikesh Jathanna

## Summary figure

| Date | Event |
|---|---|
| 2010 | Documented PFO on contrast TTE with large right-to-left shunt |
| 05-2022 | CVA of the left insular cortex (fully recovered without sequelae) |
| 06-2022 | Progressive dyspnoea during exercise<br>- Saturation at rest was 88% (92% with 4L oxygen therapy)<br>- Saturation during exercise was 80% without oxygen therapy<br>- SaO2 was 87% without oxygen therapy |
| 09-2022 | Percutaneous PFO closure for hypoxemia with immediate improvement of SaO2 to 98% without oxygen therapy |
| 10-2022 | No experience of dyspnoea<br>- Normal exercise tolerance<br>- Saturation at rest was 97% without oxygen therapy |
| 05-2023 | Stable situation without dyspnoea, contrast TTE showed a small residual right-to-left shunt |
| CVA, cerebrovascular accident; PFO, patent foramen ovale; SaO2, arterial oxygen saturation; TTE, transthoracic echocardiography | |

## Case presentation

A 73-year-old woman known with a documented PFO in 2010 on contrast echocardiography and a recent cerebrovascular accident of the left insular cortex in 2022 was referred to our outpatient clinic for hypoxaemia due to right-to-left shunting. During the recent hospital stay, computed tomography excluded pulmonary embolism or pneumonia. At the outpatient clinic, she experienced progressive shortness of breath for the last 6 months during exercise but not at rest. At physical examination, her blood pressure was 113/83 mmHg, heart rate 90 b.p.m., and saturation 88% at rest (92% with 4 L of oxygen therapy, no difference between upright and supine position). During exercise, the saturation decreased to 80% without oxygen therapy. There were no other abnormal findings during the pulmonary examination. Additional laboratory tests did not reveal a liver- or autoimmune-mediated disease. Right heart catheterization was performed in supine position and in rest, which showed a normal capillary wedge pressure (6 mmHg), pulmonary artery pressure (mean 12 mmHg), right atrium pressure (6 mmHg), cardiac output (5.3 L/min), and pulmonary vascular resistance (91 dyn·s/cm$^5$). The arterial oxygen saturation (SaO2) was 87% without oxygen therapy. Transthoracic echocardiography revealed a normal function and dimensions of the left ventricle, a moderately reduced right ventricle function with normal dimensions, and mild dilatation of the aortic root (42 mm) with mild aortic regurgitation. There was a floppy interatrial septum (*Figure 1*) and after injection of agitated saline contrast, a large RLS (>25 bubbles) was noted suspicious for a PFO (*Figure 2* and Supplementary material online, *Video 1*).

Our multidisciplinary team advised percutaneous PFO closure for hypoxaemia requiring oxygen therapy in absence of pulmonary hypertension, pulmonary disease, or emboli. The closure was performed under general anaesthesia with transoesophageal echocardiography guidance. Balloon sizing measured a defect of 16 mm (*Figure 3*). Patent foramen ovale closure was performed using a 30 mm Amplatzer™ PFO occluder device (Abbott, Chicago, IL, USA) where after no residual shunt was observed by agitated saline at the end of the procedure. Immediately post-procedure, the SaO2 increased above 95% and the oxygen therapy was discontinued after which the SaO2 was 98%. At 6-week follow-up, the patients' saturation was 97% and she had a normal exercise tolerance. Six months after the procedure, the patients' situation was stable, she did not experience any dyspnoea, and transthoracic echocardiography with contrast bubble study showed a small residual RLS.

## Discussion

Arterial hypoxaemia is defined as a SaO2 < 90% or arterial oxygen pressure < 60 mmHg with or without cyanosis. The main symptom is exertional or resting dyspnoea, and there are several known causes including platypnoea–orthodeoxia syndrome, obstructive sleep apnoea syndrome, chronic obstructive pulmonary disease, exercise desaturation, and high-altitude pulmonary oedema. In a recent meta-analysis including five studies, PFO closure in desaturation syndromes showed a significant increase in SaO2 of 9.8% (95% CI: 7.1–12.5%) in exercise desaturation and 9.6% (95% CI: 5.7–13.5%) in platypnoea–orthodeoxia syndrome.[2] Moreover, percutaneous PFO closure has proved to be successful in selected patients with underlying pulmonary diseases.[3–6] In addition, several case series showed that percutaneous PFO closure is a safe and effective therapy for resolving RLS-mediated hypoxaemia with rapid improvement of 10% SaO2 and complete resolution in four patients with unique underlying diseases (1× hemi diaphragmatic paralysis, 1× Ebstein anomaly and history of ascending aortic aneurysm repair, 1× tricuspid flail leaflet with hypermobile interatrial septum and iatrogenic right phrenic-nerve palsy, 1× valve-in-valve TAVR).[7] In our case, closure of the PFO showed immediate increase in arterial oxygen saturation and the exercise performance was restored.

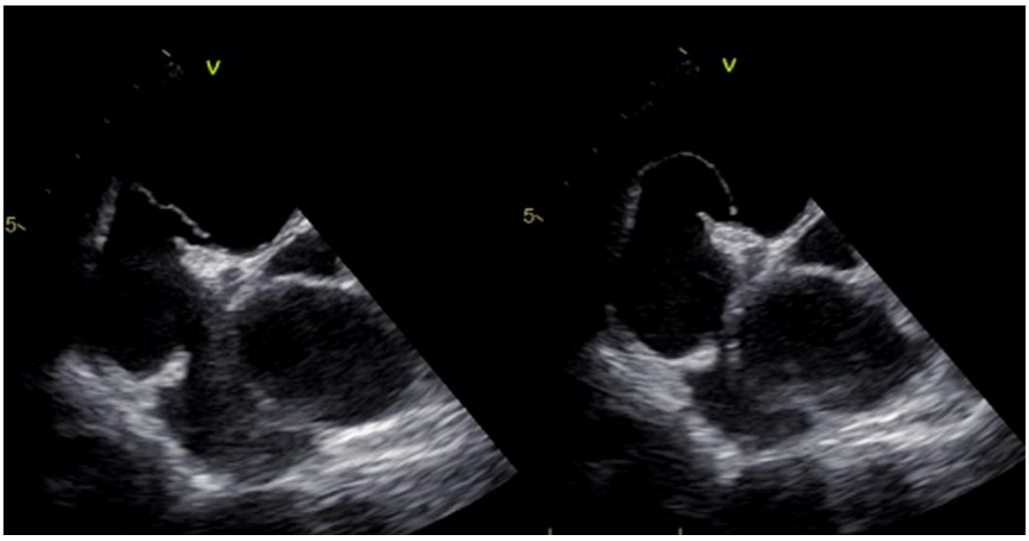

**Figure 1** Transthoracic echocardiography of the PFO with a hypermobile atrial septum.

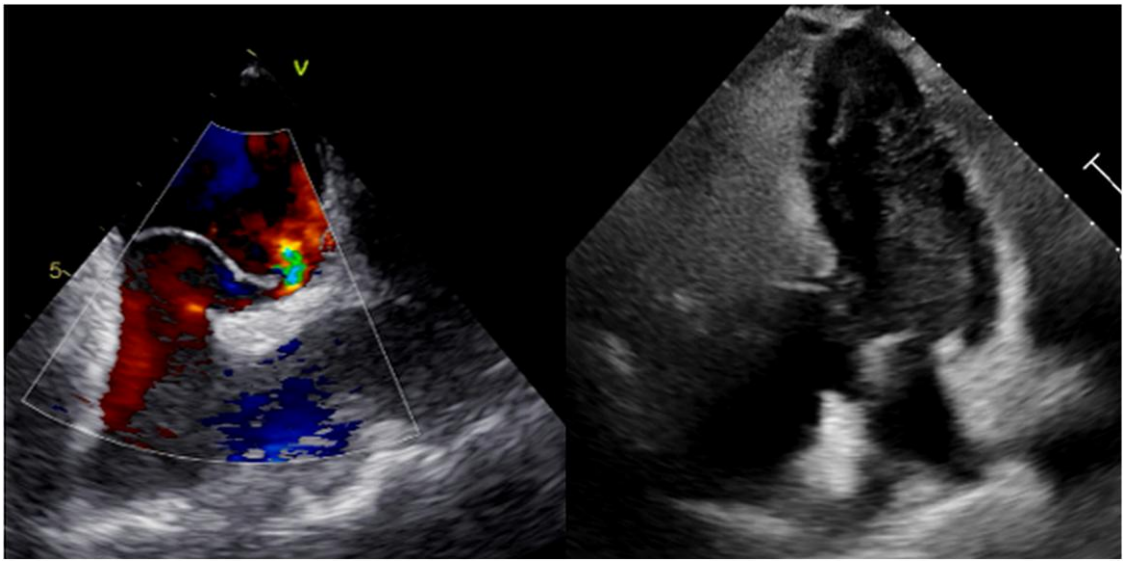

**Figure 2** Transthoracic echocardiography of the PFO with Doppler and agitated saline contrast (bubble study).

The European position paper on the management of PFO in arterial deoxygenation syndromes recommends to: (i) individually assess and weigh the role of all factors involved in the desaturation syndrome, (ii) obtain invasive evidence of the PFO role if possible, and (iii) when appropriate, propose PFO closure with shared decision-making underscoring the lack of evidence. Furthermore, it advises to *not* routinely close the PFO, especially in the presence of severe chronic pulmonary hypertension or without clear evidence of a crucial role in desaturation.[2]

The guidelines from the Society of Cardiovascular Angiography and Interventions (SCAI) suggest PFO closure for platypnoea–orthodeoxia syndrome with a conditional recommendation and a very low certainty of evidence, based on the improvement of oxygenation and quality of life in the majority of patients who underwent PFO closure.[8] The SCAI guidelines have no specific recommendations on PFO closure for non-platypnoea–orthodeoxia syndrome hypoxaemia and encourage future research for these conditions.

Since our patient experienced symptoms of hypoxaemia, we performed multiple tests and invasive measurements in accordance with the European position paper and SCAI guidelines, to analyse the desaturation syndrome and the role of the PFO, and to exclude pulmonary hypertension, pulmonary disease, or emboli before considering percutaneous PFO closure to relieve the symptoms. However, the exact mechanism of the hypoxaemia in our patient is not completely understood as platypnoea–orthodeoxia syndrome was excluded. We postulated that her symptoms might be related to the growth of the aortic

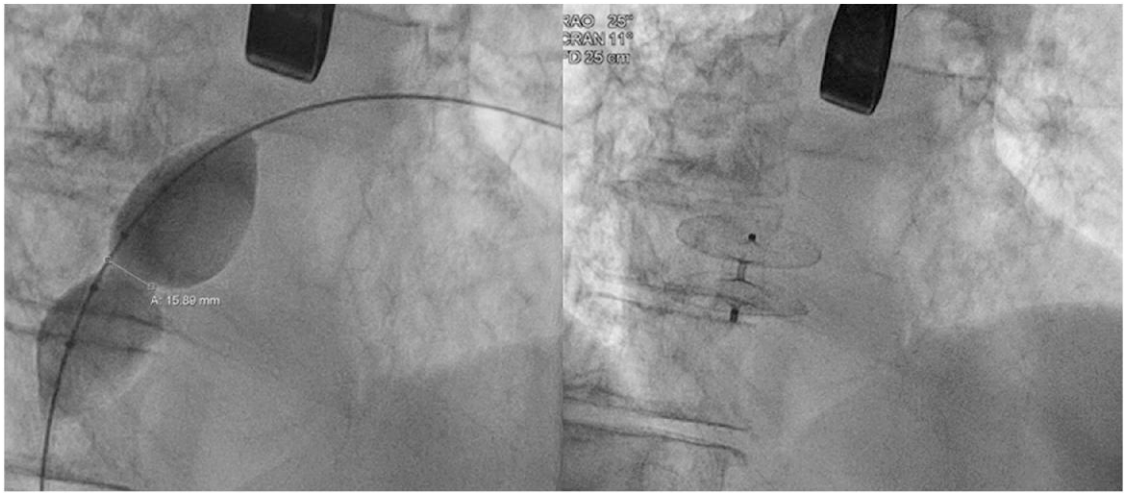


**Figure 3** Procedural angiography of the PFO with balloon sizing and the final result of the implanted PFO occluder.

root 34 mm in 2013 to 43 mm in 2022 together with a decrease in her body height from 168 cm in 2010 to 159 in 2022. This may have changed the position of the atrial septum relative to the aortic root and thereby affected the RLS size, i.e. opened up the PFO.

## Conclusion

Most PFOs remain clinically insignificant but RLS-mediated hypoxaemia is a rare phenomenon that can occur even at advanced age. It is important to perform a thorough work-up with imaging modalities, functional tests, and invasive measurements to analyse the role of PFO and exclude underlying mechanisms and pulmonary diseases. Percutaneous PFO closure is a safe and effective therapy option that provides immediate improvement of hypoxaemia.

## Lead author biography

Lars S. Witte completed his study in medicine at the Vrije Universiteit van Amsterdam, The Netherlands in 2019 and is currently a medical doctor and PhD candidate at the University of Amsterdam at the Department of Cardiology.

## Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports* online.

## Acknowledgements

Special thanks to Abdelhak el Bouziani, MD, and Danielle Robbers-Visser, MD, PhD, for their contribution.

**Consent**: The authors confirm that written consent for submission and publication of this case report including the images and associated text has been obtained from the patient in line with COPE guidance.

**Conflict of interest:** None declared.

**Funding:** None.

## Data availability

The data underlying this article are available in the article and in its online Supplementary material.

## References

1. Hara H, Virmani R, Ladich E, Mackey-Bojack S, Titus J, Reisman M, et al. Patent foramen ovale: current pathology, pathophysiology, and clinical status. *J Am Coll Cardiol* 2005;**46**:1768–1776.
2. Pristipino C, Germonpre P, Toni D, Sievert H, Meier B, D'Ascenzo F, et al. European position paper on the management of patients with patent foramen ovale. Part II—decompression sickness, migraine, arterial deoxygenation syndromes and select high-risk clinical conditions. *EuroIntervention* 2021;**17**:e367–e375.
3. Godart F, Rey C, Prat A, Vincentelli A, Chmaït A, Francart C, et al. Atrial right-to-left shunting causing severe hypoxaemia despite normal right-sided pressures. Report of 11 consecutive cases corrected by percutaneous closure. *Eur Heart J* 2000;**21**:483–489.
4. Fenster BE, Nguyen BH, Buckner JK, Freeman AM, Carrol JD. Effectiveness of percutaneous closure of patent foramen ovale for hypoxemia. *Am J Cardiol* 2013;**112**:1258–1262.
5. Mojadidi MK, Ruiz JC, Chertoff J, Zaman MO, Elgendy IY, Mahmoud AN, et al. Patent foramen ovale and hypoxemia. *Cardiol Rev* 2019;**27**:34–40.
6. Tobis JM, Narasimha D, Abudayyeh I. Patent foramen ovale closure for hypoxemia. *Interv Cardiol Clin* 2017;**6**:547–554.
7. Robl J, Vutthikraivit W, Horwitz P, Panaich S. Percutaneous closure of patent foramen ovale for treatment of hypoxemia: a case series and physiology review. *Catheter Cardiovasc Interv* 2022;**100**:471–475.
8. Kavinsky CJ, Szerlip M, Goldsweig AM, Amin Z, Boudoulas KD, Carroll JD, et al. SCAI guidelines for the management of patent foramen ovale. *J Soc Cardiovasc Angiogr Interv* 2022;**1**:100039.