Findings that shed new light on the possible pathogenesis of a disease or an adverse effect

Recurrent episodic hypoxaemic respiratory failure following a stroke

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Summary

A 68-year-old man with no cardiovascular risk factors was admitted with a stroke because of multiple brain infarcts in different vascular territories. He required mechanical ventilation for hypoxia as a result of aspiration pneumonia. Subsequent recovery was hindered by episodic, unexplained hypoxia. Investigations excluded pulmonary embolism, pulmonary hypertension and severe lung diseases. Transthoracic echocardiography (TTE) with saline bubble contrast showed mild, delayed, right-to-left shunting, thought to represent an insignificant, intrapulmonary, non-cardiac shunt. Hypoxic episodes worsened, requiring admission from community rehabilitation hospital to our centre and another period of mechanical ventilation. Elevated alveolar-arterial gradients indicated a non-hypoventilatory cause. Repeat TTE bubble contrast study and transoesophageal echocardiography (TOE) demonstrated a patent foramen ovale (PFO) with large shunt potential, associated with an aneurysmal interatrial septum. This provided a unifying explanation for cryptogenic stroke and recurrent hypoxaemia. After percutaneous PFO closure hypoxic episodes ceased and he returned successfully to rehabilitation.

BACKGROUND

Patent foramen ovale (PFO) with a right-to-left inter-atrial shunt (RLIAS) in the absence of pulmonary hypertension is an under-recognised but readily treatable cause of hypoxia. It is readily missed, particularly in the presence of other potential causes of hypoxia, as in this case of cryptogenic stroke complicated by aspiration pneumonia and hypoventilation syndrome. Awareness, vigilance and comprehensive attention to detail are required to detect the mismatch between severity of the hypoxia and the severity of its known causes, which raises suspicion of this syndrome. RLIAS may be intermittent, giving rise to false-negative results with the standard assessment techniques of bubble contrast studies using TTE or transcranial doppler ultrasound. Our case presentation seeks to raise awareness of this uncommon syndrome, add to the growing body of knowledge on this poorly understood disease, highlight the difficulties encountered in its diagnosis and demonstrate the benefits of its recognition and treatment.

CASE PRESENTATION

Our patient is a 68-year-old Chinese man who was admitted for recurrent episodes of hypoxia following a stroke. His only other medical history was schizophrenia for 15 years, for which he was on clozapine. Two months prior to admission, he was admitted to another hospital for a stroke owing to infarcts in his left pons, left cerebellar peduncle, left cerebellar hemisphere and left temporal lobe, complicated by aspiration pneumonia with hypoxaemic respiratory failure requiring mechanical ventilation.

He was successfully weaned off ventilation but subsequently still suffered occasional episodes of hypoxia. CT

pulmonary angiogram (CTPA) showed no evidence of pulmonary embolism, severe lung disease or arteriovenous malformation. Standard TTE did not demonstrate any intracardiac shunt. Bubble contrast TTE showed right-to-left shunting with mild delay in the appearance of bubble contrast in the left atrium (LA) suggestive of minor intrapulmonary shunting (figure 1). The patient

Figure 1 Transthoracic echocardiography with bubble contrast study showing non-delayed appearance of bubble contrast in the left atrium and left ventricle after injection into the venous system.

was no longer significantly hypoxic. The medical team concluded that this intrapulmonary shunt did not warrant further investigation or intervention and discharged him to a community rehabilitation hospital. Unfortunately, he made poor progress as he became hypoxic whenever he underwent physiotherapy.

On admission to our institution, the patient was intubated at the emergency department for hypoxaemia. His ventilator settings in the intensive care unit encompassed pressure support of 10 cm H₂O and positive end-expiratory pressure (PEEP) of 5 cm H₂O. He managed to maintain an oxygen saturation of 96% on the above ventilatory settings. He was extubated shortly and transferred to the general ward. Clinical examination was normal. However, his episodic hypoxia became more frequent with oxygen saturation on pulse oximetry dropping from $\geq 95\%$ between episodes to 62–92% on room air. Even when severely hypoxic, he remained largely comfortable, and was at worst only mildly tachypnoeic at 20 breaths/min. Administration of 50–60% fraction of inspired oxygen (FiO₂) using a Venturi mask and 100% FiO₂ with a non-rebreather mask raised his oxygen saturation to 90% and 98%, respectively. Central cyanosis was never noted. Hypoxic episodes were associated with activities such as feeding, physiotherapy and straining at stool or micturition, and posture, being worst when upright and best lying in the lateral position.

DIFFERENTIAL DIAGNOSIS

The patient was initially treated for aspiration pneumonia and hypoventilation when he first presented with hypoxia after the stroke but subsequent episodes of hypoxaemia were not readily explained. Deep venous thrombosis with pulmonary embolism, pulmonary oedema, pulmonary hypertension and interstitial lung disease were excluded by normal chest x-ray, TTE, lower-limb doppler ultrasound, CTPA and lung function tests. Arterial blood gases revealed type 1 respiratory failure with severe hypoxaemia without hypercarbia. Type 2 respiratory failure owing to obstructive sleep apnoea, central hypopnoea and neuromuscular disease was unlikely with elevated alveolar-arterial gradients, occasional mild tachypnoea and no hypercapnoea during hypoxia. Sleep study was inconclusive owing to poor sleep efficiency. Nerve conduction study and electromyography were normal. Repeat MRI of the brain confirmed normal anterior and posterior circulatory systems.

Exclusion of the common differential diagnoses for hypoxia in combination with multiple acute brain infarcts in different vascular territories led us to revisit the possibility of RLIAS causing hypoxia and paradoxical embolisation with stroke.

Repeat TTE with bubble contrast demonstrated the appearance of bubbles in the LA within three cardiac cycles of bubble contrast filling the right atrium (RA) suggestive of shunting at the atrial level. Trans-oesophageal echocardiogram demonstrated a PFO with large shunt potential, mild unprovoked RLIAS. PFO anatomy was complex with a thick septum secundum, left atrial tethering of the PFO flap and an aneurysmal interatrial septum, suitable for percutaneous device closure but with a higher risk of failure.

TREATMENT

The patient was started on anticoagulation and subsequently underwent percutaneous closure of his PFO under general anaesthesia. Systolic/diastolic/mean RA, LA and pulmonary artery pressures were normal at cardiac catheterisation at 6/5/5, 9/6/7 and 30/9/16 mm Hg, respectively. Oxygen saturation was 98% in both right and left pulmonary veins, but only 90% in the LA, confirming significant RLIAS. PFO diameter by Amplatzer balloon-sizing was 14 mm on transoesophageal echocardiography (TOE) and 15 mm on fluoroscopy. A 17 mm Amplatzer septal occluder was deployed across the 14 mm PFO successfully without complications.

OUTCOME AND FOLLOW-UP

The patient's oxygen saturation on room air improved from 96% on 40% FiO₂ to 91% on RA immediately post-procedure and to 95% on RA 2 days postclosure, with mild hypocapnoea indicating some residual RLIAS and no significant hypoventilation. He was discharged well and underwent rehabilitation for stroke with no further hypoxaemia. Six weeks postclosure, his oxygen saturation was 98% on room air.

DISCUSSION

Two possible mechanisms by which PFO and atrial septal aneurysm (ASA) may have caused stroke in our patient are paradoxical embolism and intracardiac thrombus formation.

There is no doubt that paradoxical embolism does occur. Cases of right-to-left passage of thrombi through PFO have been unequivocally documented.^{1–4} Many studies have documented increased incidence of PFOs in patients with cryptogenic stroke,^{5–11} with the largest (250 patients) multicentre cohort study (Patent Foramen Ovale in Cryptogenic Stroke Study, 2002) reporting an incidence of PFO of 39.2% and an incidence of large PFO of 20% compared to 15–35% in the normal population.¹² Larger PFO diameter, greater shunt potential^{1–4} and presence of ASA,⁵ possibly because of the association of ASA with larger PFO size, are associated with increased risk of stroke. This would be consistent with paradoxical embolism as an important mechanism of stroke.

Many observational studies have shown superiority of PFO closure over medical therapy in stroke prevention.¹¹ However, the only major randomised trial to date of PFO closure for cryptogenic stroke (CLOSURE-I)¹³ failed to show significant benefit of PFO closure with the STARFlex device over medical therapy in stroke prevention, raising doubts that paradoxical embolism is the predominant cause of cryptogenic stroke. Possible alternative explanations for recurrent transient ischemic attack (TIA) or stroke were apparent in 20 of 23 patients who suffered recurrent stroke or TIA within 2 years of PFO closure. This trial does not exclude potential benefit from PFO closure in selected subgroups, but criteria for identification of such patients are unknown. A propensity-matched study using a variety of closure devices showed a low-stroke recurrence rate similar to CLOSURE-I but significant reduction in recurrent stroke and TIA over 15 years.¹⁴ Longer follow-up and use of different closure devices may

show benefit, and results of other randomised trials are awaited.¹⁰

It has been suggested that PFO and ASA may increase susceptibility of some patients to atrial flutter or atrial fibrillation (AF), and hence intracardiac thrombus formation, by providing a structural substrate for AF and subtle distortion of normal cardiac haemodynamics.^{15–18} In such cases, PFO closure would not be beneficial and could potentially increase risk by causing AF.¹⁹ The CLOSURE-I trial¹³ reported an increased incidence of AF after percutaneous PFO closure (5.7% vs. 0.7% in the medical therapy group). There is increasing evidence that occult paroxysmal AF is significantly underdiagnosed in cryptogenic ischaemic stroke and associated with increased risk of stroke.²⁰

Our patient had no evidence of AF, and did suffer acute brain infarcts in multiple vascular territories, which is suggestive of an embolic cause. Unlike many of the patients included in the CLOSURE-I trial,¹³ he had no cardiovascular risk factors. Paradoxical embolism remains a possible cause of his stroke and PFO closure may incidentally reduce his risk of recurrent stroke.

However, the primary reason for PFO closure in our patient was recurrent severe hypoxia.

He had multiple reasons for development of severe RLIAS without significant pulmonary hypertension. His stroke may have caused centrally mediated hypoventilation. Central hypopnoea may have been absolute and severe in the acute phase, but improved with recovery from acute illness to only mild susceptibility and relative hypoventilation for the degree of hypoxia. His lack of respiratory distress and minimal tachypnoeic response to hypoxia is suggestive of loss of hypoxic drive.

Hypoventilation increases pulmonary and right heart pressures.²¹ This increase may be sufficient for RA pressure to exceed LA pressure without pulmonary hypertension and precipitate RLIAS. In turn, RLIAS worsens hypoventilation by mechanisms that are poorly understood, and hypoventilation may improve or even resolve completely with PFO closure, as has been documented in obstructive sleep apnoea.²²

The two episodes of mechanical ventilation with PEEP may have increased our patient's RLIAS shunt fraction^{23 24} through a mechanism akin to the Valsalva manoeuvre. Increased expiratory intrathoracic pressure with PEEP retards systemic venous return. When intrathoracic pressure drops suddenly during the inspiratory release phase, RA pressures rise more in comparison with the lesser-filled LA, establishing a temporary pressure gradient across a PFO that facilitates RLIAS.^{25 26}

Neurogenic cardiomyopathy with ECG changes and left ventricular dysfunction is well documented in association with major intracranial events.²⁷ Our patient's stroke may have caused subtle cardiomyopathy with impaired diastolic right ventricular (RV) function without systolic dysfunction of either ventricle, thus increasing the RA-to-LA pressure gradient. RV strain with aspiration pneumonia and mechanical ventilation with PEEP may also have contributed to RV diastolic dysfunction.

Recurrent central hypoventilation and RLIAS-exacerbated hypoxia may have perpetuated RV diastolic dysfunction after recovery from the acute illness. Hypoxia in normal subjects is associated with impaired diastolic function but preserved systolic function of both ventricles.²⁸

The potential importance of RV dysfunction in RLIAS is also supported by a case report of profound cyanosis in which the only predisposing factor to RLIAS was right-ventricular diastolic dysfunction.²⁹ Milder degrees of RV dysfunction may be insufficient to cause RLIAS, but sufficient to increase susceptibility to RLIAS with provocation. This could explain the intermittent nature of some RLIAS-related hypoxic syndromes, as seen in our patient.

His intermittent hypoxia after extubation was associated with a number of provocative factors for RLIAS. Episodic centrally mediated hypoventilation has already been discussed above. Inadvertent Valsalva manoeuvres would have occurred during micturition, defaecation and physiotherapy activities that involved straining. Inspiration can induce leftward deviation of the septum primum,³⁰ which reflects higher pressures in the RA than LA.^{31 32} The Valsalva manoeuvre accentuates inspiratory pressure changes, increasing RLIAS through increased pressure gradient and wider PFO opening. Provocation by upright posture was evident with platypnoea orthodeoxia syndrome although the mechanism is unclear. One possibility is atrial septal distortion from gravitational changes in cardiac position. Another is reduced RV compliance relative to the LV through RV-LV interdependence. Assumption of an upright posture causes a sudden decrease in RV filling from decreased venous return, while LV filling is unimpaired.^{30 33}

Diagnosis of RLIAS requires awareness, with clinical suspicion raised when there is a mismatch between working diagnoses and severity of hypoxia. Detection and assessment of PFO by echocardiography may not be straightforward.³⁴ Diagnosis was delayed in our patient by the misleading result of the first TTE bubble study. Understanding the limitations of investigations and persistence in the search for a satisfactory explanation of incongruous clinical findings were crucial to our patient's correct diagnosis.

The intermittent nature and variable severity of his hypoxia demonstrate the marked variability in shunt size with RLIAS according to the prevailing haemodynamic conditions. Failure of a large PFO to open widely at the time of bubble study may result in misleading false-negative or weak-positive results. Delay in opening of the PFO may delay transit of bubbles to the LA, giving the impression of intrapulmonary rather than intracardiac shunting. PFO are often not visible on TTE, and their presence is only inferred by the appearance of bubbles in the LA. Transcranial doppler bubble studies, which were not performed in our patient, are subject to the same limitations.

TOE is often regarded as the gold standard test for PFO as image resolution is much better than with TTE. PFO, atrial septal defect and their associated shunts may be visualised directly, and their anatomical suitability for percutaneous closure assessed. However, false-negative TOE can occasionally occur if a PFO does not open spontaneously, since provocative manoeuvres, such as the Valsalva, are less satisfactory during TOE (figure 2).

Benefit from PFO closure for hypoxia is immediate with complete normalisation of oxygenation at device implantation in most patients.³⁵ Partial resolution owing to incomplete PFO closure at device implantation may improve to normality over time as in our patient.

Figure 2 Transoesophageal echocardiography (TOE) showing blood flow through the patent foramen ovale (marked by *) from RA to LA. (RA=right atrium, LA=left atrium and IAS=interatrial septum).

Amplatzer septal occluders are porous and invariably allow mild shunting through the device immediately post-implantation. The flat design of most closure devices may not conform ideally to complex PFO three-dimensional anatomy, increasing the residual shunt.³⁴ However, many residual shunts resolve over the following months³⁶ as porous constructs thrombose, and devices adjust slightly in position and shape to conform better, adhere and endothelialise.

Whereas benefits of percutaneous PFO closure for stroke prevention are uncertain, the excellent outcomes of PFO closure for RLIAS-mediated hypoxia are immediate and well documented.^{22–35} The procedure carries a low risk even in sick patients³⁵ with high success rate,^{10–13–37–38} Hypoxia due to RLIAS is uncommon, underrecognised and serious but eminently treatable, and care must be taken not to miss this diagnosis.

Learning points

- ▶ The possibility of a venoarterial shunt should always be considered when severity of hypoxaemia is out of proportion to the known diagnoses.
- ▶ A right-to-left inter-atrial shunt (RLIAS) is under-recognised but eminently treatable at low risk with excellent results.
- ▶ It is important to persevere in the search for a satisfactory explanation of incongruity between clinical presentation and investigation results.
- ▶ The limitations of investigations must be understood and investigations repeated or escalated if false negatives are strongly suspected.
- ▶ Transthoracic echocardiography (TTE) bubble study may be falsely negative in RLIAS, particularly if RLIAS is intermittent. TTE bubble study and trans-oesophageal echocardiography are complementary and both should be performed in the assessment of RLIAS.

Competing interests None.

Patient consent Obtained.

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