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Right Ventricular Intramyocardial Dissecting Hematoma Caused by Contained Rupture of an Infective Pseudoaneurysm of the Right Coronary Sinus of Valsalva: A Case Report

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Abstract

Periannular extension of native aortic valve infective endocarditis may lead to abscess formation, pseudoaneurysms, and fistulous communications with adjacent cardiac structures. We report an unusual complication involving a right ventricular intramyocardial dissecting hematoma caused by contained rupture of an infective pseudoaneurysm of the right sinus of Valsalva.

A 29-year-old woman presented with severe chest pain radiating to the back, rapidly progressive dyspnea, and hemodynamic shock following one month of intermittent fever. Computed tomography angiography revealed massive hemopericardium and a suspected ruptured pseudoaneurysm of the right sinus of Valsalva. Emergency surgery initially revealed active bleeding from a right ventricular intramyocardial dissecting hematoma following evacuation of the massive hemopericardium. A subsequent aortic exploration showed destructive infective endocarditis of the native aortic valve, complicated by a pseudoaneurysm of the right sinus of Valsalva that had ruptured in a contained way into the free wall of the right ventricle. Radical debridement, a modified Bentall replacement, and coronary revascularization using a Y-shaped saphenous vein graft were performed. Despite emergency surgery, the patient died from refractory postoperative vasoplegic shock.

This case demonstrates an unusual pathway of rupture of a right sinus of Valsalva pseudoaneurysm in destructive native aortic valve infective endocarditis, in which intramyocardial dissection of the right ventricular free wall preceded hemopericardium and cardiac tamponade.

Categories: Cardiology, Cardiac/Thoracic/Vascular Surgery, Infectious Disease
Keywords: bentall procedure, cardiac tamponade, hemopericardium, infective endocarditis, intramyocardial dissecting hematoma, sinus of valsalva pseudoaneurysm

Introduction

Infective endocarditis (IE) remains a challenging disease with substantial morbidity and mortality despite advances in medical therapy and cardiac surgery. Among its most severe complications is periannular extension, which may result in abscess formation, pseudoaneurysms, or fistulous communications with adjacent cardiac structures and frequently requires surgical intervention [1-3]. Periannular complications occur in approximately 10%-30% of native valve IE cases, particularly in aortic valve infections. Echocardiography plays a central role in their diagnosis, while cardiac computed tomography provides complementary anatomical information, especially when abscesses or pseudoaneurysms are suspected [4]. Pseudoaneurysms of the right sinus of Valsalva may rupture into right-sided cardiac chambers, whereas extension into the ventricular free wall is distinctly uncommon.

Myocardial dissection associated with sinus of Valsalva aneurysms has previously been reported, including involvement of the interventricular septum and left ventricular wall [5,6]. However, these reports did not describe an infective origin, right ventricular free wall involvement, or hemopericardium. We report an unusual case in which a contained rupture of an infective pseudoaneurysm of the right sinus of Valsalva dissected into the right ventricular free wall, forming an intramyocardial dissecting hematoma that subsequently caused massive hemopericardium and cardiac tamponade. The anatomical pathway, rather than tamponade itself, is the main focus of this case.

Case Presentation

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A 29-year-old woman with no known medical history presented to the emergency department with severe acute chest pain radiating to her back, rapidly worsening shortness of breath, and hemodynamic shock. She reported intermittent undocumented fever for the past month, for which she had not seen a doctor. She had no known cardiovascular disease or risk factors, no prior surgeries, no regular medications, and no history of intravenous drug use. On examination, she was afebrile, hypotensive (90/50 mmHg), and tachycardic (105 beats/min), with muffled heart sounds, cold extremities, and weak peripheral pulses, consistent with a low cardiac output state.

In the context of sudden-onset chest pain radiating to the back, rapidly progressing shortness of breath, and hemodynamic shock, the differential diagnosis included acute aortic syndrome and pulmonary embolism. Acute coronary syndrome was considered less likely given the patient's young age, the absence of cardiovascular risk factors, and the nature of the chest pain. Infective endocarditis was not initially suspected because she was afebrile on admission.

Laboratory tests were requested immediately upon admission. However, due to the patient's hemodynamic instability and the initial suspicion of an acute aortic syndrome, an emergency CT angiography was performed without delay. The results showed a huge hemopericardium (Figure *1a*) and a cavity filled with contrast material next to the right coronary sinus, suggesting a possibly ruptured aortic root pseudoaneurysm (Figures *1b*, *1c*).

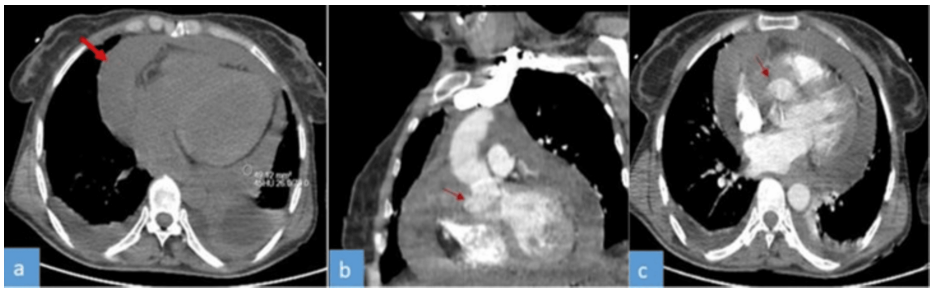


FIGURE 1: Preoperative computed tomography angiography

(a) Unenhanced axial image showing massive hemopericardium (45 HU; arrow). (b, c) Coronal and axial arterial-phase images demonstrating the right coronary sinus of Valsalva pseudoaneurysm (arrow) and associated hemopericardium.

HU: Hounsfield Units

The patient was immediately taken to the operating room (OR) for emergency surgery without a preoperative transthoracic echocardiogram because she was hemodynamically unstable. Laboratory results subsequently revealed anemia, leukocytosis, markedly elevated inflammatory markers, and increased troponin levels (Table *1*). The troponin elevation may have reflected direct myocardial injury associated with the right ventricular intramyocardial dissecting hematoma, although myocardial ischemia related to coronary ostial involvement could not be excluded.

Parameter	Patient value	Reference range
Hemoglobin	8.3 g/dL	13.0–16.0 g/dL
White blood cell count	16.4 ×10³/μL	4.0–10.0 ×10³/μL
C-reactive protein	245 mg/L	<5 mg/L
Serum urea	0.5 g/L	0.18–0.55 g/L
Creatinine	12 mg/L	7.2–12.5 mg/L
Troponin I	246 ng/L	<34.2 ng/L
Platelet count	152 × 10³/μL	150–450 × 10³/μL

TABLE 1: Laboratory findings on admission

Note: Reference ranges are based on those provided by our institutional laboratory. The hemoglobin reference interval is the same for male and female patients.

Blood cultures obtained in the operating room before skin incision and antibiotic administration subsequently grew *Staphylococcus aureus*, with results becoming available seven days after surgery.

Empirical intravenous antibiotic therapy with imipenem and amikacin was initiated before skin incision to provide broad-spectrum antimicrobial coverage in the setting of profound hemodynamic shock and an undetermined infectious source. Emergency surgery was performed through a median sternotomy. Upon opening the inflamed pericardium, approximately 800 mL of hemopericardium was evacuated. Localized cardiopericardial adhesions were subsequently released, revealing active bleeding over a broad area of the anterior right ventricular free wall near its base, without a discrete epicardial tear (Video *1*).



VIDEO 1: Initial intraoperative findings

Active bleeding from a right ventricular intramyocardial dissecting hematoma following rupture of an infective right coronary sinus of Valsalva pseudoaneurysm.

View video here: https://youtu.be/hN1-y1BKHwg?si=OC8c60TKSA24BCTo

Cardiopulmonary bypass was established via ascending aortic and bicaval venous cannulation. Following aortic cross-clamping, myocardial arrest was achieved using antegrade cardioplegia delivered directly into the coronary ostia, and a transverse aortotomy was performed. Intraoperative examination revealed extensive destruction of the native aortic valve secondary to infective endocarditis, with vegetations and perforations involving both the right and left coronary cusps, severe periannular tissue destruction, and a pseudoaneurysm arising from the right coronary sinus of Valsalva (Figure *2*).

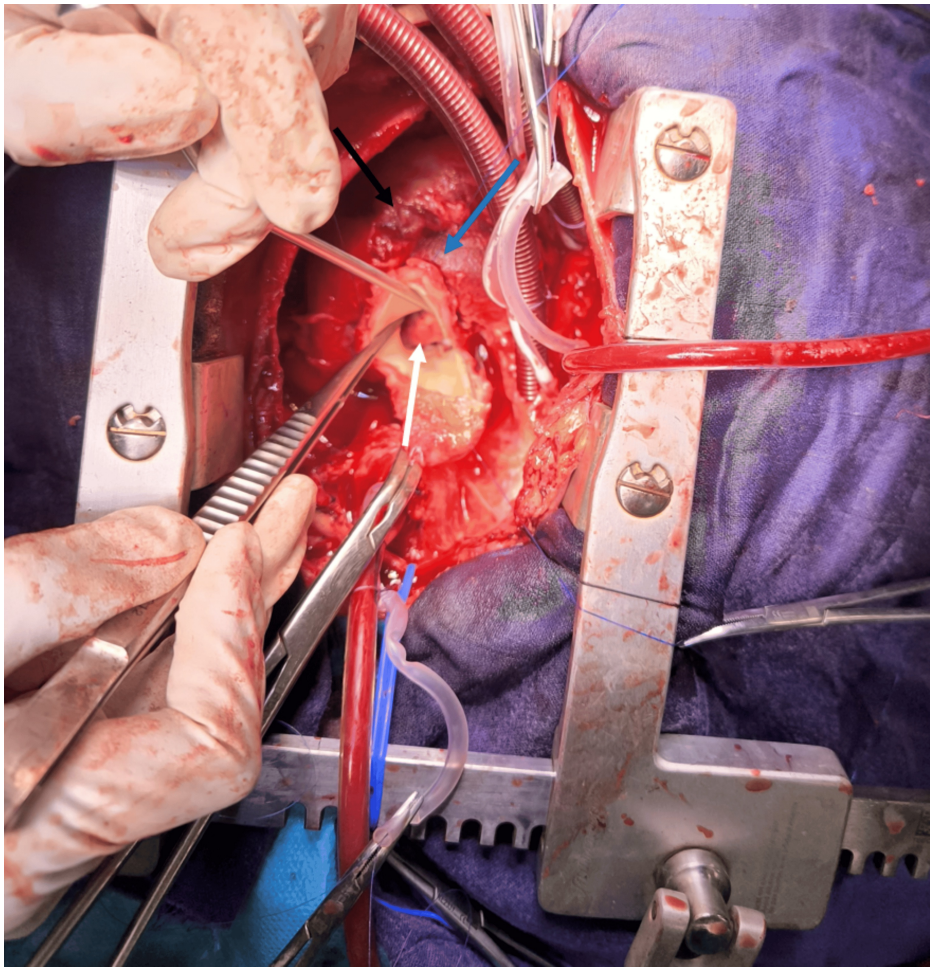


FIGURE 2: Intraoperative findings

Aortic valve vegetation (white arrow), infective right sinus of Valsalva pseudoaneurysm (blue arrow), and bluish discoloration of the right ventricular free wall corresponding to the intramyocardial dissecting hematoma (black arrow).

The aortic opening of the pseudoaneurysm communication was directly visualized, and its continuity with the right ventricular free wall was confirmed using a surgical dissector. No direct rupture into the pericardial cavity was identified. These findings established that the pseudoaneurysm had ruptured in a contained manner into the right ventricular free wall, resulting in an intramyocardial dissecting hematoma with subsequent bleeding into the pericardial cavity and cardiac tamponade. Following radical debridement of all infected and necrotic tissues, a modified Bentall procedure was performed using a 21-mm mechanical valved conduit. Excision of the infected aortic root, including the right sinus of Valsalva containing the pseudoaneurysm, eliminated the source of bleeding. No additional repair of the right ventricular free wall was required, as bleeding had ceased following aortic root replacement. Extensive infectious destruction of both coronary ostia precluded conventional coronary button reimplantation. Both native coronary ostia were ligated using 5-0 polypropylene sutures. Coronary revascularization was therefore achieved using a Y-shaped saphenous vein graft, with its proximal end anastomosed to the Dacron conduit and its two distal branches anastomosed to the left anterior descending and right coronary arteries, respectively (Figure *3*). The circumflex territory was perfused through the native left coronary circulation supplied by the graft to the left anterior descending artery. The aortic cross-clamp time was 137 minutes, and the total cardiopulmonary bypass time was 208 minutes.

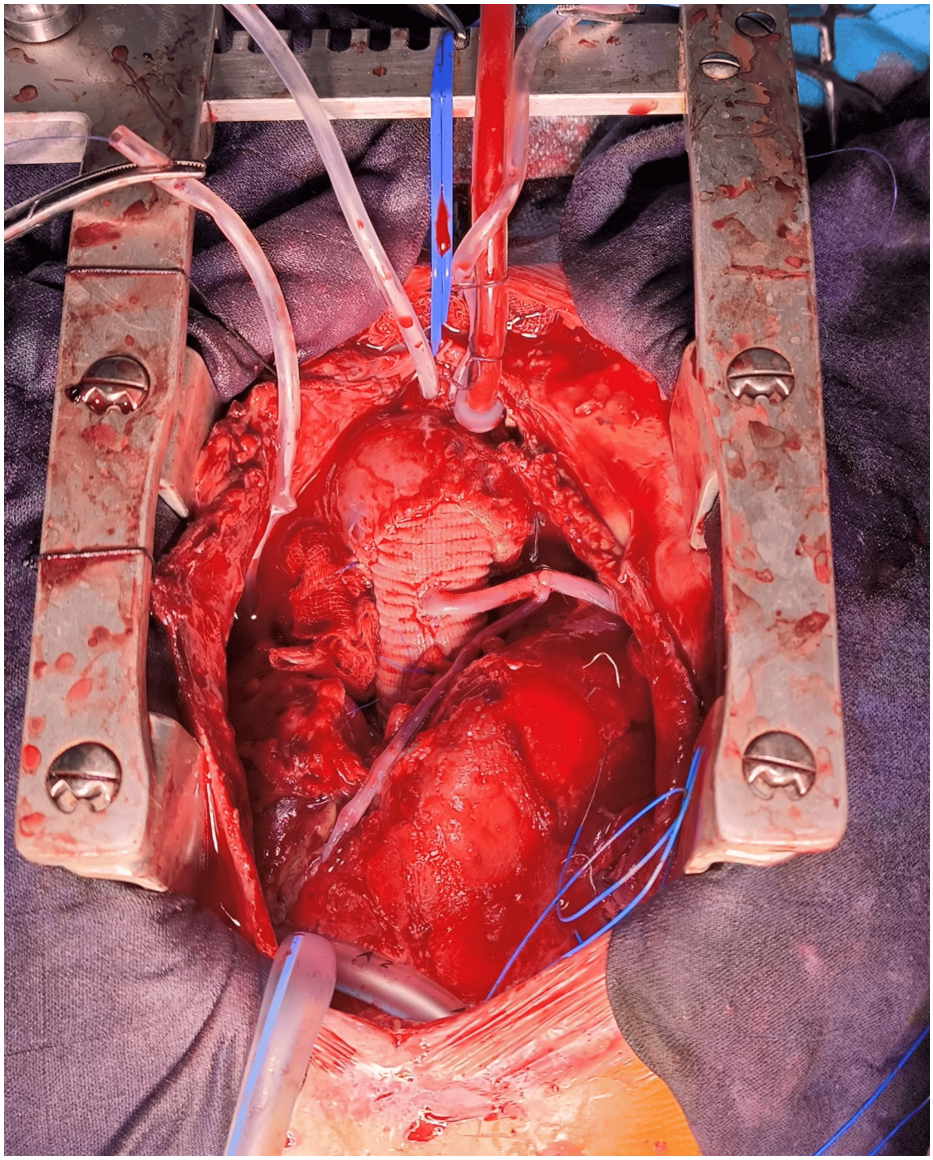


FIGURE 3: Surgical reconstruction

Modified Bentall procedure with saphenous vein Y-graft coronary bypass following radical debridement and aortic root reconstruction.

Separation from cardiopulmonary bypass was subsequently achieved with high-dose dobutamine and norepinephrine support.

The postoperative course was complicated by refractory circulatory shock requiring dobutamine at 7 µg/kg/min and escalating norepinephrine support up to 3 µg/kg/min, with a lactate level of 11 mmol/L. Postoperative transthoracic echocardiography showed moderate right ventricular dysfunction, hyperdynamic left ventricular systolic function, a left ventricular outflow tract velocity-time integral of 18 mm, and a markedly small inferior vena cava despite fluid resuscitation and vasopressor support. No pericardial effusion or significant postoperative bleeding was observed. Despite continued hemodynamic support and intensive care management, the patient remained profoundly unstable and died on postoperative day 2 (Figure *4*).

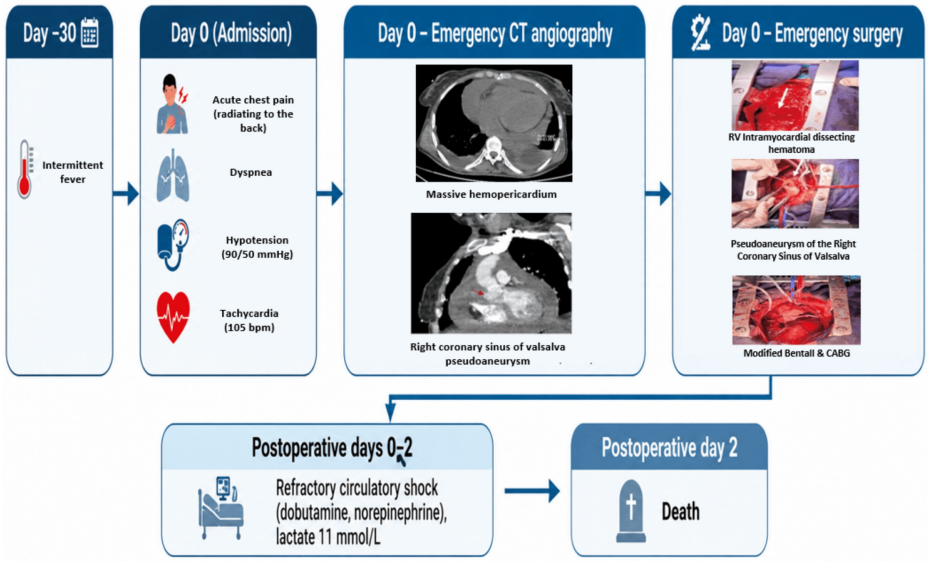


FIGURE 4: Visual summary

Clinical timeline illustrating the progression from intermittent fever to acute presentation, emergency CT angiography, surgical intervention, refractory postoperative circulatory shock, and death on postoperative day 2.

CT: computed tomography; RV: right ventricle; CABG: coronary artery bypass grafting

The image was initially generated with the assistance of ChatGPT (OpenAI, San Francisco, CA, US) and subsequently manually edited and finalized by the author using Microsoft Word (Microsoft Corporation, Redmond, WA, US).

Discussion

Periannular extension is one of the most severe complications of aortic valve infective endocarditis and is associated with extensive tissue destruction, abscess formation, pseudoaneurysm, and fistulous communication with adjacent cardiac structures [1]. Infective pseudoaneurysms of the sinus of Valsalva are uncommon and result from progressive extension of infection beyond the aortic annulus [1]. Owing to the close anatomical relationship between the right coronary sinus and the right-sided cardiac chambers, rupture most commonly occurs into the right atrium or right ventricle, creating an aorto-cardiac fistula with left-to-right shunting [2-7]. Extension into the interventricular septum, pericardium, or other adjacent structures is considerably less frequent [2].

The present case illustrates an exceptionally rare anatomical pathway of rupture of an infective right sinus of Valsalva pseudoaneurysm. Rather than rupturing directly into a cardiac chamber or the pericardial cavity, the pseudoaneurysm underwent a contained rupture into the right ventricular free wall, resulting in an intramyocardial dissecting hematoma. The communication between the pseudoaneurysm and the hematoma was directly identified during surgical exploration.

Intramyocardial dissecting hematoma represents a form of incomplete myocardial rupture in which hemorrhage tracks between myocardial fibers, creating a blood-filled neocavity within the ventricular wall [8]. Although this condition may progress to complete myocardial rupture, particularly in the presence of hemodynamic instability, hematoma enlargement, or pericardial effusion, right ventricular involvement remains exceptionally rare [8,9]. Panduranga et al. reported a right ventricular free-wall intramyocardial hematoma following coronary artery bypass surgery [9]. Zhou et al. described myocardial dissection caused by a ruptured sinus of Valsalva aneurysm associated with a bicuspid aortic valve [5], while Bouziane and Haboub reported intramyocardial dissection associated with a sinus of Valsalva aneurysm following surgical aortic valve replacement [6]. Unlike these reports, our case involved destructive native aortic valve infective endocarditis, with a direct communication between an infective right sinus of Valsalva pseudoaneurysm and a dissecting hematoma of the right ventricular free wall. Furthermore, the hemopericardium resulted from active bleeding originating from the intramyocardial hematoma rather than direct rupture of the pseudoaneurysm into the pericardial cavity. This distinctive anatomical sequence explains the unusual mechanism of cardiac tamponade observed in our patient.

The initial presentation posed a major diagnostic challenge. The sudden onset of chest pain radiating to the back, accompanied by profound hemodynamic instability, strongly suggested an acute aortic syndrome. Consequently, computed tomography angiography was performed and rapidly demonstrated both the

hemopericardium and a contrast-filled cavity adjacent to the right coronary sinus, prompting emergency surgical exploration. Although bedside echocardiography is generally the first-line imaging modality in hemodynamically unstable patients and remains the cornerstone for diagnosing infective endocarditis and evaluating vegetations, valvular dysfunction, and intracardiac hemodynamics, cardiac computed tomography provides complementary anatomical information and is particularly valuable for detecting periannular complications, including abscesses and pseudoaneurysms [10]. Furthermore, this case highlights that infective endocarditis should be included in the differential diagnosis for patients presenting with hemopericardium associated with a lesion of the aortic root, even when the initial clinical picture strongly suggests an acute aortic syndrome.

Surgery for locally destructive aortic valve infective endocarditis relies on radical debridement of all infected and necrotic tissue followed by reconstruction tailored to the extent of anatomical destruction [11]. Depending on the severity of periannular involvement, surgical options range from valve replacement with localized patch reconstruction to complete aortic root replacement [11,12]. In our patient, extensive destruction of the aortic root and both coronary ostia precluded valve preservation and coronary button reimplantation, necessitating a modified Bentall procedure combined with coronary artery bypass grafting using a saphenous vein Y graft to the left anterior descending and right coronary arteries. Despite prompt surgical intervention, the patient developed refractory postoperative vasoplegic shock and died on postoperative day 2.

The fatal outcome was likely multifactorial, reflecting the prolonged infectious course, profound hemodynamic instability at presentation, and extensive destruction of the aortic root and coronary ostia requiring complex emergency reconstruction. Refractory postoperative circulatory shock may have further contributed to the unfavorable outcome. The main limitations of this report include the absence of preoperative and intraoperative echocardiography, the delayed availability of microbiological results, and the lack of an autopsy to establish the precise cause of death.

Conclusions

This case expands the recognized spectrum of mechanical complications associated with aortic valve infective endocarditis by describing an exceptionally rare anatomical mechanism of cardiac tamponade. The fatal outcome despite emergency surgical intervention underscores the severity of this complication and the challenges of its management. Infective endocarditis should be considered in the differential diagnosis of hemopericardium associated with an aortic root lesion, particularly in patients with a history of prolonged unexplained fever, even when the acute presentation suggests an acute aortic syndrome.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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Figure 4 was initially generated with the assistance of ChatGPT (OpenAI, San Francisco, CA, US) and subsequently manually edited and finalized by the author using Microsoft Word (Microsoft Corporation, Redmond, WA, US).

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