

Long-term multimodal imaging, biomarker monitoring and late supraventricular tachycardia-associated collapse in a Maltese dog with giant right atrial diverticulum

WOO DAE PARK

wdvet@hanmail.net

Pet in Zoo Animal Medical Center

Case Report

Keywords: Dog, right atrial diverticulum, cardiac anomaly, thrombus, rivaroxaban

Posted Date: July 9th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-10147128/v1>

License:   This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Additional Declarations: No competing interests reported.

CASE REPORT

Canine

Long-term multimodal imaging, biomarker monitoring and late supraventricular tachycardia-associated collapse in a Maltese dog with giant right atrial diverticulum

Woo Dae Park

Pet in Zoo Animal Medical Center, 1336 Hoguk-ro, Uijeongbu-si, Gyeonggi-do 11693, Republic of Korea

Correspondence: Woo Dae Park, Pet in Zoo Animal Medical Center, 1336 Hoguk-ro, Uijeongbu-si, Gyeonggi-do 11693, Republic of Korea, Email: wdvvet@hanmail.net

Keywords: Dog | right atrial diverticulum | cardiac anomaly | thrombus| rivaroxaban.

25 **Abstract**

26 **Background:** Right atrial diverticulum is an uncommon cardiac anomaly in dogs, and its
27 long-term clinical behaviour remains poorly characterized. This case was reported
28 because it documents prolonged clinical control, intracavitary blood stasis, suspected
29 thrombus formation, serial biomarker changes and late supraventricular tachycardia-
30 associated collapse in a dog with giant right atrial diverticulum.

31 **Case presentation:** A 10-year-old neutered male Maltese dog was evaluated for
32 recurrent collapse associated with chronic cough and excitement. Thoracic radiography
33 suggested a cranial cardiac or cranial mediastinal abnormality. Contrast-enhanced
34 computed tomography revealed a large blood-pool enhancing cystic structure measuring
35 approximately 41 × 38 × 34 mm adjacent to the cranial vena cava–right atrial region,
36 with dependent contrast pooling consistent with slow or stagnant intracavitary blood
37 flow. Echocardiography demonstrated communication between the cystic cavity and the
38 right atrial region through an approximately 7.5-mm orifice, and multiple mobile
39 echogenic structures within the cavity were interpreted as suspected intracavitary
40 thrombi. The lesion was diagnosed as a giant right atrial diverticulum. Initial

management was directed toward cough control and thromboembolic risk reduction. Following marked D-dimer elevation, aspirin was replaced with rivaroxaban at 0.5 mg/kg orally every 12 hours. Serial biomarker monitoring showed intermittent D-dimer elevation, persistent N-terminal pro-B-type natriuretic peptide increase and repeated cardiac troponin I elevation, supporting thromboembolic risk, progressive cardiac strain and recurrent myocardial injury, respectively. The dog was followed for approximately 45 months after initial presentation, during which cough and collapse were clinically controlled for a prolonged period. Late in the disease course, recurrent cough and repeated syncope developed, and electrocardiography documented a regular narrow-complex supraventricular tachycardia at 178 beats/min. Terminal cardiopulmonary decompensation subsequently occurred with azotemia and hypercapnic respiratory acidosis.

Conclusions: This case highlights that giant right atrial diverticulum in a dog may remain clinically controlled for several years but may later become clinically important through intracavitary blood stasis, suspected thrombus formation, progressive cardiac strain, recurrent myocardial injury and supraventricular tachycardia-associated collapse. Multimodal imaging, thromboembolic risk monitoring, serial cardiac biomarker

assessment and rhythm evaluation may be clinically useful in dogs with right atrial diverticulum.

Background

Right atrial diverticulum is an uncommon cardiac anomaly characterized by a chamber-like outpouching that communicates with the right atrium. In dogs, this condition has been only rarely described, and its clinical relevance, long-term behaviour and optimal management remain poorly established. Park et al. [1] reported a canine case of giant right atrial diverticulum in which echocardiography demonstrated an anechoic cavity lateral to the right atrium communicating with the right atrial free wall through a 13-mm defect. Although that report established right atrial diverticulum as an important differential diagnosis for a third chamber-like structure adjacent to the right atrium, information regarding long-term clinical course, thromboembolic risk monitoring, cardiac biomarker trends, arrhythmia development and terminal outcome remains limited.

Right-sided cystic or chamber-like thoracic lesions are diagnostically challenging because congenital, vascular and mass-like conditions may have overlapping radiographic,

echocardiographic and computed tomographic appearances. Important differential diagnoses include cranial mediastinal mass, pericardial cyst, right atrial aneurysm, right auricular or atrial appendage aneurysm, cranial vena caval aneurysm or venous diverticulum and cor triatriatum dexter. Canine right auricular or atrial appendage aneurysm has been described as a rare condition in which computed tomography and echocardiography are useful for defining lesion origin and morphology [2, 10, 11]. Cor triatriatum dexter is also relevant because it results from persistence of the embryologic right sinus venosus valve, creating an intra-atrial membrane that divides the right atrium into two chambers and may be associated with right-sided congestive signs or other congenital cardiac abnormalities [3, 4]. Therefore, multimodal imaging is essential for distinguishing an external diverticular cavity from aneurysmal dilation, an intra-atrial membrane or a non-cardiac mediastinal lesion.

The clinical significance of a giant right atrial diverticulum may extend beyond its anatomic appearance. A large blood-filled cavity with slow intracavitary flow may predispose to blood stasis, spontaneous echo contrast, intracavitary thrombus formation or thromboembolic complications. In the previously reported canine case, echogenic material within the diverticulum was suspected to represent thrombi, and pulmonary

thromboembolism was considered a possible complication [1]. In dogs, D-dimer concentration may support suspicion of thromboembolic disease but is nonspecific and cannot independently confirm pulmonary thromboembolism [5]. Cardiac biomarkers may also provide supportive information during long-term follow-up. N-terminal pro-B-type natriuretic peptide elevation may reflect cardiac strain, whereas cardiac troponin I elevation indicates myocardial injury; however, these biomarkers are not specific for right atrial diverticulum and must be interpreted in relation to imaging findings, rhythm status and systemic clinical condition. In addition, structural right atrial abnormalities may provide a substrate for atrial electrical instability, although arrhythmia development and long-term rhythm outcome in dogs with right atrial diverticulum are poorly characterized.

This report describes a Maltese dog with giant right atrial diverticulum diagnosed by echocardiography and contrast-enhanced computed tomography. The lesion communicated with the right atrial region through an approximately 7.5-mm echocardiographic orifice and showed blood-pool enhancement with dependent contrast pooling on computed tomography, consistent with slow or stagnant intracavitary blood flow. Multiple mobile intracavitary echogenic structures were interpreted as suspected

108 thrombi. The dog was managed medically for approximately 45 months after initial
109 presentation, during which prolonged clinical control was achieved before late recurrence
110 of cough, repeated syncope, electrocardiographically documented regular narrow-
111 complex supraventricular tachycardia and terminal cardiopulmonary decompensation.
112 Serial biomarker monitoring showed intermittent D-dimer elevation, persistent N-
113 terminal pro-B-type natriuretic peptide increase and repeated cardiac troponin I
114 elevation, supporting thromboembolic risk, progressive cardiac strain and recurrent
115 myocardial injury, respectively. This case is educational because it illustrates the
116 diagnostic value of multimodal imaging for right-sided chamber-like thoracic lesions and
117 highlights potential long-term complications of giant right atrial diverticulum, including
118 intracavitary blood stasis, suspected thrombus formation, progressive cardiac strain and
119 late supraventricular tachycardia-associated collapse.

120 **Clinical findings**

121 A 10-year-old neutered male Maltese dog weighing 3.4 kg was presented approximately
122 7 months after the first recognized collapse episode for evaluation of recurrent collapse
123 associated with chronic cough and excitement. The owner reported a chronic cough that

124 was most pronounced during excitement. At the initial onset of clinical signs, coughing
125 and gagging during excitement were followed by sudden weakness, falling to one side,
126 limb movements, body stiffness and spontaneous recovery. Approximately 7 months
127 later, immediately before referral evaluation, a more severe episode occurred in which
128 the dog collapsed, developed open-mouth breathing with tongue protrusion and
129 became transiently unresponsive. After the episode, the dog was weak and unable to
130 stand normally. Based on the temporal association with cough and excitement, the
131 episodes were considered most consistent with syncope or collapse associated with
132 cardiopulmonary compromise.

133 Thoracic radiographs obtained at initial presentation showed a broad soft tissue opacity
134 associated with the cranial cardiac silhouette or cranial mediastinal region (Fig. 1). No
135 obvious diffuse alveolar pulmonary oedema or pleural effusion was identified. Because
136 thoracic radiography could not differentiate a mediastinal mass, pericardial cystic lesion
137 or vascular cardiac anomaly, further evaluation with echocardiography and contrast-
138 enhanced computed tomography was performed.

139 **Diagnostic imaging**

140 Contrast-enhanced computed tomography was performed using a Woorien MyVet CT
141 i3D system shortly after initial presentation, approximately 7 months after the first
142 recognized collapse episode. At that time, the dog weighed 3.2 kg. Computed
143 tomography revealed a large cystic structure at the cranial aspect of the heart, adjacent
144 to the cranial vena cava–right atrial region (Fig. 2). The lesion measured approximately 41
145 × 38 × 34 mm and enhanced similarly to adjacent vascular structures, indicating a
146 blood-filled vascular or cardiac origin rather than a solid mediastinal mass. Dependent
147 contrast pooling was present within the cavity, consistent with slow or stagnant
148 intracavitary blood flow (Fig. 2). No pulmonary parenchymal lesion, mediastinal
149 lymphadenopathy, thoracic wall lesion or rib involvement was identified. Based on these
150 findings, a nonvascular mediastinal mass, invasive thoracic neoplasm or pericardial cyst
151 was considered unlikely.

152 Echocardiography was performed using a Philips EPIQ Elite ultrasound system to define
153 the relationship between the cystic structure and the right atrium. A large anechoic
154 chamber-like cavity was identified adjacent to the right atrium, and multiple mobile
155 echogenic structures were observed within the diverticular cavity (Fig. 3; Additional file 1).
156 A communicating orifice between the cavity and the right atrial region was not clearly

visible on the still image but was identified on cine-loop assessment and was estimated to measure approximately 7.5 mm (Additional file 1). The mobile echogenic structures were considered consistent with suspected intracavitary thrombi. The combination of computed tomographic blood-pool enhancement, dependent contrast pooling, echocardiographic communication with the right atrial region and mobile intracavitary thrombus-like structures supported a diagnosis of giant right atrial diverticulum. No definite intra-atrial membrane dividing the right atrium was identified, making cor triatriatum dexter less likely. Right auricular aneurysm, cranial vena caval aneurysm and atypical cranial vena caval–right atrial junction anomaly remained differential considerations, but were considered less likely based on the multimodal imaging findings.

Medical management and biomarker monitoring

Medical management was elected. Initial treatment was individualized and focused on control of cough-associated collapse and reduction of thromboembolic risk associated with the low-flow diverticular cavity and suspected mobile intracavitary thrombi. Cough-directed therapy and antithrombotic management were adjusted according to the dog's

173 clinical status. After treatment initiation, cough and collapse episodes resolved clinically,
174 and the dog remained stable during long-term outpatient follow-up.

175 Serial monitoring included complete blood count, serum biochemistry, D-dimer
176 concentration and cardiac biomarkers. Complete blood count and serum biochemistry
177 were assessed using a Fujifilm FDC NX700 analyzer, and D-dimer, N-terminal pro-B-type
178 natriuretic peptide and cardiac troponin I were measured using a Bionote Vcheck V200
179 system. Approximately 19 months after the first recognized collapse episode, D-dimer
180 concentration increased markedly to 3748.35 mg/dL (reference interval, 0–250 mg/dL). In
181 the context of a giant low-flow right atrial diverticulum containing mobile thrombus-like
182 structures, this result was interpreted as evidence of increased fibrin turnover and
183 thromboembolic risk, rather than confirmation of pulmonary thromboembolism. Aspirin
184 was discontinued, and rivaroxaban was initiated at 0.5 mg/kg orally every 12 hours.

185 Clopidogrel was not included in the final treatment regimen. During anticoagulant
186 therapy, the dog was monitored for clinical evidence of bleeding, haematocrit changes
187 and renal or hepatic abnormalities.

188 Approximately 24 months after the first recognized collapse episode, follow-up

echocardiography showed no left atrial enlargement or left ventricular dilation. The left atrium-to-aortic root ratio was 1.02, and normalized left ventricular internal diameter in diastole was 1.2. Fractional shortening was 54.7%, and Teichholz-derived ejection fraction was 88.0%. These findings did not support clinically relevant left-sided volume overload or systolic dysfunction at that time. Between approximately 24 and 52 months after the first recognized collapse episode, serial N-terminal pro-B-type natriuretic peptide measurements were persistently increased and were interpreted as evidence of progressive cardiac strain rather than as lesion-specific biomarkers. Values recorded as cProBNP or c.NT-proBNP in the medical records were presented as N-terminal pro-B-type natriuretic peptide because they were generated using the same in-house biomarker platform and reference interval. Supportive cardiovascular therapy, including pimobendan and other adjunctive medications, was subsequently adjusted according to clinical status and biomarker trends.

Late clinical deterioration and outcome

Approximately 51 months after the first recognized collapse episode, cough recurred and became more severe. The cough was described as harsh and frequent, with suspected

205 mucus or phlegm in the throat. Resting respiration during sleep was reportedly
206 acceptable, but appetite had decreased. Approximately 52 months after the first
207 recognized collapse episode, the dog experienced another syncopal episode.
208 Electrocardiography obtained during recurrent collapse documented a regular narrow-
209 complex supraventricular tachycardia at 178 beats/min (Fig. 4). The QRS duration was 66
210 ms, and the PR interval was 132 ms. The automated electrocardiographic interpretation
211 also suggested high-voltage QRS complexes and right atrial enlargement. In the clinical
212 context of recurrent syncope, this rhythm was considered compatible with
213 tachyarrhythmia-associated low-output collapse. Laboratory evaluation at that time
214 showed increased D-dimer concentration, azotemia, hyperphosphatemia, increased N-
215 terminal pro-B-type natriuretic peptide, increased cardiac troponin I, neutrophilic
216 leukocytosis and hypercapnic respiratory acidosis. The blood gas findings supported
217 ventilatory compromise during acute deterioration.

218 Within the same terminal month, the dog was evaluated for respiratory difficulty. The
219 owner reported transient improvement with oxygen supplementation. Respiratory effort
220 and rate worsened during excitement or barking. Systolic blood pressure was
221 approximately 160 mmHg when the dog was excited. Oxygen cage therapy was provided

222 during episodes of respiratory distress, although the fraction of inspired oxygen was not
223 consistently recorded.

224 Later in the terminal month, laboratory evaluation continued to show increased N-
225 terminal pro-B-type natriuretic peptide and cardiac troponin I, together with persistent
226 azotemia. D-dimer concentration had decreased into the reference range. This finding
227 did not exclude thromboembolic disease but provided no laboratory support for active
228 fibrin turnover at that time.

229 Approximately 52 months after the first recognized collapse episode, and approximately
230 45 months after initial presentation, the dog developed clustered collapse episodes. The
231 owner reported two syncopal episodes during the previous day. On the day of terminal
232 presentation, vomiting occurred for the first time at approximately 04:00, after which the
233 dog was moved to an oxygen cage at home. One additional syncopal episode occurred
234 during the day, followed by another episode in the evening. In contrast to previous
235 episodes, in which the dog typically recovered within approximately 1 minute after
236 oxygen cage placement, recovery was delayed during the evening episode. The dog
237 defecated in the oxygen cage, failed to recover normal posture and was unable to stand

238 appropriately. Appetite for palatable food remained acceptable, but water intake and
239 urine output were decreased.

240 During transport to the hospital, the respiratory pattern improved, and the dog began to
241 maintain sternal posture. The owner had not brought the dog's oral medications and
242 requested hospitalization. At emergency presentation, respiratory effort was acceptable.
243 The dog initially rested in sternal recumbency and began to stand after several minutes.
244 Mentation was bright, alert and responsive.

245 Thoracic radiographs obtained at emergency presentation showed persistent broadening
246 of the cranial cardiac or cranial mediastinal silhouette, corresponding to the previously
247 identified right atrial diverticular lesion (Fig. 5). No obvious severe diffuse pulmonary
248 oedema or massive pleural effusion was identified on the available views. These
249 radiographs did not reveal a definitive cause of terminal collapse.

250 Acute deterioration occurred shortly after admission, and the dog died approximately 1
251 hour after emergency presentation. Cardiopulmonary resuscitation was not performed in
252 accordance with the owner's decision. Necropsy was not performed; therefore, terminal
253 rhythm, pulmonary thromboembolism, myocardial pathology and the definitive cause of

254 death could not be confirmed. The terminal event was interpreted as acute
255 cardiopulmonary decompensation, with possible contributions from supraventricular
256 tachycardia-associated low cardiac output, thromboembolic complications, ventilatory
257 compromise and impaired systemic perfusion.

258

259 **Discussion and conclusions**

260 This case describes the long-term clinical course of a Maltese dog with a giant right
261 atrial diverticulum diagnosed using echocardiography and contrast-enhanced computed
262 tomography. The principal findings were a large blood-pool enhancing cystic cavity
263 adjacent to the cranial vena cava–right atrial region, dependent contrast pooling within
264 the cavity, an approximately 7.5-mm communication with the right atrial region identified
265 on echocardiographic cine-loop assessment, and multiple mobile intracavitary echogenic
266 structures interpreted as suspected thrombi. The dog was medically managed for
267 approximately 45 months after initial presentation, with prolonged clinical control of
268 cough and collapse before late recurrence of respiratory signs, repeated syncope,
269 electrocardiographically documented regular narrow-complex supraventricular

270 tachycardia and terminal cardiopulmonary decompensation.

271 Right atrial diverticulum has been rarely reported in dogs. Park et al. [1] described a
272 canine case in which an anechoic chamber-like cavity lateral to the right atrium
273 communicated with the right atrial free wall through a 13-mm defect. In the present
274 case, the diverticular cavity measured approximately 41 × 38 × 34 mm on computed
275 tomography and communicated with the right atrial region through an approximately
276 7.5-mm orifice. Although the orifice was not extremely small, it was relatively narrow in
277 relation to the size of the cavity. This anatomic configuration may have contributed to
278 slow intracavitary flow, contrast retention and suspected thrombus formation.

279 Multimodal imaging was essential for differentiating the lesion from other right-sided
280 thoracic abnormalities. Initial thoracic radiographs demonstrated a broad cranial cardiac
281 or cranial mediastinal soft tissue opacity (Fig. 1), but radiography alone could not
282 determine whether the lesion represented a mediastinal mass, pericardial cystic lesion or
283 vascular cardiac anomaly. Contrast-enhanced computed tomography showed blood-pool
284 enhancement and dependent contrast pooling within the cavity (Fig. 2), supporting a
285 vascular or cardiac origin and slow or stagnant intracavitary blood flow.

286 Echocardiography further demonstrated a large anechoic chamber-like cavity adjacent to
287 the right atrium and multiple mobile intracavitary echogenic structures interpreted as
288 suspected thrombi (Fig. 3; Additional file 1). A communicating orifice between the
289 diverticular cavity and the right atrial region was identified on cine-loop assessment and
290 was estimated to measure approximately 7.5 mm (Additional file 1). In this dog, the
291 combination of computed tomographic blood-pool enhancement, dependent contrast
292 pooling, echocardiographic communication with the right atrial region and mobile
293 intracavitary thrombus-like structures supported giant right atrial diverticulum as the
294 most likely diagnosis. However, because surgical inspection, histopathology and necropsy
295 were not performed, an atypical cranial vena caval–right atrial junction anomaly could
296 not be completely excluded.

297 The mobile intracavitary echogenic structures were a clinically important feature of this
298 case. These structures were interpreted as suspected mobile thrombi, although histologic
299 confirmation was unavailable. Together with dependent contrast pooling on computed
300 tomography, this finding supported clinically relevant blood stasis within the
301 diverticulum. Echogenic material suspected to represent thrombi was also described in
302 the previously reported canine right atrial diverticulum case [1]. These observations

303 suggest that thrombus formation should be considered a potential complication of giant
304 right atrial diverticulum, particularly when a large blood-filled cavity is associated with
305 slow intracavitary circulation.

306 D-dimer monitoring contributed to clinical decision-making but was not used as a stand-
307 alone diagnostic test. Serial D-dimer concentrations showed intermittent rather than
308 persistent elevation (Fig. 6A). Approximately 19 months after the first recognized collapse
309 episode, D-dimer concentration increased markedly to 3748.35 mg/dL (reference interval,
310 0–250 mg/dL) and was interpreted as evidence of increased fibrin turnover and
311 thromboembolic risk in the context of a low-flow right atrial diverticulum containing
312 mobile thrombus-like structures. D-dimer concentration is nonspecific and cannot
313 independently confirm pulmonary thromboembolism in dogs [5, 9]. Nevertheless, in this
314 clinical context, marked D-dimer elevation provided additional support for intensifying
315 antithrombotic management.

316 Rivaroxaban was administered at 0.5 mg/kg orally every 12 hours after aspirin was
317 discontinued. This was an empirical antithrombotic strategy rather than a disease-specific
318 protocol for right atrial diverticulum. Direct oral factor Xa inhibitors, including

319 rivaroxaban, have been increasingly investigated in dogs, and repeated oral
320 administration can produce measurable anticoagulant effects, although interindividual
321 pharmacodynamic variability remains a concern [6–8]. In the present case, rivaroxaban
322 was selected because of the combination of a giant low-flow vascular cavity, computed
323 tomographic contrast stasis, suspected mobile intracavitary thrombi and marked D-dimer
324 elevation. This case, however, does not establish the efficacy of rivaroxaban for
325 preventing thromboembolic complications in dogs with right atrial diverticulum.

326 The prolonged period of clinical control suggests that individualized medical
327 management may provide meaningful stabilization in selected dogs with giant right atrial
328 diverticulum. Initial management was directed primarily toward cough control and
329 thromboembolic risk reduction. Broader supportive cardiovascular therapy, including
330 pimobendan and other adjunctive medications, was introduced or adjusted after serial N-
331 terminal pro-B-type natriuretic peptide elevation suggested progressive cardiac strain
332 during follow-up. This distinction is important because follow-up echocardiography
333 approximately 24 months after the first recognized collapse episode showed no left atrial
334 enlargement, left ventricular dilation or systolic dysfunction. Therefore, these medications
335 should be interpreted as supportive management for cardiac strain and cardiopulmonary

336 status rather than disease-specific treatment for right atrial diverticulum.

337 The contribution of individual medications could not be determined retrospectively
338 because treatment was modified empirically over a long follow-up period. Rivaroxaban
339 use was most directly supported by imaging and laboratory findings, including
340 dependent computed tomographic contrast pooling, mobile intracavitary thrombus-like
341 structures and marked D-dimer elevation. Later rate-control therapy was supported by
342 electrocardiographic documentation of supraventricular tachycardia during recurrent
343 collapse. In contrast, the specific benefit of other supportive cardiovascular or respiratory
344 medications was uncertain. Some agents with positive inotropic, vasodilatory,
345 bronchodilatory or preload-modifying effects may have influenced systemic blood
346 pressure, preload or rhythm stability, particularly during terminal deterioration.

347 Serial cardiac biomarker monitoring provided supportive evidence of progressive
348 cardiopulmonary stress but did not identify a lesion-specific mechanism. N-terminal pro-
349 B-type natriuretic peptide concentrations were persistently increased during follow-up
350 and rose markedly during terminal deterioration (Fig. 6B), supporting progressive cardiac
351 strain. Cardiac troponin I concentrations were repeatedly increased (Fig. 6C), supporting

recurrent myocardial injury. These biomarker abnormalities may have reflected chronic right-sided cardiac loading, atrial stretch, tachyarrhythmia, altered cardiopulmonary haemodynamics, hypoventilation, renal perfusion changes or terminal systemic decompensation. Follow-up echocardiography showed no left atrial enlargement or left ventricular dilation, suggesting that clinically relevant left-sided volume overload was not the dominant abnormality at that time.

Late recurrent syncope with electrocardiographically documented supraventricular tachycardia was a key feature of this case. Approximately 52 months after the first recognized collapse episode, an electrocardiogram obtained during recurrent collapse showed a regular narrow-complex supraventricular tachycardia at 178 beats/min (Fig. 4). This rhythm may have contributed to syncope by shortening diastolic filling time, reducing stroke volume and limiting compensatory cardiac output. This mechanism may have been particularly relevant in a small dog with a giant right atrial structural anomaly, suspected intracavitary thrombi, increased N-terminal pro-B-type natriuretic peptide, increased cardiac troponin I and concurrent cardiopulmonary compromise. However, because continuous electrocardiographic monitoring and terminal rhythm documentation were not available, the precise arrhythmia mechanism and its role in the final death

369 event could not be definitively determined.

370 The terminal event was best interpreted as acute cardiopulmonary decompensation
371 rather than death attributable to a single confirmed mechanism. During the terminal
372 period, the dog had recurrent cough, clustered syncope, delayed recovery despite
373 oxygen cage placement, decreased water intake and urine output, azotemia, increased
374 cardiac biomarkers and hypercapnic respiratory acidosis. These findings suggest a
375 combination of ventilatory compromise, cardiac strain, impaired systemic perfusion and
376 systemic deterioration. Pulmonary thromboembolism was clinically relevant because of
377 the low-flow diverticular cavity and suspected mobile intracavitary thrombi, but it was
378 not confirmed. The normalization of D-dimer shortly before death did not exclude
379 thromboembolic disease, but it provided no laboratory support for active fibrin turnover
380 at that specific time point.

381 Terminal thoracic radiographs demonstrated persistence of the cranial cardiac or cranial
382 mediastinal soft tissue opacity corresponding to the known vascular cavity (Fig. 5), but
383 did not show obvious severe diffuse pulmonary oedema or massive pleural effusion.
384 Therefore, terminal death could not be attributed to radiographically evident left-sided

385 congestive heart failure. However, thoracic radiographs could not exclude pulmonary
386 thromboembolism, terminal tachyarrhythmia, low-output decompensation or acute
387 respiratory failure.

388 Several treatment-related factors should be considered. Cough control was clinically
389 relevant because collapse episodes were temporally associated with coughing and
390 excitement. However, methylxanthine-associated tachyarrhythmia could not be excluded
391 if bronchodilator therapy was used during follow-up. In addition, vasodilatory or preload-
392 modifying medications may have influenced systemic blood pressure or cardiac filling
393 during late deterioration. Although systolic blood pressure was approximately 160 mmHg
394 when the dog was excited, consistent resting blood pressure data were unavailable.
395 Because azotemia and decreased water intake and urine output developed during the
396 terminal period, medication-related haemodynamic or renal perfusion effects could not
397 be completely excluded.

398 This case has several limitations. The diagnosis of right atrial diverticulum was based on
399 echocardiography and computed tomography without surgical or histopathologic
400 confirmation. The mobile intracavitary echogenic structures were interpreted as

401 suspected thrombi, but histologic confirmation was not available. The exact anatomic
402 relationship among the diverticular cavity, right atrial wall and cranial vena cava could
403 not be verified. Although supraventricular tachycardia was documented by
404 electrocardiography during late clinical deterioration, continuous electrocardiographic
405 monitoring and terminal rhythm documentation were unavailable. Necropsy was not
406 performed; therefore, pulmonary thromboembolism, terminal arrhythmia, myocardial
407 pathology and the definitive cause of death could not be confirmed. In addition, multiple
408 medications were used during long-term management, preventing assessment of the
409 independent effect of each treatment. Finally, D-dimer, N-terminal pro-B-type natriuretic
410 peptide and cardiac troponin I were supportive monitoring markers but were not specific
411 for right atrial diverticulum.

412 Despite these limitations, this case provides clinically relevant information regarding a
413 rare right atrial anomaly in a dog. It highlights the diagnostic value of combining
414 echocardiography and contrast-enhanced computed tomography when a right-sided
415 chamber-like or cranial mediastinal lesion is identified. It also suggests that giant right
416 atrial diverticulum may remain clinically compensated for several years, but may later
417 become clinically important through intracavitary blood stasis, suspected thrombus

418 formation, progressive cardiac strain, recurrent myocardial injury and supraventricular
419 tachycardia-associated collapse. Long-term follow-up of dogs with giant right atrial
420 diverticulum should include assessment of clinical signs, rhythm status, cardiopulmonary
421 function, renal parameters, cardiac biomarkers and thromboembolic risk markers.
422 Additional cases are needed to better define prognosis, optimal antithrombotic strategy
423 and risk factors for sudden deterioration.

424 In conclusion, this case illustrates that giant right atrial diverticulum in a dog may remain
425 clinically compensated for several years but may later become clinically important
426 through intracavitary blood stasis, suspected thrombus formation, progressive cardiac
427 strain, recurrent myocardial injury and supraventricular tachycardia-associated collapse.
428 Echocardiography and contrast-enhanced computed tomography were essential for
429 diagnosis, and serial D-dimer, N-terminal pro-B-type natriuretic peptide and cardiac
430 troponin I monitoring provided supportive information during long-term follow-up. This
431 case highlights the value of multimodal imaging, thromboembolic risk monitoring, serial
432 cardiac biomarker assessment and rhythm evaluation in dogs with giant right atrial
433 diverticulum. The definitive cause of death could not be confirmed because terminal
434 electrocardiography and necropsy were not performed.

435

436

437 **Declarations**

438 **Ethics approval and consent to participate**

439 Ethical approval was not required because this report describes the retrospective use of
440 clinical data obtained during routine diagnosis and treatment of a client-owned dog. All
441 diagnostic and therapeutic procedures were performed as part of standard clinical care.
442 Informed owner consent was obtained for diagnostic imaging and medical management.

443 **Consent for publication**

444 Written informed consent was obtained from the owner for publication of this case
445 report and accompanying diagnostic images.

446 **Availability of data and materials**

447 All data supporting the findings of this case report are included in this article and its
448 figures. Additional de-identified imaging files are available from the corresponding
449 author upon reasonable request.

450 **Competing interests**

451 The author declares that he has no competing interests.

452 **Funding**

453 No external funding was received.

454 **Authors' contributions**

455 WDP was responsible for case management, data collection, interpretation of clinical and
456 imaging findings, manuscript preparation and final approval of the manuscript.

457 **Acknowledgements**

458 Not applicable.

459

460

461

462

463 **References**

1. Park S, Kittleson MD, Yu D, Choi J. Echocardiographic features of giant right atrial diverticulum in a dog. *J Vet Intern Med.* 2017;31(3):879–883.
2. Masson AV, Maddox TW, Bode EF, Mortier JR. Clinical and diagnostic imaging findings in dogs with atrial appendage aneurysm: 7 cases (2014–2020). *J Vet Cardiol.* 2021;35:63–73.
3. Nadolny KE, Kelliham HB, Scansen BA, Tjostheim SS, Grint KA, Forrest LJ, Stepien RL. Cor triatriatum dexter in 17 dogs. *J Vet Cardiol.* 2019;23:129–141.
4. Pugliese M, Sicuso DA, Merola G, Bilgiç B, Previti A, Or ME, Passantino A. Cor triatriatum dexter in dogs: a silent bystander or a potential time bomb? A focused literature review from a professional perspective. *Vet Sci.* 2025;12(2):175.
5. Epstein SE, Hopper K, Mellema MS, Johnson LR. Diagnostic utility of D-dimer concentrations in dogs with pulmonary embolism. *J Vet Intern Med.* 2013;27(6):1646–1649.
6. Lynch AM, Ruterbories LK, Zhu Y, Fialkiewicz F, Papich MG, Brooks MB, Goggs R. Comparison of the pharmacokinetics and pharmacodynamics of apixaban and rivaroxaban in dogs. *J Vet Intern Med.* 2024;38(6):3242–3254.

7. Phillips EM, Blois SL, Abrams-Ogg ACG, Wood RD, Monteith G, Cuq B. Monitoring of rivaroxaban therapy in hypercoagulable dogs. *J Vet Intern Med.* 2025;39(2).
8. Bae J, Kim H, Kim W, Kim S, Park J, Jung D-I, Yu D. Therapeutic monitoring of rivaroxaban in dogs using thromboelastography and prothrombin time. *J Vet Intern Med.* 2019;33(3):1322–1330.
9. Goggs R, Benigni L, Fuentes VL, Chan DL. Pulmonary thromboembolism. *J Vet Emerg Crit Care (San Antonio).* 2009;19(1):30–52.
10. Costa A, Cordella A, Caldin M, Bertolini G. Right auricle aneurysm in dogs can be detected with multidetector-row CT and presents with varying morphology. *Vet Radiol Ultrasound.* 2023;64(1):28–35.
11. Schwarz T, Willis R, Summerfield NJ, Doust R. Aneurysmal dilatation of the right auricle in two dogs. *J Am Vet Med Assoc.* 2005;226(9):1512–1515.
12. Toaldo MB, Diana A, Morini M, Cipone M. Imaging diagnosis—intrapericardial right auricle aneurysm in a dog. *Vet Radiol Ultrasound.* 2010;51(5):512–515.

495

496

497

498

499

500

501

502

503

504

505

506

507

508

509

510

511

512

513

514

515

Figure legends

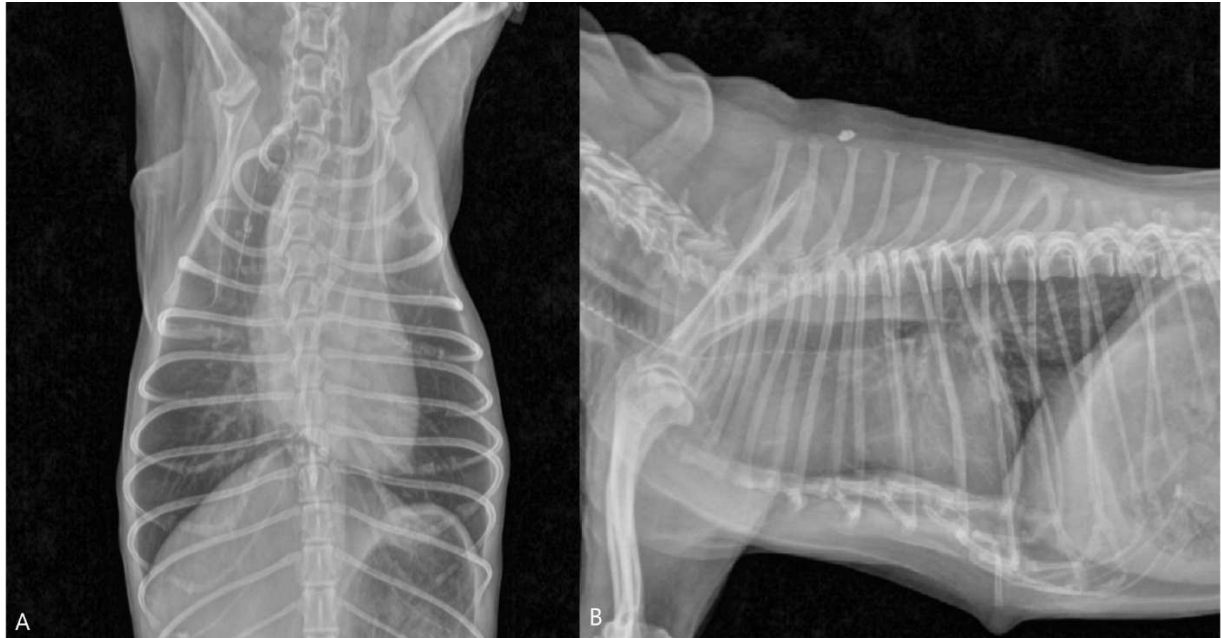


Figure 1. Initial thoracic radiographs obtained at presentation.

Ventrodorsal (A) and right lateral (B) thoracic radiographs obtained at initial presentation, approximately 7 months after the first recognized cough- and excitement-associated collapse episode. A broad soft tissue opacity is present in the cranial cardiac or cranial mediastinal region. No obvious diffuse alveolar pulmonary oedema or pleural effusion is evident on the available views. These findings prompted further evaluation with echocardiography and contrast-enhanced computed tomography.

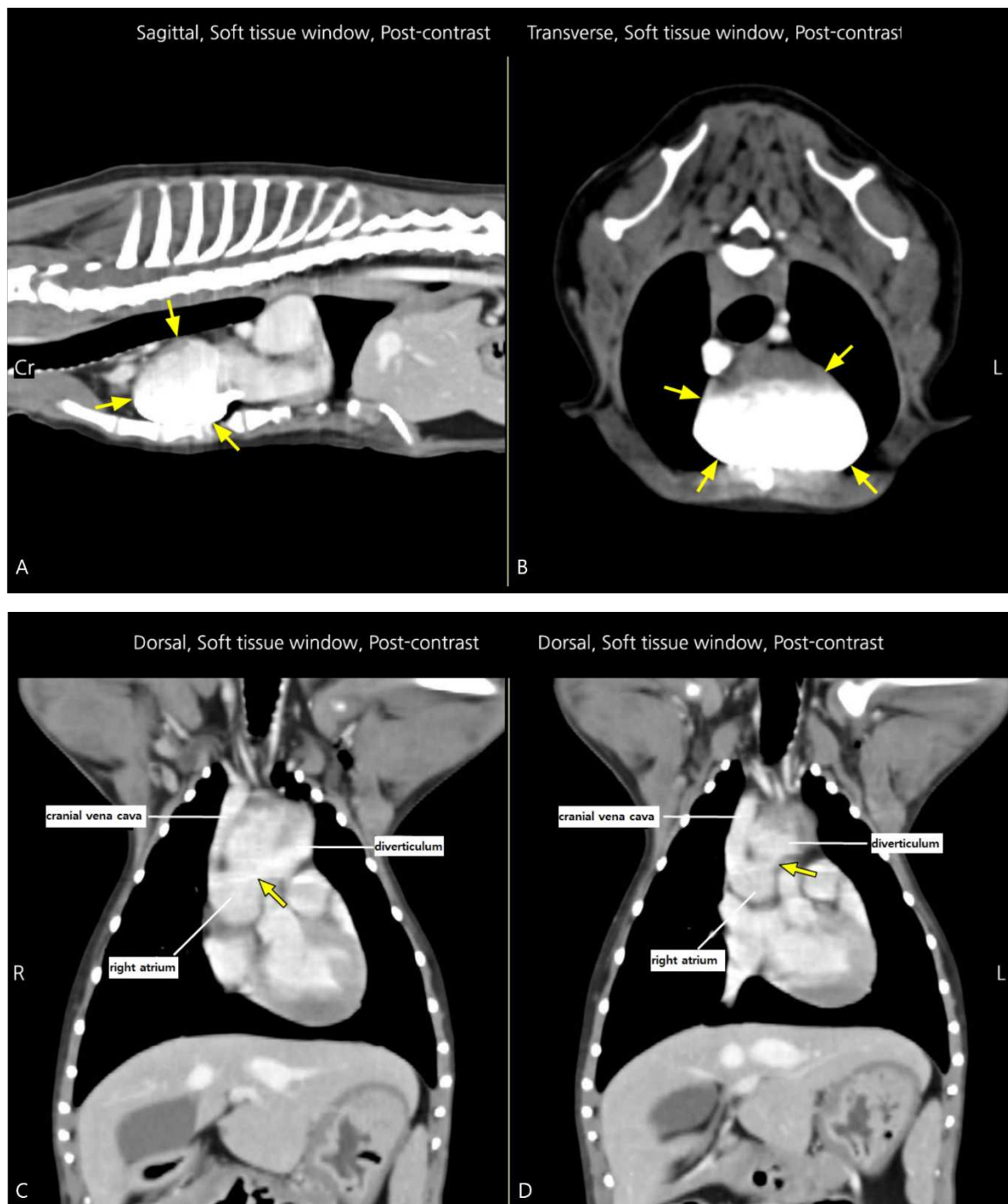


Figure 2. Contrast-enhanced computed tomography of the right atrial diverticular lesion.

Contrast-enhanced computed tomographic images showing a large blood-pool enhancing cystic structure adjacent to the cranial vena cava–right atrial region. (A) Sagittal and (B) transverse soft-tissue window images show the large contrast-enhancing

cystic cavity at the cranial aspect of the heart. (C, D) Dorsal reconstructed images show the spatial relationship among the cranial vena cava, right atrium and diverticular cavity. The lesion measured approximately 41 × 38 × 34 mm. Dependent contrast pooling within the cavity is consistent with slow or stagnant intracavitary blood flow, supporting a vascular or cardiac origin rather than a solid cranial mediastinal mass. Yellow arrows indicate the diverticular cavity and the region of communication with the right atrial area.

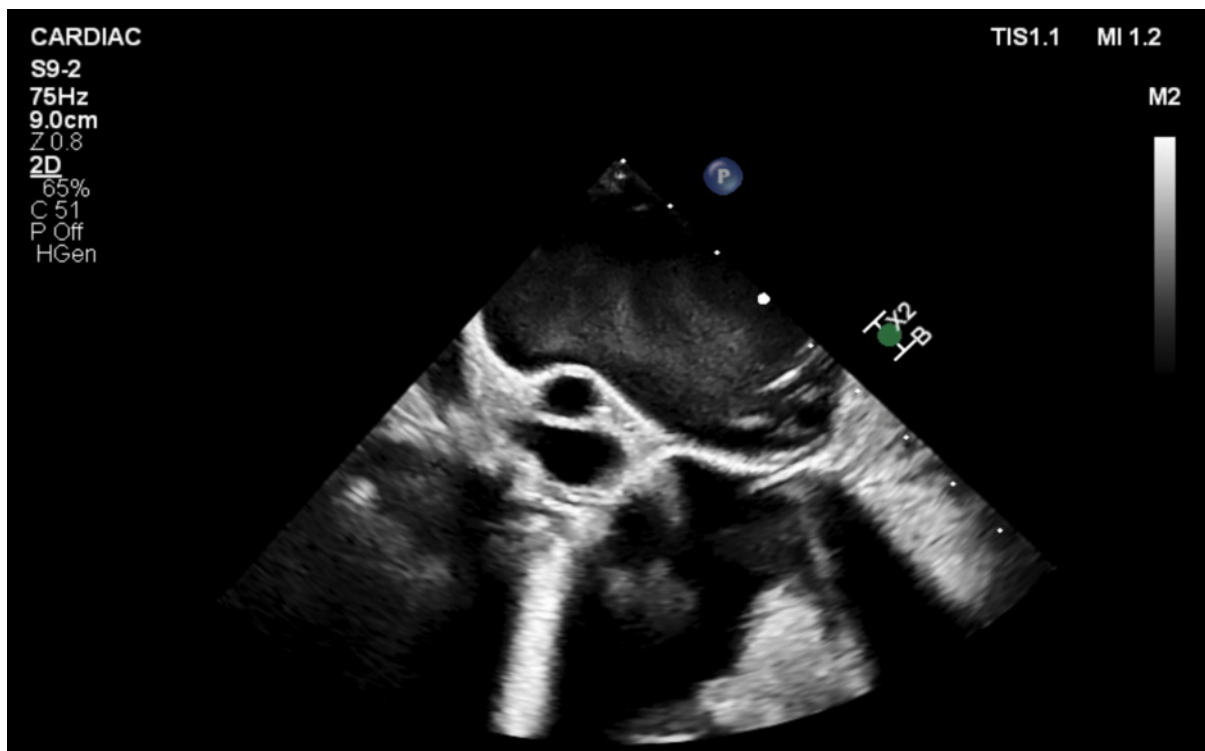


Figure 3. Echocardiographic features of the giant right atrial diverticulum.

Echocardiographic image showing a large anechoic chamber-like cavity adjacent to the right atrium and multiple echogenic structures within the cavity, interpreted as suspected intracavitary thrombi. The communicating orifice between the cavity and the right atrial region was not clearly visible on this still image but was identified on cine-loop assessment and estimated to measure approximately 7.5 mm (Supplementary Video 1). Histologic confirmation was not available; therefore, these structures were described as thrombus-like structures rather than confirmed thrombi.



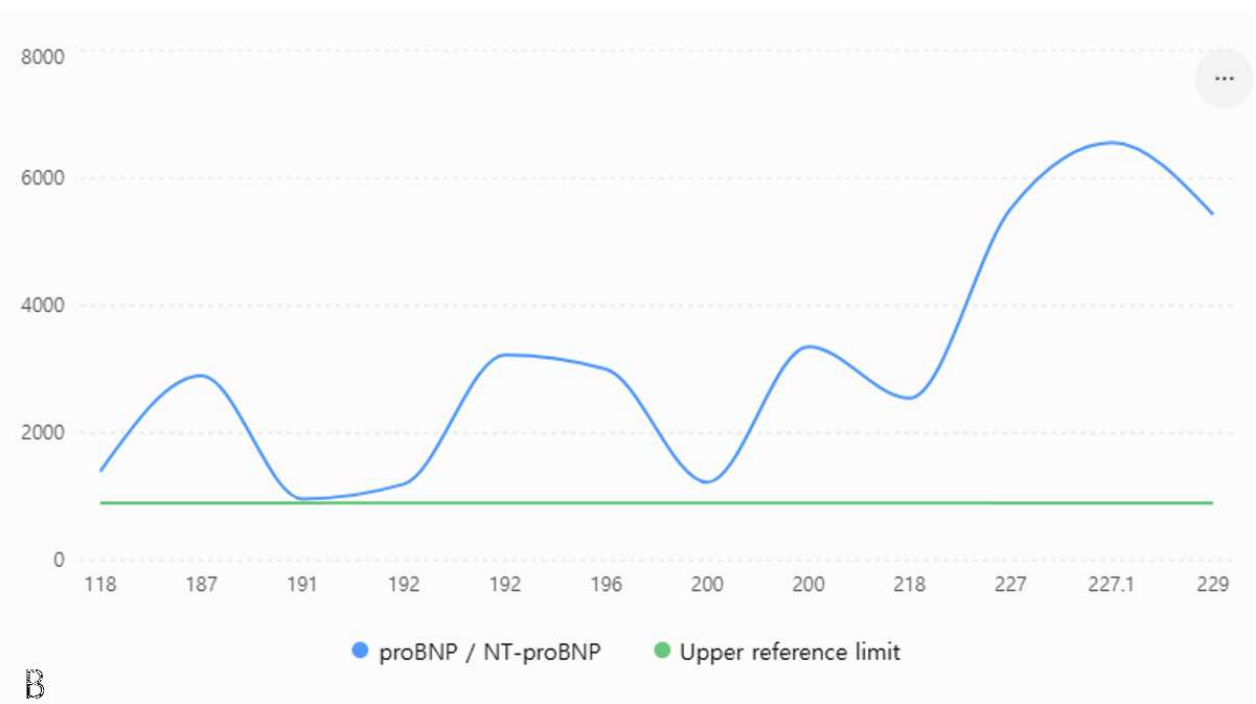
Figure 4. Electrocardiogram obtained during recurrent collapse.

Electrocardiography obtained approximately 52 months after the first recognized collapse episode documented a regular narrow-complex supraventricular tachycardia at 178 beats/min. The QRS duration was 66 ms, and the PR interval was 132 ms. In the clinical context of recurrent syncope, this rhythm was considered compatible with tachyarrhythmia-associated low-output collapse.



Figure 5. Terminal thoracic radiographs obtained at emergency presentation.

Ventrodorsal (A) and right lateral (B) thoracic radiographs obtained at terminal presentation show persistent broadening of the cranial cardiac or cranial mediastinal silhouette, corresponding to the previously identified right atrial diverticular vascular lesion. No obvious severe diffuse pulmonary oedema or massive pleural effusion is evident on the available views. These radiographs did not identify a definitive cause of terminal collapse.



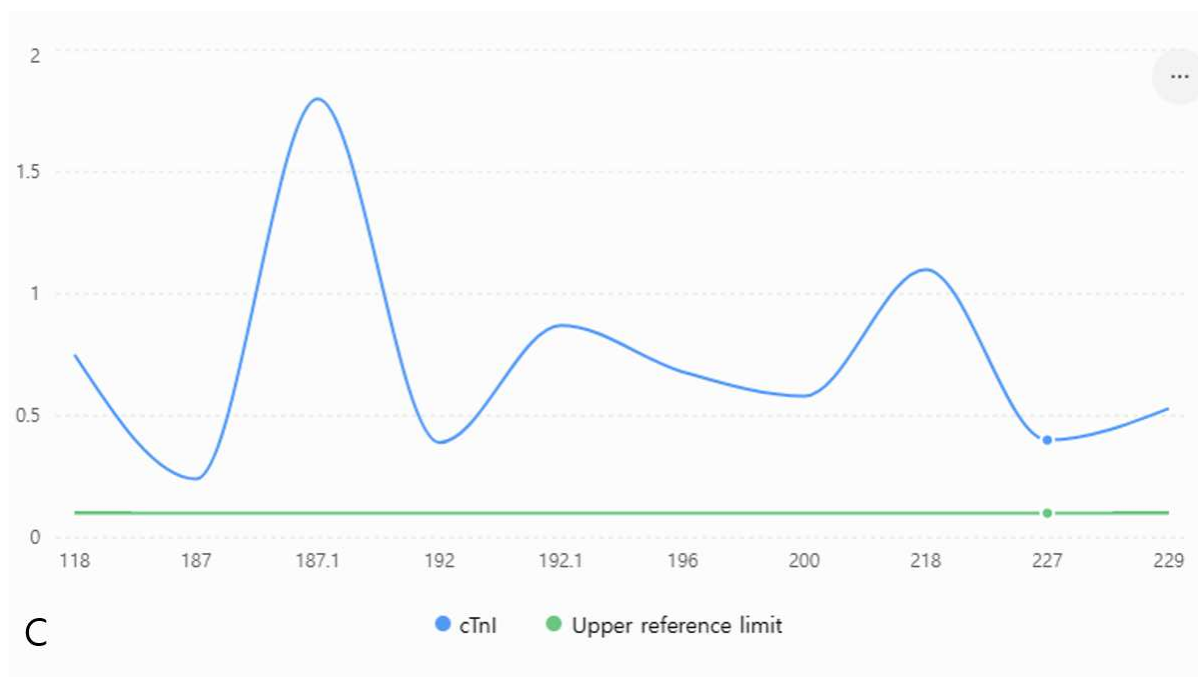


Figure 6. Serial biomarker trends during long-term follow-up.

(A) D-dimer concentrations, (B) proBNP/NT-proBNP concentrations and (C) cardiac troponin I concentrations plotted by weeks after the first recognized collapse episode. Green horizontal lines indicate the upper reference limits. Values reported as <50 mg/dL for D-dimer were plotted as 50 mg/dL for visualization. Serial biomarker changes were interpreted as supportive evidence of thromboembolic risk, progressive cardiac strain and recurrent myocardial injury, respectively, but were not diagnostic of pulmonary thromboembolism or specific for right atrial diverticulum..

Supplementary material



Right atrium
diverticulum.mp4

Supplementary Video 1. Echocardiographic cine loop of the right atrial diverticular cavity.

Echocardiographic cine loop showing the large chamber-like cavity adjacent to the right atrium and multiple mobile intracavitary echogenic structures interpreted as suspected thrombi.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [Rightatriumdiverticulum.mp4](#)