Pacemaker infection and endocarditis due to parvimonas micra: A case report and systematic review

Pacemaker infection and endocarditis due to *Parvimonas micra*: A case report and systematic review

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[1] [1] Abbreviations: CDIE, Cardiac device infective endocarditis; CT, Computed tomography; GNAR, Gram-negative anaerobic rods; GPAC, Gram-positive anaerobic cocci; IE, Infective endocarditis; MALDI-TOF MS, matrix-assisted laser desorption/ionization and time-of-flight mass spectrometry; PPM, Permanent pacemaker; TEE, transesophageal echocardiography; WBC, White blood cell count.

ABSTRACT

Infective endocarditis caused by *Parvimonas micra* is rare. Its clinical features are presented in this systematic review. We also describe the case of an 82-year-old man with infective endocarditis and pacemaker infection due to *P. micra*. There are some reports of recurrence during antimicrobial therapy; hence, careful follow-up is necessary.

Keywords: *Parvimonas micra*, pacemaker infection, endocarditis

Introduction

Infective endocarditis (IE) is associated with high morbidity and mortality [1-3] and is defined as a systemic septic disease with various manifestations [4]. The risk factors for endocarditis include increasing age [5], male sex [6], poor dentition [7], and cardiac implantable electronic devices [8]. Permanent pacemaker (PPM)-related infection has been recognized since the 1970s [9], with an occurrence of 1.19% during the lifetime of a device [10]. Cardiac device infective endocarditis (CDIE) has remarkably higher mortality than cardiac device infections without endocarditis. Approximately 60%–80% of causative pathogens of CDIE are of staphylococcal species, whereas other Gram-positive cocci accounted for only 4% of all cases [11].

Parvimonas micra (*P. micra*) is a Gram-positive anaerobic coccus that forms part of the normal commensal flora in the oral cavity and gastrointestinal tract [12]. Despite its low virulence, some patients may develop bacteremia [13], lung abscess [14], meningitis [15], cerebral and brain abscesses [16], spondylodiscitis [17], and iliopsoas abscesses [18]. *P. micra* endocarditis is uncommon, with few reports published previously [19,20]. Additionally, their pathogenic implication has been underestimated because of their slow growth and difficulty in identification.

This is the first report of right-sided IE involving a pacemaker by *P. micra*. Another

strength is that the patient deteriorated clinically despite negative blood cultures, emphasizing the importance of frequent follow-up with echocardiography given *P. micra* is difficult to grow. Herein, we present the case of pacemaker infection and a systematic review of endocarditis due to *P. micra*.

Description of the Case

An 82-year-old man came to our hospital with acute onset of fever and dizziness. He had a past medical history of pacemaker implantation due to sick sinus syndrome, undergoing synthetic graft replacement for an abdominal aortic aneurysm two decades ago, and hypertension. He was regularly taking aspirin 100 mg once a day and olmesartan medoxomil 10 mg once a day before admission. He was a social drinker but had no history of smoking. His occupation was a businessman, now retired, living in a retirement home, and originally independent in his daily activities, and he was walking with a cane. On admission, he was in acute distress, and his vital signs were clear consciousness; temperature, 38.4°C; blood pressure, 114/75 mm Hg; pulse rate, 74 beats/min (not pacemaker rhythm); respiratory rate, 18 breaths/min; and oxygen saturation, 94% at room temperature. Physical examination revealed a grade III diastolic murmur that was most audible in the left third intercostal space. He had poor oral hygiene and dental caries. No

immunological phenomena (Roth spots or Osler nodes) or vascular phenomena (Janeway lesions, conjunctival petechiae, or splinter hemorrhage) were observed. Lungs were clear to auscultation, and no abnormal abdominal findings were found. Neurological findings showed no sensory or motor deficits and no abnormalities related to central nervous system. Laboratory investigations revealed a total white blood cell count (WBC) of 15,000/μL (WBC differential comprising 87% neutrophils, 4% lymphocytes, 6% monocytes, and 3% eosinophils), hemoglobin level of 13.3 g/dL, platelet count of 121,000/μL, aspartate aminotransferase at 19 U/L, alanine aminotransferase at 8 U/L, lactate dehydrogenase of 314 IU/L, total bilirubin at 1.4 mg/dl, alkaline phosphatase of 189 IU/L, blood urea nitrogen 18 mg/dL, creatinine 0.96 mg/dL, and C-reactive protein level of 2.51 mg/dL. Urinalysis was negative for urinary white or red blood cells and nitrites, and the urine bacteriology test was negative. Contrast-enhanced computed tomography (CT) of the chest-abdomen-pelvis area revealed no inflammation or artificial blood vessel infection. After admission, we started ceftriaxone (2.0 g intravenously [IV]) every 24 hours empirically.

Blood cultures on admission grew clusters of Gram-positive cocci on day 5. Transthoracic echocardiography revealed moderate tricuspid valvular regurgitation and no signs of vegetation; however, on day 7, transesophageal echocardiography (TEE)

79 revealed vegetation on the tricuspid valve and pacemaker lead (mainly the atrial lead)
80 (Figure 1). Therefore, ceftriaxone was replaced with ampicillin (2.0 g IV) every 4 hours
81 and gentamicin (180 mg IV) every 24 hours. On day 12, we removed the pacemaker and
82 leads surgically and inserted a temporary pacemaker. On day 13, the blood cultured
83 pathogen on admission was confirmed to be *P. micra* using matrix-assisted laser
84 desorption/ionization and time-of-flight mass spectrometry (MALDI-TOF MS) (Bruker
85 Daltonic, Bremen, Germany). Since the patient was receiving appropriate antimicrobial
86 regimens, the culture pathogen tests for the pacemaker body and leads were negative.
87 Modified Duke criteria [21] supported the diagnosis of endocarditis: one major criterion
88 (evidence of endocardial involvement) and three minor criteria (predisposing heart
89 condition, fever over 38°C, and microbiologic evidence). With known susceptibility
90 results, gentamicin was terminated after a total of 6 days, and the subsequent
91 antimicrobial regimen was reduced to ampicillin (2.0 g IV) every 4 hours alone. Follow-
92 up blood cultures were negative._On day 22, repeated TEE was performed, and new
93 vegetation was noted on the temporary pacemaker lead and tricuspid valve (Figure 2). As
94 the patient was not dependent on the temporary pacemaker, it was removed entirely. The
95 patient's clinical signs and symptoms improved, and ampicillin 2.0 g IV every 4 hours
96 was continued for six weeks after removing the temporary pacemaker, and no subsequent

antimicrobial regimen was necessary. We did not change regular medications during the hospitalization or after discharge. The patient was discharged on day 64 without any sequelae, and no recurrence of infection and cardiac events were observed during 12 months of follow-up.

Systematic Literature Review

Two authors independently reviewed the titles and abstracts of database records, retrieved full texts for eligibility assessment, and extracted data (Figure 3). The respective search strategies for PubMed, Embase, and Ichushi-web are described in Figure 3, and twenty-eight articles were retrieved. After removing duplicates and irrelevant reports, four articles were selected [22,23]. After full-text evaluation, four articles comprising four patients were included (Figure 3 and Table 1).

Discussion

P. micra was originally classified as *Peptostreptococcus micros*. The classification of the Gram-positive anaerobic cocci (GPAC) group has been remarkably transformed by a new genetic classification method based on the 16S rRNA gene sequence introduced in the late 1990s. Thereafter, *P. micros* was reclassified as *P. micra* in 2006 [24]. GPAC are

bacilli of low virulence detected as frequently as Gram-negative anaerobic rods (GNAR), but their pathogenic implication has been underestimated because of their slow growth and difficulty in identification compared to GNAR [25]. As GPAC sometimes cause serious infections, identification of various GPAC species and recognition of their resistance patterns to antimicrobial agents are important. Recently, as MALTI-TOF MS has been introduced at various facilities, the identification of *P. micra* has become practical, and the number of reports has increased [26,27].

In the present case, the portal of entry of *P. micra* was considered to be the oral cavity. As *P. micra* is a part of the oral microbiota, the main risk factors for *P. micra* infection are dental procedures, periodontitis, tooth extraction, and abscesses or caries of the tongue apex [28]. Three of the five cases in our literature review had one of these factors.

Right-sided IE accounts for 5%–12% of all IE cases [29,30]. Causes of right-sided IE include (1) intravenous drug use, (2) CDIE, and (3) congenital heart disease. The recommended way to remove infected devices is to extract all leads and the pulse generator, which is associated with a complication rate of less than 2% [31]. Immediate removal of infectious devices is associated with a lower recurrence rate and mortality than retention or late removal [32,33]. Fortunately, the infected pacemaker in our case

was implanted approximately six months earlier. Transvenous extraction of leads with less than one year of dwelling time is generally possible with simple traction [34]; therefore, we were able to successfully remove the entire system transvenously.

In the current case, we found that the temporary pacemaker was initially infected with new vegetation, despite two subsequent negative blood cultures. There is limited conclusive evidence on the optimal timing of reinsertion of the cardiac device or central venous catheter after removal in cases of bacteremia. Some studies recommended that new transvenous lead placement should be delayed by at least 14 days after removing infectious devices [35]. Although the negative blood culture was confirmed by appropriate antimicrobial administration, clinicians should observe cases for persistent bacteremia if the patient's clinical signs deteriorate, as it may be difficult to detect *P. micra* in blood cultures because of the characteristics of anaerobic bacteria [36]. A prospective registry of CDIE reported that 11 of 434 enrolled patients with cardiovascular implantable electronic device infections developed another infection within six months [35]. The authors suggested that all such patients should be thoroughly evaluated, and the need for device re-implantation should be reevaluated This was particularly relevant in the current case. As negative blood cultures may not necessarily indicate improvement in IE due to *P. micra,* clinicians should repeat

echocardiograms to evaluate for the presence of IE.

In our review, two of the five cases had severe valve dysfunction, eventually requiring surgical intervention [19,20]. Despite appropriate antimicrobial therapy, invasive procedures with CT-guided drainage were also required in one case presenting with a lung abscess [23]. In another case, antimicrobial de-escalation resulted in a flare-up of the infection, and antimicrobial susceptibility was detected [22], suggesting that the possibility of mixed infections caused by anaerobic bacteria should always be considered. Although it is difficult to make generalizations from this small number of cases, *P. micra* sometimes progresses to abscess formation inconspicuously, and some studies have reported cases of mixed infections. Therefore, when blood tests show an increased inflammatory response or fever during the treatment period, it is advisable to consider the possibility of recurrence, new lesions, or mixed infection.

In summary, *P. micra* can be a causative pathogen of IE and pacemaker infections. Because of the difficulty in identifying the organism using traditional methods, the introduction of MALDI-TOF MS is expected to improve the diagnostic rate of IE caused by *P. micra*, which can sometimes lead to serious complications, suggesting the importance of frequent follow-up. As only five cases have been reported, the accumulation of future cases is expected.

Declarations

Ethics approval and consent to participate

The treatment for the patient was performed in accordance with the tenets of the Declaration of Helsinki. The patient reported in the study provided written informed consent for all procedures.

Consent for publication

The patient enrolled in the study provided written informed consent for the publication of his clinical details and accompanying images. A copy of the written informed consent is available for the journal.

Availability of data and materials

Not applicable.

Conflicts of interests

The authors have no financial disclosures or conflicts of interest to declare.

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187 **Authors' contributions**

188 T.S. wrote a draft of the manuscript. K.I., T.M., Y.K., and N.M. supervised the study and
189 edited the manuscript. Y.K., H.A., and N.K. were the attending physicians of this
190 patient. F.K. and K.I. participated in the literature review. All authors reviewed the final
191 manuscript and approved its contents.

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197 **References**

198 [1] D.R. Murdoch, G.R. Corey, B. Hoen, J.M. Miró, V.G. Fowler Jr, A.S. Bayer, et al.,
199 Clinical Presentation, Etiology, and Outcome of Infective Endocarditis in the 21st
200 Century: the International Collaboration on Endocarditis–Prospective Cohort Study,
201 Arch. Intern. Med. 169 (2009) 463–473.

202 [2] X. Duval, C.S. Suty, F. Alla, M. Salvador-Mazenq, Y. Bernard, M. Weber, et al.,
203 Endocarditis in patients with a permanent pacemaker: a 1-Year epidemiological survey
204 on infective endocarditis due to valvular and/or pacemaker infection, Clin. Infect. Dis.

205 39 (2004) 68–74.

206 [3] E. Mylonakis, S.B. Calderwood, Infective endocarditis in adults, N. Engl. J. Med.

207 345 (2001) 1318–1330.

208 [4] T.J. Cahill, B.D. Prendergast, Infective endocarditis, Lancet. 387 (2016) 882–893.

209 [5] E. Durante-Mangoni, S. Bradley, C. Selton-Suty, M.F. Tripodi, B. Barsic, E. Bouza,

210 et al., Current features of infective endocarditis in elderly patients: results of the

211 International Collaboration on Endocarditis Prospective Cohort Study, Arch. Intern.

212 Med. 168 (2008) 2095–2103.

213 [6] E.E. Hill, P. Herijgers, P.Claus, S. Vanderschueren, M.C. Herregods, W.E.

214 Peetermans, Infective endocarditis: changing epidemiology and predictors of 6-month

215 mortality: a prospective cohort study, Eur. Heart J. 28 (2007) 196–203.

216 [7] P.B. Lockhart, M.T. Brennan, M. Thornhill, B.S. Michalowicz, J. Noll, F.K. Bahrani-

217 Mougeot, et al., Poor oral hygiene as a risk factor for infective endocarditis-related

218 bacteremia, J. Am. Dent. Assoc. 140 (2009) 1238–1244.

219 [8] L. Østergaard, N. Valeur, A. Wang, H. Bundgaard, M. Aslam, G. Gislason, et al.,

220 Incidence of infective endocarditis in patients considered at moderate risk, Eur. Heart J.

221 40 (2019) 1355–1361.

222 [9] I.S. Schwartz, N. Pervez, Bacterial endocarditis associated with a permanent

223 transvenous cardiac pacemaker, JAMA. 218 (1971) 736–737.

224 [10] T. Olsen, O.D. Jørgensen, J.C. Nielsen, A.M. Thøgersen, B.T. Philbert, J.B.

225 Johansen, Incidence of device-related infection in 97 750 patients: clinical data from the

226 complete Danish device-cohort (1982-2018), Eur. Heart J. 40 (2019) 1862–1869.

227 [11] M.R. Sohail, D.Z. Uslan, A.H. Khan, P.A. Friedman, D.L. Hayes, W.R. Wilson, et

228 al., Management and outcome of permanent pacemaker and implantable cardioverter-

229 defibrillator infections, J. Am. Coll. Cardiol. 49 (2007) 1851–1859.

230 [12] D.A. Murdoch, Gram-positive anaerobic cocci, Clin. Microbiol. Rev. 11 (1998)

231 81–120.

232 [13] M. Boattini, G. Bianco, R. Cavallo, C. Costa, Parvimonas micra bacteremia

233 following endoscopic retrograde cholangiopancreatography: A new route of infection,

234 Anaerobe. 54 (2018) 136–139.

235 [14] S.S. Yun, H.S. Cho, M. Heo, J.H. Jeong, H.R. Lee, S. Ju, et al., Lung abscess by

236 Actinomyces odontolyticus and Parvimonas micra co-infection presenting as acute

237 respiratory failure: a case report, Med (Baltimore). 98 (2019) e16911.

238 [15] J.H. Ko, J.Y. Baek, C.I. Kang, W.J. Lee, J.Y. Lee, S.Y. Cho, et al., Bacteremic

239 meningitis caused by Parvimonas micra in an immunocompetent host, Anaerobe. 34

240 (2015) 161–163.

241 [16] J.P. Frat, C. Godet, G. Grollier, J.L. Blanc, R. Robert, Cervical spinal epidural
242 abscess and meningitis due to Prevotella oris and Peptostreptococcus micros after
243 retropharyngeal surgery, Intensive Care Med. 30 (2004) 1695.
244 [17] H. Uemura, K. Hayakawa, K. Shimada, M. Tojo, M. Nagamatsu, T. Miyoshi-
245 Akiyama, et al., Parvimonas micra as a causative organism of spondylodiscitis: a report
246 of two cases and a literature review. Int. J. Infect. Dis. 23 (2014) 53–55.
247 [18] T. Sawai, S. Koga, S. Ide, S. Yoshioka, N. Matsuo, H. Mukae, An iliopsoas abscess
248 caused by Parvimonas micra: a case report, J. Med. Case Rep. 13 (2019) 47.
249 [19] C.A. Gomez, D.A. Gerber, E. Zambrano, N. Banaei, S. Deresinski, B.G.
250 Blackburn, First case of infectious endocarditis caused by Parvimonas micra, Anaerobe.
251 36 (2015) 53–55.
252 [20] D. Ho, G. Ang, C. Er, S.F. Yap, V. Meyyur Aravamudan, An unusual presentation
253 of Parvimonas micra infective endocarditis, Cureus. 10 (2018) e3447.
254 [21] L.M. Baddour, W.R. Wilson, A.S. Bayer, V.G. Fowler, I.M. Tleyjeh, M.J. Rybak, et
255 al., Infective endocarditis in adults: diagnosis, antimicrobial therapy, and management
256 of complications: a scientific statement for healthcare professionals from the American
257 Heart Association, Circulation. 132 (2015) 1435–1486.
258 [22] M. Maki, A. Kawakami, I. Suzuki, S. Fujie, M. Arai, E. Watanabe, J. Kunimatsu, et

al., A case of infective endocarditis and lung abscess associated with Parvimonas micra bacteremia, The Japanese Society of Internal Medicine. 632 (2017) 34. [Japanese]

[23] K. Iwasaki, K. Sumida, D. Yoshimura, H. Inoue, A case of infective endocarditis caused by multiple anaerobic bacteria, Journal of Infectious Diseases. 91 (2017) 858. [Japanese]

[24] B.J. Tindall, J.P. Euzéby, Proposal of Parvimonas gen. nov. and Quatrionicoccus gen. nov. as replacements for the illegitimate, prokaryotic, generic names Micromonas Murdoch and Shah 2000 and Quadricoccus Maszenan et al. 2002, respectively, Int. J. Syst. Evol. Microbiol. 56 (2006) 2711–2713.

[25] E.C. Murphy, I.M. Frick, Gram-positive anaerobic cocci--commensals and opportunistic pathogens, FEMS Microbiol. Rev. 37 (2013) 520–553.

[26] T. Watanabe, Y. Hara, Y. Yoshimi, Y. Fujita, M. Yokoe, Y. Noguchi, Clinical characteristics of bloodstream infection by Parvimonas micra: retrospective case series and literature review, BMC Infect. Dis. 20 (2020) 578.

[27] A.C. Veloo, M. Erhard, M. Welker, G.W. Welling, J.E. Degener, Identification of gram-positive anaerobic cocci by MALDI-TOF mass spectrometry, Syst. Appl. Microbiol. 34 (2011) 58–62.

[28] F. Cobo, J. Rodríguez-Granger, A. Sampedro, L. Aliaga-Martínez, J.M. Navarro-

277 Marí, Pleural effusion due to Parvimonas micra. A case report and a literature review of
278 30 cases, Rev. Esp. Quimioter. 30 (2017) 285–292.
279 [29] G. Habib, P. Lancellotti, M.J. Antunes, M.G. Bongiorni, J.P. Casalta, F. Del Zotti,
280 et al., 2015 ESC Guidelines for the management of infective endocarditis: the task force
281 for the management of infective endocarditis of the European Society of Cardiology
282 (ESC). Endorsed by: European Association for Cardio-Thoracic Surgery (EACTS), the
283 European Association of Nuclear Medicine (EANM), Eur. Heart J. 36 (2015) 3075–
284 3128.
285 [30] S. Leone, V. Ravasio, E. Durante-Mangoni, M. Crapis, G. Carosi, P.G. Scotton, et
286 al., Epidemiology, characteristics, and outcome of infective endocarditis in Italy: the
287 Italian Study on Endocarditis, Infection. 40 (2012) 527–535.
288 [31] J.A. Sandoe, G. Barlow, J.B. Chambers, M. Gammage, A. Guleri, P. Howard, et al.,
289 Guidelines for the diagnosis, prevention and management of implantable cardiac
290 electronic device infection. Report of a joint Working Party project on behalf of the
291 British Society for Antimicrobial Chemotherapy (BSAC, host organization), British
292 Heart Rhythm Society (BHRS), British Cardiovascular Society (BCS), British Heart
293 Valve Society (BHVS) and British Society for Echocardiography (BSE), J. Antimicrob.
294 Chemother. 70 (2015) 325–359.

295 [32] K.Y. Le, M.R. Sohail, P.A. Friedman, D.Z. Uslan, S.S. Cha, D.L. Hayes, et al.,
296 Impact of timing of device removal on mortality in patients with cardiovascular
297 implantable electronic device infections, Heart Rhythm. 8 (2011) 1678–1685.
298 [33] E. Athan, V.H. Chu, P. Tattevin, C. Selton-Suty, P. Jones, C. Naber, et al., Clinical
299 characteristics and outcome of infective endocarditis involving implantable cardiac
300 devices, JAMA. 307 (2012) 1727–1735.
301 [34] M. Döring, S. Richter, G. Hindricks, The diagnosis and treatment of pacemaker-
302 associated infection, Dtsch. Arztebl. Int. 115 (2018) 445–452.
303 [35] T.A. Boyle, D.Z. Uslan, J.M. Prutkin, A.J. Greenspon, L.M. Baddour, S.B. Danik,
304 et al., Reimplantation and repeat infection after cardiac-implantable electronic device
305 infections: Experience From the MEDIC (Multicenter Electrophysiologic Device
306 Infection Cohort) Database, Circ. Arrhythm. Electrophysiol. 10 (2017). E004822.
307 [36] A. Durovic, N. Eberhard, S. Schären, A.F. Widmer, Parvimonas micra as a rare
308 cause of spondylodiscitis - case series from a single centre, Swiss Med. Wkly. 150
309 (2020) w20272.
310

311 **Figure Captions**

312 Figure 1. Vegetation on the atrial lead of the pacemaker and tricuspid valve

313 Transesophageal echocardiography reveals a 15.7-mm fluttering structure in the atrial

314 lead of the pacemaker and a 10.4-mm vegetation-like fluttering structure in the tricuspid

315 valve.

316

317 Figure 2. Removal of the pacemaker atrial lead

318 A red deposit on the pacemaker atrial lead that was removed is shown.

319

320 Figure 3. Flow diagram of the systematic literature review

321

322

Figure Legends

Figure 1. Flow diagram of the systematic literature review

Figure 2. Vegetation on the atrial lead of the pacemaker and tricuspid valve

Transesophageal echocardiography revealed a 15.7 mm fluttering structure in the atrial lead of the pacemaker and a 10.4 mm vegetation-like fluttering structure in the tricuspid valve

Figure 3. Removed pacemaker atrial lead

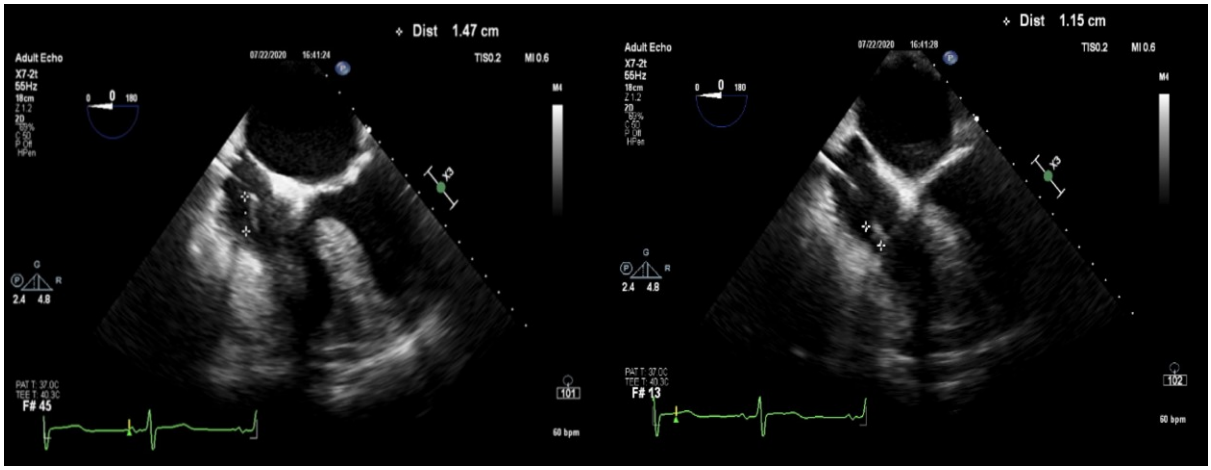
A red deposit on the pacemaker atrial lead that was removed was found

Tables

Table 1. Characteristics of patients with endocarditis due to *Parvimons micra*

Case	Year of Report	Ref. No	Age (years)	Gender	Risk Factor for Infective Endocarditis	Infected Valves	Echocardiographic findings/Comorbidities	Diagnostic method	Days to Diagnosis	Days to positive Blood Culture	Antimicrobial therapy	Cardiac Surgery
1	2015	[19]	71	M	None	A, M-N	Perivalvular abscess	TEE	8 days	5 days	Vancomycin plus Nafcillin plus Gentamicin → Ampicillin/Sulbactam	Aortic valve and mitral valve replacement
2	2017	[40]	48	F	Tooth Extraction	T-N, PL	Vegetation Septic pulmonary embolism Bloodstream infection of *Fusobacterium nucleatum* (Co-infection)	TEE	69 days	4 days	Ampicillin/Sulbactam → plus Gentamicin → Ampicillin → Plus Metronidazole	No
3	2017	[39]	78	M	Laryngeal cancer and total laryngectomy, Poor oral hygiene	A-N	Septic Pulmonary embolism, lung abscess	TTE	NA	NA	Ampicillin/Sulbactam → Clindamycin	Drainage for lung abscess
4	2018	[20]	42	M	Tooth Extraction	M-ME	Mobile friable/ vegetations arising from the mitral prosthetic valve	TEE	22 days	NA	Ceftriaxone plus Vancomycin → Penicillin G	Reoperation of Mitral Valve for control heart failure
5	2020	PR	82	M	Poor Oral Hygiene	T-N, PL	Vegetation along pacemaker leads and on tricuspid valve	TEE	7 days	5 days	Ceftriaxone → Ampicillin plus Gentamicin → Ampicillin	Removal of permanent pacemaker

PR: present report; N/A: not available; Valve: T, tricuspid; A: aortic; M: mitral; N: native; P: prosthetic; ME: mechanical; PL: pacemaker lead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55Hz
07/22/2020 16:41:24
TIS0.2 MI 0.6
F# 45
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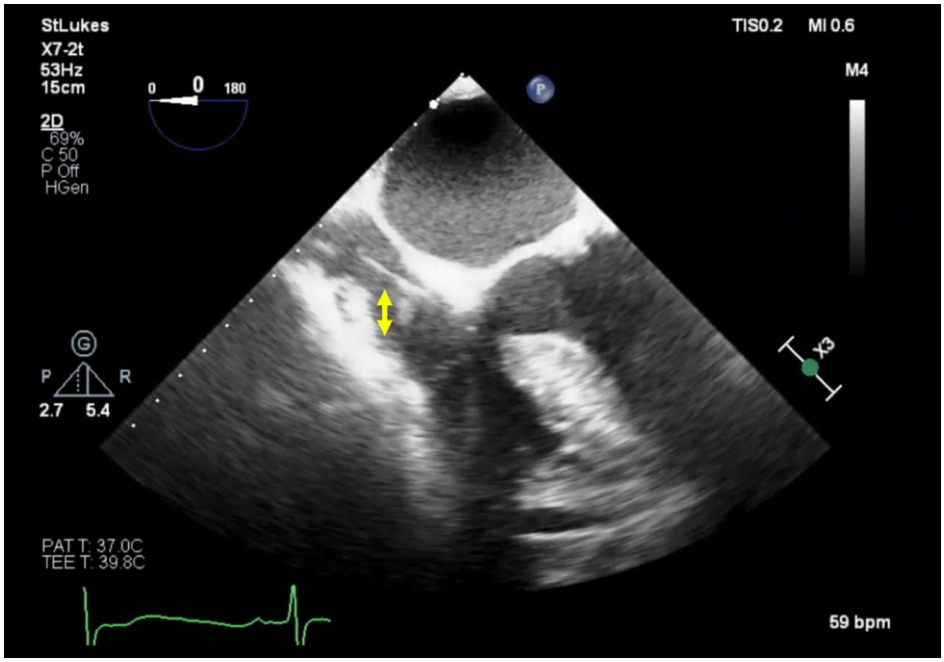
F# 13


StLukes
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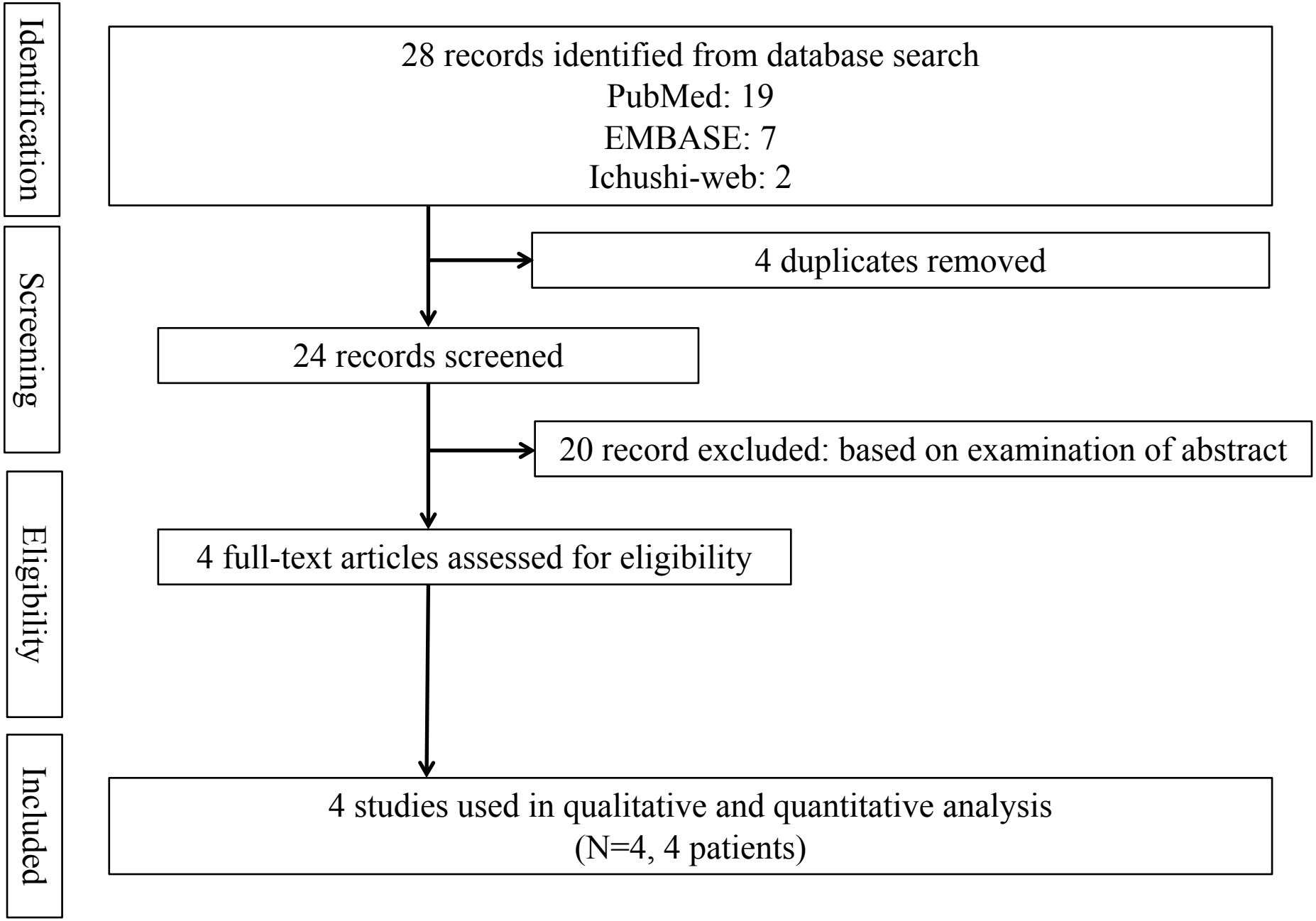
53Hz
15cm
2D
69%
C 50
P Off
HGen
0 0 180
TIS0.2 MI 0.6
M4
P R
2.7 5.4
PAT T: 37.0C
TEE T: 39.8C
59 bpm


Identification
Screening
Eligibility
Included
28 records identified from database search
PubMed: 19
EMBASE: 7
Ichushi-web: 2
4 duplicates removed
24 records screened
20 record excluded: based on examination of abstract
4 full-text articles assessed for eligibility
4 studies used in qualitative and quantitative analysis
(N=4, 4 patients)