Intracolonic cardiac pacemaker: A case of device migration with colon perforation out of a subcutaneous epifascial pocket



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Introduction

In common practice, pacemaker generators are implanted in a subcutaneous/submuscular pectoral or axillary pocket. However, an abdominal (or even intra-abdominal) implantation sometimes represents a valuable alternative site, particularly in young or slim patients, including children. We describe an impressive patient scenario, with intra-abdominal device migration complicated by colon perforation, in an adult patient with congenital complete heart block. The postoperative course was additionally complicated by secondary cardiovascular implantable electronic device (CIED) infection. To our best knowledge, this has been described very rarely so far, mostly in pediatric patients and never before out of a subcutaneous epifascial pocket. This report highlights possible complications and their management in patients with abdominally implanted pacemaker devices, taking in account current practice recommendations.

Case report

A 53-year-old woman consulted our emergency department for increasing colic-like abdominal pain starting 4 days before admission. She was known to have a congenital third-degree atrioventricular block that required implantation of a transvenous dual-chamber pacemaker in 1982. Extended-length electrodes were used and implanted through the right subclavian vein and then tunneled to a subcutaneous left epigastric pocket, where the device was implanted. Over time, multiple pulse generator changes were uneventful. The last generator replacement (Medtronic Adapta ADDDR06, Minneapolis, MN) with reimplantation in the right abdominal epifascial space was performed in 2011. Other than obesity

KEYWORDS CIED infection; Colon perforation; Device migration; Endocarditis; Heart block; Lead extraction; Pacemaker (Heart Rhythm Case Reports 2018;4:497–500) (body mass index 33.8 kg/m^2), the patient's medical history was uneventful. She was not on any permanent medication. At presentation, our patient was subfebrile (37.7°C), blood pressure 161/88 mm Hg, and in regular sinus rhythm at 88 beats/min with atrioventricular synchronous pacing on the surface electrocardiogram. On examination the pacemaker scar was free of irritation, but we found a slight tenderness on palpation in the right and left upper abdominal quadrant. Only C-reactive protein was elevated (55 mg/L). A consecutively ordered computed tomography (CT) scan revealed migration of the generator into the left intraluminal colic flexure with a sealed perforation at the point of entry of the electrodes (right atrium / right ventricle: Vitatron Helifix unipolar 6 mm, Maastricht, The Netherlands) in the right third of the transverse colon (Figure 1). Pacemaker interrogation showed normal values with an underlying junctional escape rhythm of 35-40 beats/min. Therapeutic options were immediately discussed in a multidisciplinary team and we decided to extract the complete pacemaker system. Since the risk of unsuccessful transvenous lead extraction needing sternotomy and on-pump cardiopulmonary bypass was considered high, given the advanced age of the electrodes, we decided to undertake a stepwise approach with primary removal of the generator and capping of the leads to minimize the perioperative risks.

After placement of a temporary transjugular pacemaker wire, the patient underwent surgery. First, the subcutaneous electrodes were cut through and capped using a right-sided submammarian approach. There was no sign of local inflammation. Consecutively, the explorative laparotomy revealed penetration of the electrodes through the abdominal wall into the transverse colon with, surprisingly, no signs of peritonitis. The pulse generator and electrodes were removed from the point of entry into the transverse colon and a segment colectomy with end-to-end anastomosis was performed. An incidental superficial tear of the upper splenic pole capsule with minor bleeding could be managed conservatively (Figure 2). The postoperative course was uneventful. The echocardiogram showed no signs of lead or valve

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KEY TEACHING POINTS

- Transperitoneal cardiac pacemaker migration is a very rare complication in abdominally implanted devices.
- In case of abdominal symptoms, further imaging is needed in order to rule out device-related colon perforation.
- Device-related colon perforation should be regarded as an "extended" pocket infection and complete system extraction should be performed.

endocarditis, and left ventricular ejection fraction was normal. Blood cultures remained negative. However, sonication of the submammarian electrode samples showed polymicrobial flora, including, for example, Enterococcus avium, Pseudomonas aeruginosa, Citrobacter freundii, and Proprionibacterium acnes. After an antibiotic regimen containing piperacillin/tazobactam for 8 days, we opted for a complete transvenous extraction of the remaining electrodes, with an intended contralateral transvenous implantation at the earliest 3 days later. Recuperating from acute surgical procedure, the patient declined the suggested staged lead extraction owing to the potential peri-interventional risks and prolonged temporary transjugular pacemaker dependency. In agreement with our infectious disease specialist, we finally performed a direct left pectoral dual-chamber pacemaker implantation through the subclavian vein (Medtronic E3DR01; RA Medtronic 5076-52, RV Medtronic 5076-58). The potential risks of future CIED infection were taken in account and an absorbable antibacterial envelope (TYRX, Medtronic) was used. Owing to a microdislocation, the right atrial lead had to be replaced 2 days later. Finally, the patient was discharged after a total of 14 days.

Two months later the patient developed malaise, chills, and weight loss. C-reactive protein was slightly elevated

while leukocytes remained normal. Blood cultures were again negative; however, transesophageal echocardiography showed a 10-mm mobile mass adhering to one of the atrial leads. A positron emission tomography–CT showed strong fludeoxyglucose uptake of the capped old "1982" electrodes along the thoracic wall and less uptake of the atrial electrodes, correlating to the echocardiographic mass. There was physiologic uptake in the left pectoral pocket (Figure 3).

Postulating an ongoing low-grade CIED infection, the indication for total extraction of both systems was given. First, an epicardial dual-chamber pacemaker (Medtronic A3DR01; LA Medtronic 4968-60; LV Myopore 511212 [Greatbatch Medical, Minneapolis, MN]) was implanted over a left anterior mini-thoracotomy with a submammarian/submuscular pocket. Then complete transvenous extraction of the left pectoral system as well as of the remaining right-sided electrodes was performed using locking stylets and gentle traction; the tip of the atrial "1982" electrode remained in situ. Sonication of the electrode samples were positive for Enterococcus avium (electrodes/generator), Pseudomonas aeruginosa, and Escherichia coli. According to the recommendations of the guidelines, the patient was prescribed a 4-week antibiotic regimen with piperacillin/tazobactam, with adoption of the regimen to vancomycin combined with ciprofloxacin after 3 weeks owing to beta-lactam-induced neutropenia. The patient additionally developed asymptomatic paroxysmal atrial fibrillation. Considering the patient's low CHA2DS2-VASc score (1 point for female sex), we did not initiate oral anticoagulation. Twelve months after complete extraction the patient is well and with no signs of recurrent infection.

Discussion

In common practice, pacemaker generators are implanted in a subcutaneous/submuscular pectoral or axillary pocket. However, in young and slim patients, including children, an abdominal approach is still used and intra-abdominal device migration has rarely been described. Intraperitoneal pacemaker migration resulting in bowel perforation is an even



Figure 1 Computed tomography overview posteroanterior (A) and axial view (B) showing intracolonic generator and electrodes (*white arrows*).



Figure 2 A: Submammarian incision showing no signs of inflammation/infection; electrodes were capped at this site. B: Surgical view during laparotomy showing entry site of generator and electrodes into the colon.

rarer event and has been described in a handful of pediatric patients, where the generator had to be placed into the abdominal cavity because of insufficient subcutaneous or muscle tissue.¹

Whereas in adults, only 2 cases of colon perforation out of an abdominal pocket have been described almost 3 decades ago, in 1986 Siclari and colleagues² mentioned the pulse generator reaching the ascending colon out of the right flank via a retroperitoneal pathway. Three years prior a pocket infection was managed conservatively, so chronic subclinical infection of the new pocket most likely promoted protrusion into the colon. Even earlier, Metzger and colleagues³ reported perforation of the device into the transverse colon out of a submuscular pocket after multiple generator changes. Those cases happened in a period where pacemaker devices were not as widely used and their complication management differed from current practice. Nevertheless, one needs to be aware that bowel perforation is related to high morbidity and mortality, including peritonitis and potentially lethal sepsis. Therefore, involved physicians should always aim for timely viscerosurgical intervention.

The exact pathogenesis of delayed pocket erosions and device migrations remains incompletely understood. However, several studies have suggested that chronic pocket infection is often the most likely cause.¹ Da Costa and colleagues⁴ showed that chronic smoldering infection related to perioperative contamination with skin flora causes pocket erosion many months to years later. The use of an antibacterial envelope (AigisRx/TYRX, Medtronic) has shown promising results, significantly reducing CIED infections in conventional pocket sites.⁵ Owing to the postulated pathomechanism, the use of an antibacterial envelope might reduce the risk of chronic pocket infections and, therefore, the consequences of device migration.

In our case, the last generator change was performed 5 years prior to manifestation of transperitoneal migration, suggesting a slow pathophysiologic process. The device had been placed in a vital subcutaneous pocket, which should represent a strong barrier toward the abdominal cavity. In retrospect, the device must have slowly "wandered" laterally. This was noted on the annual pacemaker interrogations since the last generator change, but was not considered an issue





Figure 3 A: Positron emission tomography–computed tomography showing fludeoxyglucose uptake of the capped, old "1982" electrodes (*black arrow*). B: Transesophageal echocardiography 40° , mobile mass (*white arrow*) adhering to the electrodes at the right atrial level.

owing to the patient's well-being. Additionally, palpation of the generator was difficult owing to the presence of extensive abdominal fat tissue. Finally, the exact time of migration through the abdominal muscular layer into the abdominal cavity remains unclear.

In the case of a pocket infection, current guidelines recommend complete pacemaker system extraction.⁶ Prior to extraction, transesophageal echocardiography or positron emission tomography–CT might also be mandated to identify lead vegetations along the greater venous system and cardiac cavities. This is a strategy we strongly support; however, in this specific case, we respected the patient's preferences to follow a primarily conservative and abbreviated therapeutic approach (including intravenous antibiotics and early contralateral transvenous pacemaker implantation), initially bypassing complete lead extraction and prolonged temporary pacemaker dependency. As described above, this strategy unfortunately led to repeat surgery. However, it remains uncertain if an initially prolonged antibiotic regimen would have resulted in a different outcome.

Regarding antimicrobial management of lead endocarditis or pocket infections, it is uniformly suggested to initially treat empirically with broad-spectrum antibiotics covering common gram-positive bacteria, such as S. aureus and streptococci groups. Once the causative pathogen has been identified, the antimicrobial regimen needs to be tailored. Overall, it remains currently unknown if an antimicrobial therapy alone, without device and lead extraction, might be clinically efficient enough in selected cases, the latter being a similar situation that we encountered, including negative blood cultures and no visible vegetations on echocardiography. Noteworthy in this context, it has been shown that up to 30% of all cases with proven lead endocarditis show negative blood cultures.⁷ In our case, we stopped the antibiotic treatment owing to lack of potential benefit and followed a "wait-and-see" strategy. After diagnosis of unilateral, possibly bilateral endovenous lead endocarditis weeks later, we implanted an epicardial system over a left anterior mini-thoracotomy, which is a safe state-of-the-art procedure, especially in cases of proven active endovenous infection and pacemaker dependency.

To our best knowledge, this is the first described case of a pacemaker migration with colon perforation out of a subcutaneous epifascial pocket. This must be regarded as an "extended" pocket infection with high likelihood of complete lead contamination, and a total system extraction should always be performed. Therefore, it is important to have a high index of suspicion for transperitoneal device migration if a patient suffers from abdominal symptoms any time after pacemaker implantation in the abdominal wall. As shown, delays in therapy might have relevant, even life-threatening consequences.

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References

- Dodge-Khatami A, Backer CL, Meuli M, Pretre R, Tomaske M, Mavroudis C. Migration and colon perforation of intraperitoneal cardiac pacemaker systems. Ann Thorac Surg 2007;83:2230–2232.
- Siclari F, Uhlschmid G, Zwicky P, Turina M. Intracolonic migration of a pacemaker generator. Thorac Cardiovasc Surg 1986;34:338–339.
- Metzger B, Lachmann W. [Cardiac pacemaker perforation of the transverse colon in the infected pacemaker system]. Z Gesamte Inn Med 1980; 35:345–347.
- Da Costa A, Lelievre H, Kirkorian G, et al. Role of the preaxillary flora in pacemaker infections: a prospective study. Circulation 1998;97:1791–1795.
- Mittal S, Shaw RE, Michel K, et al. Cardiac implantable electronic device infections: incidence, risk factors, and the effect of the AigisRx antibacterial envelope. Heart Rhythm 2014;11:595–601.
- Baddour LM, Epstein AE, Erickson CC, et al. Update on cardiovascular implantable electronic device infections and their management: a scientific statement from the American Heart Association. Circulation 2010;121:458–477.
- Deharo JC, Quatre A, Mancini J, et al. Long-term outcomes following infection of cardiac implantable electronic devices: a prospective matched cohort study. Heart 2012;98:724–731.