A 53-year-old man presented to an outpatient clinic with fever of unknown origin and anemia. His past medical history was remarkable for hypertension, diabetes mellitus, ST-segment elevation myocardial infarction 3 years earlier, complicated by severe left ventricular systolic dysfunction (left ventricular ejection fraction, 32%), implantable cardioverter-defibrillator implantation 2 years earlier, and elective percutaneous coronary intervention of the circumflex coronary artery with drug-eluting stent implantation 6 months earlier. He complained of chills, spiking fevers up to 41ºC with dizziness, malaise, and weakness for 1 month despite use of broad-spectrum antibotics. So far, no potential inflammation site has been identified in diagnostic work-up.

On physical examination, the patient was pale and cachectic. No heart murmurs or signs of peripheral microvascular thrombosis – Janeway lesions or Osler nodes – were present. Blood tests revealed elevated inflammatory markers (C-reactive protein, 236 mg/l; procalcitonin, 33 ng/ml; leukocytosis, 11,400/mm³ with neutrophilia; and normocytic hypochromic anemia with hemoglobin levels of 9.7 g/dl). Transesophageal echocardiography showed multiple large vegetations attached to the ventricular lead (FIGURE 1A), the largest of 21 × 13 mm with adjacent multiple echolucent spaces at the base, resembling medusa (FIGURE 1B) and suggesting an abscess. No patent foramen ovale (PFO) was observed. Empiric broad-spectrum antibiotics (vancomycin and rifampicin) were administered just after blood cultures were drawn, but the patient remained febrile (up to 41ºC) with shaking chills and sweats.

On the fifth day of hospitalization, transvenous lead extraction (TLE) was performed under general anesthesia with cardiosurgical back-up. An electrode with vegetation was removed using a lead extraction system (Cook Medical Inc., United States) with 2 large-size (white) telescopic polypropylene Byrd’s dilators (FIGURE 1C), wide enough to pull out the largest, fibrous part of the vegetation through the lumen (FIGURE 1D). Lead manipulation resulted in releasing peri-lead abscess content causing transient hypotension (40/0 mmHg) and desaturation suggestive of pulmonary embolization with infective material. The vegetation culture was positive for Staphylococcus hominis. There was right-sided pneumonia following TLE. The patient gradually improved and was discharged after 1 month of antibiotic therapy in a good clinical condition.

TLE is associated with a significant risk, including pulmonary1,2 and paradoxical embolism.3 In contrast to a more detailed approach in left-sided endocarditis, echocardiographic criteria for risk assessment in peri-TLE embolization are less well-defined by the current guidelines.4 This is the first report of a peri-lead abscess with subsequent peri-interventional embolization to have demonstrated that, in addition to size, morphological features shown on transesophageal echocardiography (TEE) (in particular, nonuniform vegetation texture with echolucent appearance) indicate higher peri-TLE risk.

In this context, preprocedural diagnosis of PFO is mandatory to be aware of potential paradoxical embolism.5
Based on our case, we conclude that in selected patients with the suspicion of peri-lead abscess, TLE can be performed under general anesthesia with cardiosurgical back-up and the awareness of higher risk related to embolization. Preprocedural TEE for risk assessment should include vegetation size, morphology, and mobility as well as the diagnosis of PFO.

REFERENCES


