Case Report: Non-infective Endocarditis (NIE) vs Culture Negative Endocarditis (CNE) Leading to Severe Mitral Regurgitation and Bioprosthetic Mitral Valve Replacement

Matthew McCurdy, DO, MS; Saleem Sameer, DO; Richard Pham, DO; Paul Moore, MD; Muhammed-Abdul Waheed, MD; Jacqueline Dawson, MD; Aniruddha Singh, MD


Introduction
Infective endocarditis (IE) occurs annually at ~3.3 cases per 100,000 in the U.S., with a gradual rise in the incidence over the last decade (case-fatality ~21-35%) [1]. Ordinarily, IE occurs in the setting of aberrant cardiac anatomy that is “colonized” by microorganisms. The contributing organisms are identified in ~93-98% of cases, with the remaining cases referred to as culture negative endocarditis (CNE) [2,3]. Non-infective endocarditis (NIE) represents a range of rare disease that is often only seen postmortem. Most cases of NIE are asymptomatic unless systemic embolization occurs (~50% of NIE patients) [4,5]. Sadly, the most common risk factor for NIE is advanced malignancy, especially adenocarcinoma (~80% of cases). Other potential risk factors include chronic wasting diseases, DIC, antiphospholipid syndrome, and autoimmune disorders (most commonly SLE).

Additionally, NIE most commonly involves the mitral and aortic valves (MV, AV), while IE generally involves the tricuspid valve (TV) and less frequently the MV or AV [3,5]. Here we discuss a case of suspected NIE with its associated clinical presentation and management.

Case Report
Mr. BL is a 65-year-old male with PMH of essential HTN, HLD, non-obstructive CAD, paroxysmal a-fib (on Eliquis), COPD (2L O2 continuous), prostate CA (s/p radiation/prostatectomy), and former tobacco use (90 pack year with 2-year abstinence). Patient presented with a 3-day history of intermittent “severe chest pressure” along the left sternal border that had acutely worsened over the past 1-2 hours. No provoking features, including exertion. No improvement with ASA and SL Nitroglycerin x3. Other symptoms included mild dyspnea with diaphoresis, with remainder of ROS unremarkable. Similar history of chest pressure 3 years prior, for which LHC was performed (non-obstructive CAD). No history of EoH or illicit substance abuse. Of note, patient recently seen by PCP for dyspnea (dx of mild COPD exacerbation) and prescribed oral prednisone without antibiotics.

At the time of evaluation, patient appeared moderately uncomfortable with diaphoresis. Vital signs: temp 98.7 F, Pulse 114, Resp 26, BP 123/99, SPO2 99% on 2L NC. Physical exam notable for tachycardia with RR, no MRG, no JVD, no extremity edema, diminished breath sounds, and anxious affect. Selected laboratory results include: leukocytosis (15.2 k/ul, 94.8% seg neutrophil and other GDMT for CAD. (TG 313 mg/dL, LDL 39, HDL 56).

In conclusion, NIE is uncommon and refers to the development of sterile thrombi on endocardial structures, as a possible response to trauma, circulating immune complexes, or hypercoagulable states [2,5]. Delineation of NIE from CNE is challenging and requires a systematic approach to rule out confounders. The main confounders of CNE are antibiotic administration preceding cultures or difficult to culture pathogens. As standard cultures and serologic workup are negative in NIE, histologic examination of removed valve tissue may be performed for diagnosis [5]. Delineation of NIE from CNE is challenging and requires a systematic approach to rule out confounders. The main confounders of CNE are antibiotic administration preceding cultures or difficult to culture pathogens. As standard cultures and serologic workup are negative in NIE, histologic examination of removed valve tissue may be performed for diagnosis [5].

References

Figure 1: (A) Apical 4C View (TTE) showing MV vegetation, (B) Corresponding color-doppler showing MR (TEE), (C) TEE showing 1.7 cm vegetation, (D) TEE showing severe MR

Figure 2: (A) Intraoperative view of MV vegetation from atra, (B) MV before being sent to pathology.