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Corynebacterium diphtheriae as a very rare cause of infective endocarditis

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While toxigenic Corynebacterium diphteriae causing diphteria has been virtually eliminated due to mandatory vaccinations, non-toxigenic strains are increasingly recognized as opportunistic pathogens responsible for invasive infections, including bacteremia and infective endocarditis (IE). The incidence of such infections has increased since the 1980s [1], but it remains exceedingly rare cause of IE- a serious and potentially fatal disease with an estimated

incidence of 3–10 cases per 100 000 people annually. Increasing antibiotic resistance among corynebacteria further complicates therapeutic management [2].

We report the case of a 40-year-old male patient with a history of alcohol abuse but no other significant comorbidities, who presented with two-week history of fever up to 40°C, hypotension, generalized weakness, abdominal pain, and rigors lasting two weeks. Physical examination revealed blood pressure 80/50 mmHg without compensatory tachycardia, tenderness in the left lower abdomen, and trophic changes of the left lower extremity with an old ulcer. Laboratory investigations demonstrated markedly elevated inflammatory markers: CRP 1466 nmol/L (reference range 0–47.6), PCT 2.37 ng/mL (reference range <0.046), leukocyte count 8.85×10^9/L (reference range 4.30–9.64×10^9/L) with neutrophilic predominance (Figure 1A), ALT 70 U/L (reference range 0-50), AST 63 U/L (reference range 0–50), mild microcytic anemia, and thrombocytopenia 63×10^3/L (reference range 163–347×10^3/L). Computed tomography revealed splenomegaly with moderately extensive infarctions. Empirical antimicrobial therapy with intravenous piperacillin/tazobactam (4.5 g three times daily) was initiated. Four sets of blood cultures grew Corynebacterium diphtheriae demonstrating susceptibility to benzylpenicillin, cefotaxime, ciprofloxacin, clindamycin, erythromycin, linezolid, meropenem, and trimethoprim/sulfamethoxazole. Later confirmatory testing at the National Institute of Public Health – National Institute of Hygiene identify a non-toxigenic strain. Wound culture from the lower extremity ulcer isolated Streptococcus pyogenes. Transthoracic echocardiography demonstrated large vegetations (up to 10–11mm) on both the aortic and mitral valves with severe aortic and moderate mitral regurgitation. Antimicrobial therapy was subsequently modified to benzylpenicillin (6 g four times daily) combined with gentamicin (320 mg once daily). Piperacillin/tazobactam was continued in order to ensure adequate initial coverage of likely nosocomial respiratory pathogens and was discontinued at the earliest opportunity following initial clinical improvement in pneumonia-related signs. Several hours following blood

cultures collection and transthoracic echocardiography, the patient developed acute-onset dysarthria and left-sided hemiparesis. Computed tomography confirmed acute ischemic stroke and mechanical thrombectomy was performed. Subsequent imaging demonstrated hemorrhagic transformation with associating cerebral edema (Figure 1B), necessitating discontinuation of antiplatelet therapy and initiation of osmotic therapy with mannitol and furosemide. Cardiac surgical intervention was contraindicated due to intracranial hemorrhagic transformation. Dental evaluation identified a potential infectious source and twelve teeth were extracted. Following respiratory deterioration ,after consultation with microbiologist, linezolid (600 mg intravenously twice daily) was added to the regimen. After transient clinical improvement, the patient's condition deteriorated, requiring transfer to the intensive care unit. Transesophageal echocardiography revealed extensive valvular destruction, vegetations measuring up to 11x6.3 mm involving all aortic cusps (Figure 1C and 1D), a 15x7 mm abscess cavity at the junction of the left and right coronary sinuses of Valsalva communicating with the right sinus (Figure 1E) and an additional 8 mm vegetation on the anterior mitral leaflet causing significant regurgitation (Figure 1D). Despite intensive multidisciplinary management, progressive multi-organ failure developed, culminating in patient's death.

Despite widespread immunization, non-toxigenic Corynebacterium diphtheriae remains a rare but increasingly recognized causative agent of IE [3]. Most reported cases involve patients with predisposing risk factors, including pre-existing structural heart disease, prosthetic heart valves, alcohol use disorder, chronic cutaneous lesions, homelessness, intravenous drug use, or inadequate sanitary conditions; however, cases have also been documented in patients lacking traditional risk factors. Cutaneous colonization and chronic skin wounds are considered significant portals of entry facilitating bacteremia. Given the rarity of C. diphtheriae IE, no standardized therapeutic guidelines exist [4], and management relies predominantly on individual case reports and limited case series. [5]. Despite appropriate antimicrobial therapy,

the infection may pursue an aggressive clinical course characterized by severe embolic and neurological complications, as well as extension to adjacent cardiac structures.

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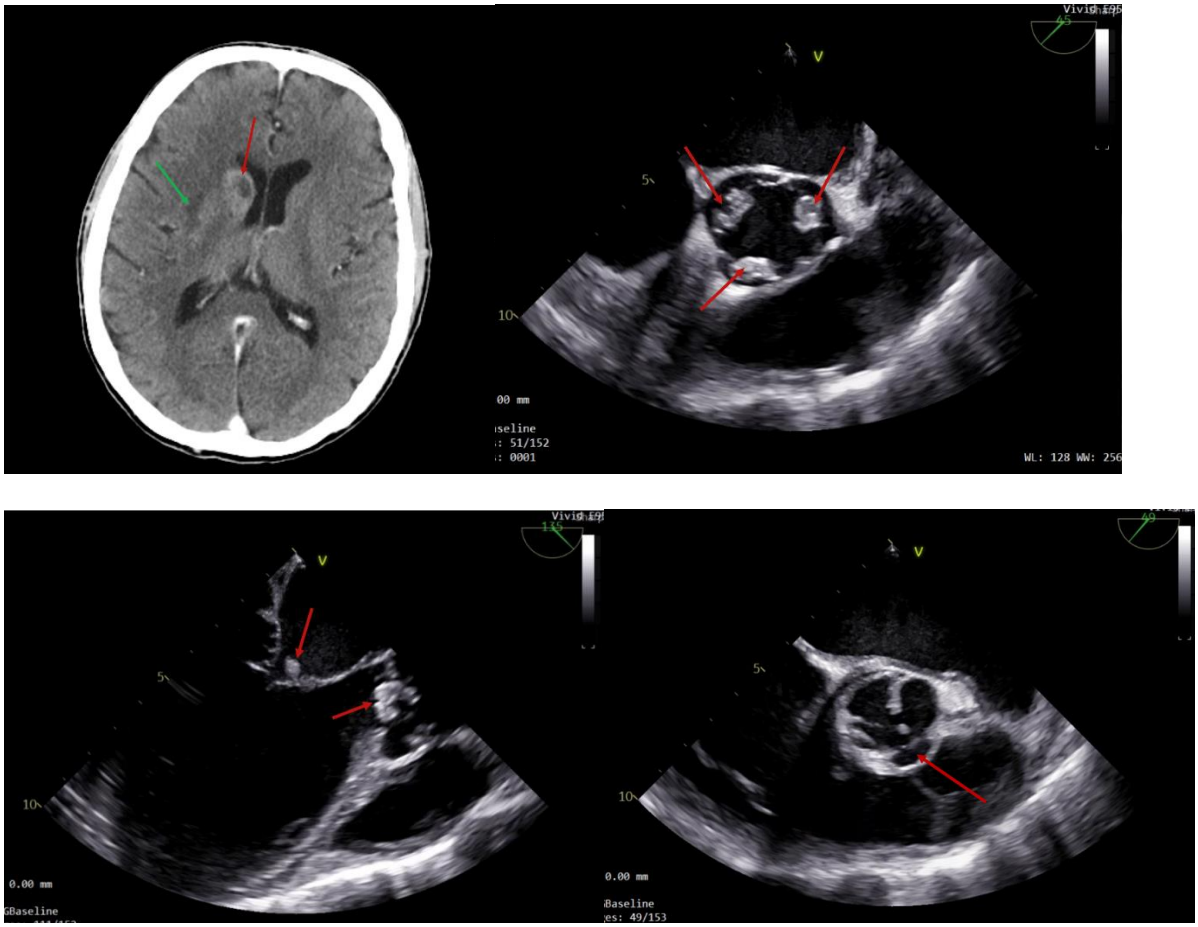

Figure 1 A – Contrast-enhanced computed tomography scan of the head- edema (red arrow) and scar tissue (green arrow); **B** –vegetations on the aortic valve mid-esophageal short-axis view, transesophageal echocardiogram (TEE) (red arrows); **C** – vegetations on the aortic valve and the anterior mitral leaflet mid-esophageal long-axis view, TEE (red arrows); **D** – cavity after drainage of the abscess mid-esophageal short-axis view, TEE (red arrow)

Short title: Corynebacterium diphtheriae-a rare cause of endocarditis