

# Acute Myocardial Infarction after Dog Bite: A Case Report

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## Abstract

### Key words:

Myocardial infarction,  
Dog bite,  
Capnocytophaga canimorsus.

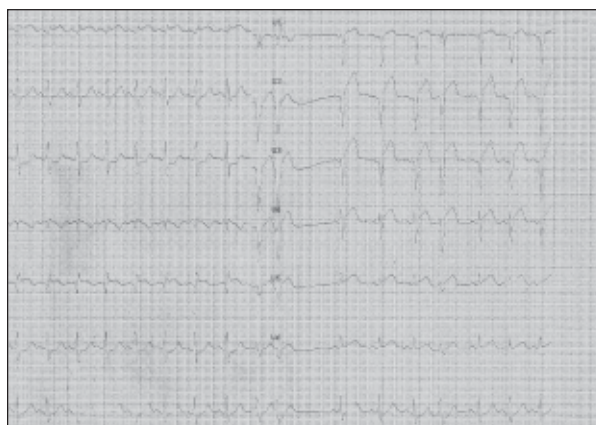
Acute myocardial infarction (AMI) after dog bite is rare. Capnocytophaga canimorsus, a Gram-negative, commensal organism of canine buccal cavity may cause bacteremia and sepsis. Hypotension-induced stasis, septic emboli to the coronary artery, endothelial dysfunction and plaque vulnerability induced by acute inflammation may lead to acute coronary syndrome in this situation. Isolation of the bacteria may be challenging due to fastidious nature of the organism. Management is as per standard protocol. However, appropriate antibiotic therapy is crucial. Like many other parts of the world, dog bite is endemic in Bangladesh. The case presented here reminds us of the need for preparedness to deal with myocardial infarction associated with dog bite effectively.

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## Case History

Mr. MU, a 40-year-old normotensive, non-diabetic but dyslipidaemic smoker was admitted into the NICVD with central compressive chest pain and sweating for 3 hours. He had history of dog bite in the lower limb 5 days back. On examination, he was found apprehended, his pulse was 80 beats per minute, blood pressure 130/85 mm of Hg, temperature 98<sup>0</sup>F, and respiratory rate was 18/min. The apex beat was in the left 5<sup>th</sup> intercostal space, no left parasternal heave, palpable P<sub>2</sub> and thrill. The first heart sound (S<sub>1</sub>) was rather soft, without any added sound. Trachea was normal position. Breath sound was vesicular with no added sounds. Examination of other systems revealed normal findings. He received ciprofloxacin, tetanus toxoid and anti-rabies vaccine, the schedule of the later was completed after admission into the NICVD.

The ECG of the patient revealed ST segment elevation in leads V<sub>1</sub> – V<sub>6</sub>, I and aVL (Figure 1a) and resolution of ST segment after thrombolytic therapy (Figure 1b). CK-MB was 95 units/L, cardiac troponin I was positive (17 ng/ml). Echo revealed mild anterolateral hypokinesia with left ventricular EF 40% (Figure 2a & 2b). Blood biochemistry including blood sugar, serum creatinine and serum electrolytes were normal. He was diagnosed as a case of acute anterior myocardial infarction with history of dog bite. The patient was managed as per standard protocol with inhalational oxygen, aspirin, clopidogrel, metoprolol and oral nitrate. Streptokinase 1.5 MU was infused for thrombolysis. Further investigation revealed normal total and differential leucocyte counts, hemoglobin and ESR. Microbiological



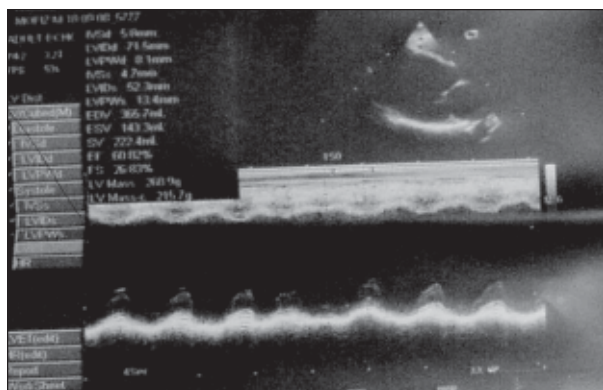
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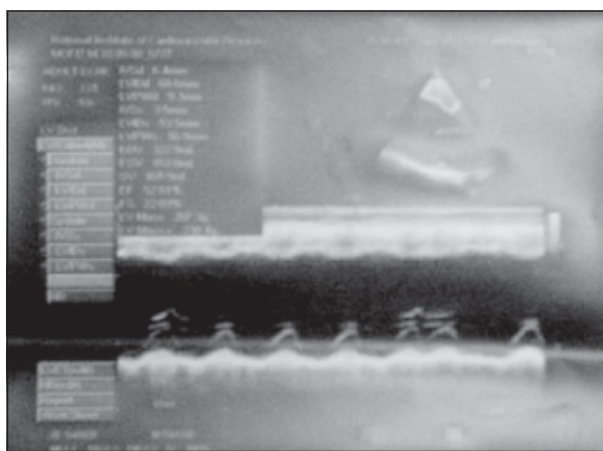
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Fig.-1. a. ECG of the patient a. before thrombolytic therapy b. after thrombolytic therapy.

examination did not show any microorganism in the peripheral blood film, also blood culture was negative. Coronary angiography, 5 days after admission, demonstrated insignificant lesion in the



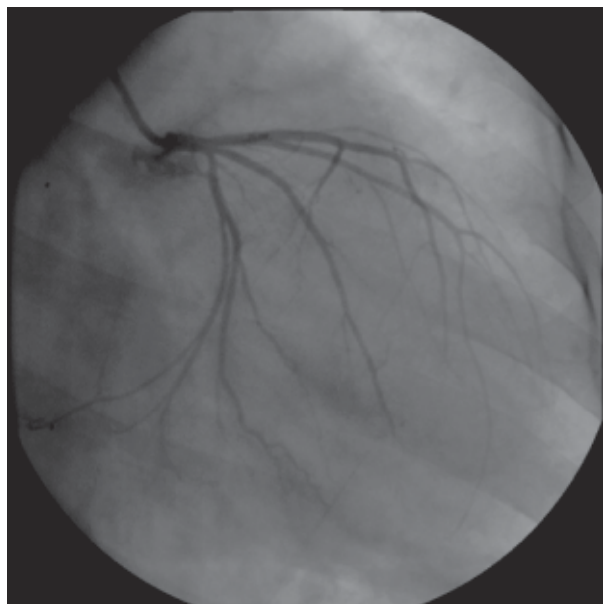
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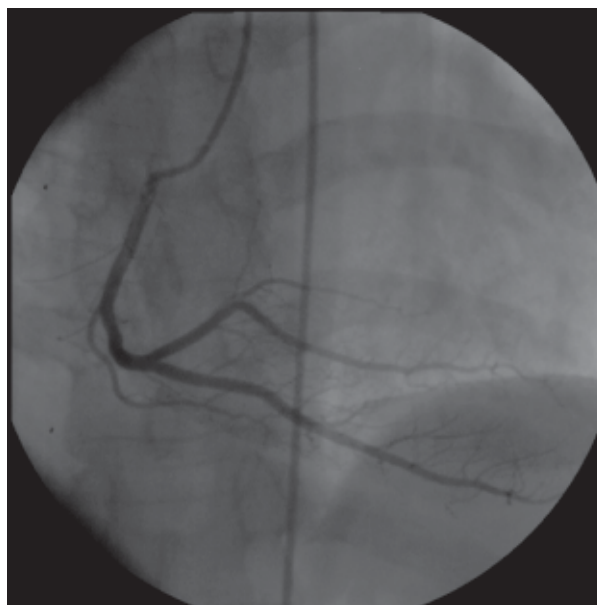
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**Fig.-2a &b:** Echocardiography of the patient showing left ventricular anterior wall hypokinesia.

ramus, otherwise normal epicardial coronaries (Figure 3a & 3b).



**Fig.-3 (a) :** Coronary angiogram of the patient showing normal left coronary system.



b

**Fig.-3 (b) :** Coronary angiogram of the patient showing normal right coronary system.

**Discussion**

Dog bite has been associated with acute myocardial infarction (AMI).<sup>1,2,3</sup> A number of commensal organisms are known to reside within the buccal cavity of dog, *Capnocytophaga canimorsus* is one of them. *C. canimorsus*, formerly known as dysgonic fermenter 2 (DF 2), is a fastidious, Gram-negative rod.<sup>4</sup> It causes fulminant sepsis following dog and cat bites, particularly in asplenic patients and alcoholics.<sup>5</sup> Infection and bacteremia may precipitate an AMI<sup>1</sup>, which usually results from hypotension or from endocarditis with septic emboli to the coronary arteries<sup>2</sup>. Alternatively, inflammation may lead to elevation of acute inflammatory proteins like CRP and amyloid A, as well as, to activation of monocytes and adhesion molecules. All these factors may lead to endothelial dysfunction and make the atherosclerotic plaque more vulnerable to acute coronary syndrome.<sup>6,7,8</sup> *C. canimorsus* sometimes cause fulminant myopericarditis which may mimic AMI.<sup>9</sup> In case of *C. canimorsus* infection, blood counts are usually indicative of acute inflammation, Gram-negative rods may be found in PBF and blood culture may yield specific organism. In our case, signs of acute inflammation were almost absent and no organism was found in PBF and blood culture. This may be due to antibiotic therapy after dog bite. *C. canimorsus* has been reported to be susceptible to

penicillin, third-generation cephalosporins, quinolones, doxycycline, erythromycin, rifampicin, vancomycin and imipenem in vitro.<sup>10</sup> Besides this, blood culture in case of *C. canimorsus* infection may be missed or incorrectly identified unless specific laboratory methods are followed e.g. prolonged subculture in CO<sub>2</sub> incubation.<sup>11</sup>

Another possibility is that the patient might actually presented with acute myopericarditis, rather than acute myocardial infarction. Acute myopericarditis can cause chest pain, ST elevation in ECG and elevation of cardiac biomarkers including CK-MB and troponin I.<sup>9,11,12</sup> Presence of near-normal epicardial coronary arteries in coronary angiography was an expected findings in that case. However, rapid regression of ST-elevation after streptokinase infusion favours the diagnosis of MI.

Dog bite is an endemic in many parts of the world including Bangladesh. It may be a rare cause of AMI. The clinicians should have appropriate knowledge and preparedness to deal with such an uncommon condition.

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