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Care of Patients With Cardiovascular Complications

Inflammation-induced atrial fibrillation: Pathophysiological perspectives and clinical implications

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ABSTRACT

Although atrial fibrillation (AF) is the most common type of cardiac arrhythmia, its etiology is unknown in nearly 10% of cases. Growing evidence suggests that inflammation plays a significant role in the onset and recurrence of AF. The role of inflammation in the pathogenesis of AF has important clinical implications of which many practitioners are unfamiliar. In this article, we describe a case of a 29-year-old male, who presents the emergency department with inflammation-induced AF, secondary to acute appendicitis. The latter condition was initially missed due to the unclear link between both presentations. By the time the AF was pharmacologically managed, the inflamed appendix perforated, resulting in unnecessary pain and suffering. After the perforated appendix was drained and removed surgically, the patient recovered well, and a follow up echocardiogram was normal. Here we give a brief overview of the pathophysiological perspective linking AF to inflammation and subsequent clinical considerations in patient management.

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Introduction

Atrial fibrillation (AF) is the most common arrhythmia in the United States, with a lifetime risk of approximately 25%. Nearly 6 million Americans are affected, and incidence rates are higher in whites, males, and older individuals.[1] AF increases the risk for ischemic stroke, dementia, and heart failure by a factor of 2–5. This accounts for more than 100,000 deaths per year in the United States alone, and the incremental annual cost of AF is estimated to exceed $25 billion.[1]

The fundamental mechanisms that govern the pathogenesis of AF are still poorly understood; therefore, available treatments are suboptimal.[2] The onset and recurrence of AF vary depending on the etiology. Common risk factors for AF include diabetes, hypertension, coronary artery disease, valvular defects, heart failure, hyperthyroidism, and alcohol intoxication. These modifiable risk factors explain 90% of all AF cases, but the etiology of the remaining cases (10%) remains unknown.[3,4] There is growing evidence that inflammatory processes may contribute to atrial injury by modifying the electrical activity of cardiac myocyte and, therefore, precipitating AF.[5] This suggested involvement of inflammation with AF is important, given that oxidative stress is a common pathology in many comorbidities (e.g., diabetes) that can serve as initiating factors in the development of AF. Importantly, severe bacterial infections are well known events that initiate and maintain a systematic inflammatory process, which can serve as an initiating factor in the onset of AF in some patients.[6]

Case presentation

A 29-year-old white male was brought to the emergency department (ED). He had been suffering excessive vomiting and diarrhea for 12 h. A sudden episode of palpitation and weakness was observed just prior to seeking medical attention. Upon first medical contact, the patient was alert and oriented but very exhausted. His blood pressure was 90/50 mm Hg, his heart rate was irregular at 152 beats per minute, and his temperature was 37.9 °C. The patient believed he was experiencing food poisoning due to consuming some non-refrigerated leftovers the day before.

Initial complete blood count and blood chemistry values were non-significant and revealed a slightly elevated white blood count (Table 1, Day 1). A 12-lead electrocardiogram (ECG) exhibited persistent AF (Fig. 1A), but the patient reported having no history of cardiac disease or any other past medical or surgical history. The patient was admitted to the telemetry unit for rehydration and was treated with intravenous Diltiazem and Heparin drips to control the AF. In addition, the patient was medicated as needed with intravenous morphine for mild to moderate, non-specific abdominal pain that was believed to be attributed to the gastrointestinal upset.

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Table 1
Blood workup values during hospitalization.

Blood test	First hospitalization		Second hospitalization		
	Day 1 (admission)	Day 3 (discharge)	Day 6 (admission)	Day 11 (discharge)	Normal reference
CBC/Differential					
White blood cell count	**H 12.8**	**H 13.9**	**H 18.6**	**H 13.7**	3.8–10.6 K/ul
Red blood cell count	5.5	5.2	5.8	5.3	4.2–5.6 M/ul
Hemoglobin	14.1	13.3	15.0	13.6	12.9–16.7 g/dL
Hematocrit	43.2	41.0	45.1	41.4	37.0–46.0%
Platelet count	L 130	L 132	259	206	150–450 K/ul
Neutrophils	**H 92%**	**H 80%**	**H 77%**	71%	35–75%
Lymphocyte	L 6%	L 13%	L 14%	21%	18–44%
Monocyte	L 2%	6%	9%	5%	0–10%
Chemistry					
Sodium	141	141	138	140	135–145 mmol/L
Potassium	3.6	3.6	3.6	4.1	3.5–5.1 mmol/L
Chloride	109	111	103	107	98–107 mmol/L
Blood urea nitrogen	13	6	15	11	6–24 mg/dL
Creatinine	0.9	0.8	1.03	0.9	0.7–1.3 mg/dL
Bilirubin	0.7	–	0.7	–	0.1–1.0 mg/dL
Protein	7.5	–	H 8.3	–	6.7–8.2 g/dL
Albumin	4.5	–	3.9	–	3.4–5.0 g/dL
Calcium	9.4	8.6	10.0	9.1	8.5–10.1 mg/dL
Magnesium	1.8	L 1.6	–	–	1.7–2.2 mg/dL
AST	18	–	20	–	14–37 U/L
ALT	15	–	22	–	12–67 U/L
Alkaline phosphate	44	–	64	–	39–119 U/L
Amylase	67	–	64	–	23–85 U/L
Lipase	15	–	87	–	50–393 U/L
CK	73	–	–	–	38–120 ng/ml
CK-MB	1.1	–	–	–	0–3 ng/ml
Troponin I	Negative	–	–	–	Neg.
Endocrinology					
TSH	1.77	–	–	–	0.4–4.5 U/ml
Coagulation					
PT	14.0	–	14.7	–	11.7–15.3 s
PTT	25.0	H 72.1	30.7	–	22.7–35.6 s
INR	1.1	–	1.2	–	0.8–1.2
Urinalysis					
Specific gravity	1.009	–	–	–	1.002–1.1030
Protein	Negative	–	–	–	Neg.
Glucose	Negative	–	–	–	Neg.
Ketones	Negative	–	–	–	Neg.
Alcohol	Negative	–	–	–	Neg.
Opiates	Negative	–	–	–	Neg.
Benzodiazepines	Negative	–	–	–	Neg.
Culture	–	–	Negative	–	Neg.

Bold indicates abnormal finding.

The patient was also treated with intravenous metoclopramide to control his nausea and vomiting. No abdominal imaging or radiological studies were performed during this hospitalization. Two days later, the patient's heart rhythm spontaneously reverted to normal sinus (Fig. 1B). The patient could tolerate a soft diet, and he was discharged home and instructed to follow up with a cardiologist after a few days. The discharge workup was non-significant, with the exception of a slightly elevated white blood count (Table 1, Day 3).

At home, the patient continued to have mild abdominal tenderness that gradually worsened; 3 days later, he started to experience severe chills. The patient sought medical attention, and he was taken again to the ED. Medical examination revealed that the patient's abdomen was "board-like" and exhibited profound tenderness. Blood tests indicated a significantly elevated white blood count (Table 1, Day 6); an urgent abdominal computed tomography showed (1) extensive regional inflammatory changes and (2) a dilated, disrupted appendix, suggesting perforation and acute peritonitis. No signs of mesenteric ischemia were detected. The patient underwent an urgent laparotomy; the appendix was drained and removed, followed by intravenous antibiotic treatment with Piperacillin-Tazobactam and Metronidazole injections for 5 days. A surgical pathology culture obtained during surgery revealed a streptococcal beta hemolytic group F bacterial infection. Pre- and post-surgery 12-lead ECGs showed normal sinus rhythm (Fig. 2). The patient recovered without complication and was discharged home on oral Augmentin for 10 days. Blood workup upon discharge again revealed a slightly elevated white blood count (Table 1, Day 11). At follow up, 4 weeks after surgery, the patient was in reasonably good health, and repeat blood workup showed that the white blood count had returned to normal. Follow-up 12-lead Holter ECG and echocardiogram images were insignificant and revealed no recurrent episodes of AF or any structural heart abnormalities.

Discussion

Nearly 25% of all patients hospitalized with sepsis experience AF, of which one third are reported to be of new onset. The literature suggests that acute factors (e.g., organ dysfunction), rather than cardiovascular comorbid conditions, increase the risk of new onset AF during bacterial infections.[7] Therefore, mechanisms precipitating AF in these patients are believed to be different from those in the general patient population with AF. In this case study,

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