CardioCase of the Month

Heart Failure With Gouty Arthritis

Are nonsteroidal anti-inflammatory the answer?

By Anita W. Asgar, MD; and Jonathan Howlett, MD, FRCPC

CardioCase Presentation

Mr. C.P., 68, has a three-year history of heart failure. He has been hospitalised twice in the past two years. His medical history is notable for hypertension, ischemic heart disease with a remote myocardial infarction, heart failure with an ejection fraction of 31%, chronic obstructive lung disease, peripheral vascular disease, and gout. He has been well managed for the past six months on the following medications: digoxin, furosemide, an angiotensin-converting enzyme inhibitor, a calcium channel blocker, long-acting nitrates, a beta blocker, and acetylsalicylic acid. One week ago, he developed an acute attack of gout and was treated with a nonsteroidal anti-inflammatory drug. He now presents to your clinic with symptoms of increased shortness of breath, orthopnea, paroxysmal nocturnal dyspnea, and increased peripheral edema. A chest X-ray shows interstitial edema with small bilateral pleural effusions. The patient is admitted to hospital for further management.

Can you identify the cause of this patient’s deterioration? How might this have been avoided?

About the authors ...  
Dr. Asgar is a cardiology fellow at Dalhousie University, Halifax, Nova Scotia.

Dr. Howlett is an associate professor of medicine at Dalhousie University and medical director, Queen Elizabeth II Heart Function and Transplantation Clinic, Halifax, Nova Scotia.

What's Your CardioCase Diagnosis?
Overview of heart failure

Heart failure is a complex syndrome that has become increasingly prevalent. One per cent to 2% of the general population in developed countries has heart failure and the average age at diagnosis is 76. This syndrome has a major impact on longevity and quality of life for patients. The typical heart failure patient is taking an average of seven to eight different medications daily and the combined annual rate of death and re-hospitalisation is 58%.

This case illustrates some of the pitfalls in the management of heart failure.

What’s the best treatment?

Hyperuricemia and gout are often associated with other major disorders, such as hypertension and atherosclerosis. The mainstay of treatment for an acute attack of gouty arthritis is an anti-inflammatory, particularly a nonsteroidal anti-inflammatory drug (NSAID). These drugs accelerate the natural regression of acute attacks and reverse pain and disability within several days. Their use in patients with heart failure can, however, be problematic.

Patients with treated hypertension or heart failure have elevated levels of angiotensin II and norepinephrine, serving to increase the release of vasodilator prostaglandins from the kidney. NSAIDs inhibit this compensatory response and result in increased renal and systemic vascular resistance. This in turn results in deteriorating renal function, elevated blood pressure, and worsening of heart failure.

The major mechanism of worsening heart failure is an increase in afterload resulting from NSAID-induced systemic vasoconstriction, which can lead to a further reduction in cardiac output. Patients at greatest risk are usually hyponatremic at presentation. In these conditions, hyponatremia is a marker for advanced disease, which is associated with increased antidiuretic hormone, as well as angiotensin II and norepinephrine.

The use of NSAIDs was not associated with a first occurrence of heart failure, as illustrated in the Rotterdam Study of 7,277 patients over age 55. These drugs were, however, linked to worsening of pre-existing heart failure; adjusted relative risk of heart failure exacerbation with NSAID use was 9.9 [95% confidence interval (CI) 1.7 to 57]. A case-control study of elderly patients showed similar findings. Use of NSAIDs was associated with an odds ratio of 10.5 (95% CI, 2.5 to 44.9) admission for heart failure in patients with a history of heart disease. Cyclooxygenase (COX)-2 inhibitors do not appear to be any different, as COX-2 is found in high concentrations in endothelium as opposed to gastric epithelium.
Are there alternatives?

Alternatives to NSAIDs in the treatment of acute gouty arthritis include colchicine, intra-articular steroids, oral steroids, and adenocorticotropic hormone (ACTH).

Oral colchicine, at doses of 0.6 mg every eight to 12 hours, is useful if taken early. There may be significant gastrointestinal side effects, such as diarrhea; however, most patients are able to tolerate low doses of colchicine. If diarrhea does develop, it is important to watch for hypovolemia and hypokalemia.

Intra-articular steroids are useful for monoarticular attack once septic arthritis is ruled out and are preferable to oral prednisone. Oral prednisone (30 mg/day for one to two days and then in reduced doses for seven to 10 days) can also reduce inflammation, but may be associated with a rebound attack during tapering.

Finally, intramuscular ACTH, 40 U.S. Pharmacopeia (USP) units to 80 USP units, can be administered twice daily for two days and then once daily for several days. Such treatment has been reintroduced into clinical practice with fewer rebound attacks than previously thought.

Gout is a self-limited illness that is not uncommon in patients with heart failure. Treatment of the acute episode should focus on options that will not worsen cardiac function. Between attacks, allopurinol can be used to prevent recurrences.

References