Drug Information Office / Jordan University of Science and Technology

New prospective study finds that NSAIDs Linked to Higher Atrial Fibrillation Risk

Atrial fibrillation (AF) is the most common sustained rhythm disorder observed in clinical practice and predominantly associated with cardiovascular disorders such as coronary heart disease and hypertension. However, several classes of drugs may induce AF in patients without apparent heart disease or may precipitate the onset of AF in patients with preexisting heart disease.[1]

Recently, some studies suggested that non-steroidal anti-inflammatory drugs (NSAIDs) are associated with a higher risk of AF.[2]

Previously, a population based case-control study showed that use of non-aspirin NSAIDs was associated with an increased risk of atrial fibrillation or flutter. Compared with non-users, the association was strongest for new users (lowest for non-selective NSAIDs and highest for COX 2 inhibitors).[3]

A new prospective study finds that taking (NSAIDs) drugs appears to be associated with an increased risk for atrial fibrillation (AF).[2]

This prospective study involved 8423 participants from the Rotterdam Study, a well-known population-based prospective cohort study examining risk factors for disease in older adults. The mean age of the study population was 68.5 years, and most participants (58.6%) were women. Researchers obtained a resting (ECG) for participants at baseline and then followed participants with ECG assessments during follow-up visits.[2, 4]

The investigators gathered data on NSAID use from collaborating pharmacies and categorized participants into current users (14 or fewer days, 15 to 30 days, and more than 30 days), past users (stopped for 30 or fewer days, 31 to 180 days, or more than 180 days), and never users. Over a mean follow-up of 12.9 years, 857 participants developed AF. At the time of diagnosis, 261 had never used NSAIDs, 554 had used NSAIDs in the past, and 42 were currently using NSAIDs.[2, 4]

As for mechanisms linking NSAID use to AF, because these drugs inhibit COX enzymes expressed in the kidneys, such inhibition may lead to fluid retention and increased blood pressure. NSAIDs also increase end-diastolic and end-systolic dimension, and these changes could explain part of the association. Alternatively, because NSAIDs are used as anti-inflammatory drugs, the underlying inflammatory conditions and pain they treat may be associated with AF.[2, 4]

In conclusion, the use of NSAIDs is associated with an increased risk of AF. The underlying mechanism behind this association deserves further attention

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