Is Aspirin Part of Your Chest Pain Protocol?

While many aspects of current EMS practice are based on anecdotal data, the prehospital use of aspirin (ASA) in the treatment of chest pain and acute coronary syndromes (ACS) is based on considerable scientific evidence. In fact, the American Heart Association, in its Guidelines 2000 for Emergency Cardio Care lists ASA as a Class I intervention. Class I interventions are always acceptable, proven safe, and definitely useful.

ACS are a spectrum of clinical syndromes that include unstable angina, non-Q-wave myocardial infarction (MI), and Q-wave MI. Sudden death may occur with any of these syndromes. ACS is a result of atherothrombotic events within the coronary arteries. With these lesions, commonly called plaques, form on the inner surface of the vessel walls and may include the coronary arteries. The plaques contain lipid substances that may eventually rupture. As the plaques begin to rupture, the lipid core spills into the bloodstream, forming the affected coronary artery. Eventually, these plaques can rupture. Following this rupture, a third layer of platelets coats the surface of the ruptured plaque in a process called platelet adherence. This is followed by the recruitment of additional platelets to the injury site, in a process called platelet aggregation. When this occurs, thrombin, a specialized substance in the blood, causes the platelets to become cross-linked. Then, the coagulation system is activated and a clot (thrombus) forms at the site of the lesion. This clot may either partially or completely obstruct blood flow through the affected coronary artery. If the artery is partially obstructed to a degree enough to cause coronary ischemia, the resultant condition is called unstable angina. If the clot results in intermittent occlusion of the coronary artery, a non-Q-wave MI (also called a subendocardial MI) may develop. Complete occlusion of the coronary artery by the clot will eventually lead to a Q-wave MI (also called transmural MI).

ASA in Coronary Care

So, what does all of this have to do with ASA? Quite a bit, actually. ASA was developed in 1899 by German chemist Felix Hoffman in a treatment for his father's arthritis. The basic ingredients of aspirin, called salicylate, had long been known. In fact, it is said that the Fifth Century B.C., Hippocrates, often considered the "Father of Medicine," is said to have used ground willow bark to ease arthritis pain. Willow bark contains a salicylate known as salicin.

In addition to its anti-inflammatory effect, ASA, it is also found to inhibit platelet aggregation. ASA inhibits an enzyme called cyclooxygenase (COX), which in turn inhibits the formation of thromboxane A2, which is a potent platelet activator. Thus, ASA inhibits platelet activation and therefore inhibits platelet aggregation. ASA's antiplatelet actions have been known for years, and it is commonly used to help prevent strokes in patients who have had transient ischemic attacks and similar events. Likewise, daily ASA has been largely recommended for patients with unstable coronary artery disease. However, only in recent years has ASA been widely used early in the management of ACS. Because it is so effective, the AHA has started to promote the use of the mnemonic MONA for patients with suspected ACS: MONA stands for:

- Morphine IV (if pain not relieved with nitroglycerin)
- Oxygen at 4 L/min
- Nitroglycerin sublingual or spray
- Aspirin 160-325 mg by mouth
They further recommend using the memory aid “MONA greets all patients” for patients with suspected ACS.

The prompt administration of ASA to patients suffering ACS is essential for better patient outcomes and increased survival. A recent Israeli study examined 1,200 patients with documented acute myocardial infarction (AMI), as evidenced by ST segment elevation, who were treated with either recombinant tissue plasminogen activator (rtPA) or streptokinase. Of these, 364 patients either self-administered aspirin or had it administered to them in the ambulance, ED or CCU before thrombolytic therapy (the average duration to ASA dosing was 1.6 hours). These were compared with 836 patients who received aspirin within one hour after receiving thrombolytic therapy (the average duration to ASA dosing was 3.5 hours). Researchers found that the mortality rate was significantly lower for patients who received ASA early rather than later. The differences in mortality rates are compared as follows:

- 2.5% in early treatment group vs. 5.6% in late treatment group at seven days.
- 3.3% in early treatment group vs. 7.3% in late treatment group at 30 days.
- 5.0% in early treatment group vs. 10.6% in late treatment group at one year.

This study showed that prompt administration of aspirin, ideally in the prehospital setting, resulted in a significant reduction in mortality for patients suffering AMI compared with those who received ASA after thrombolytic therapy.

Underuse of Aspirin in ACS

Although ASA therapy is an essential part of emergency care for chest pain and ACS, several recent studies found that aspirin was underutilized in the prehospital treatment of chest pain and ACS. In one study, only 13% of patients took aspirin for chest pain before EMS’s arrival, while only 74.9% received ASA within 24 hours of hospital admission.

In another study, researchers found that only 19.9% of eligible patients in the Portland, OR, area received ASA prior to a paramedic educational program on ASA and only 61.3% received ASA after paramedics attended the ASA in-service.

Why then is ASA often left out of prehospital chest pain and ACS protocols? There are several possible explanations for this. First, ASA is a generic drug, and manufacturers have little interest in promoting the drug as they have with other drugs recommended in the most recent AHA guidelines. In addition, unfounded fear of gastric bleeding and GI upset have kept the drug from some EMS system formulaters. These fears, for the most part, are unfounded—especially for a single dose. Regardless, the evidence is clear that ASA can save lives and should be administered in the prehospital setting to patients with chest pain or ACS.

Paramedics are certainly capable of safely administering ASA in the prehospital setting to patients suspected of suffering ACS. The standard dose for ASA in ACS is 160–325 mg. Products that contain only ASA should be used in prehospital care. Other products, such as the popular pain reliever Excedrin, contain caffeine and acetaminophen in addition to ASA. ASA tablets can be swallowed whole or chewed and swallowed. If the patient has difficulty swallowing the ASA tablet, consider using children’s chewable aspirin (160 mg). One to two children’s tablets may be administered based upon local protocols. ASA should not be used in patients who have a known allergy to the drug or to the nonsteroidal anti-inflammatory class of medications.

Aspirin is truly a wonder drug and an inexpensive, yet effective, treatment for ACS. If it is not part of your system’s chest pain/ACS protocol, it should be.

References


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