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A left ventricular and left middle cerebral artery iatrogenic AGE following AICD placement

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Aims:

- Present the case study
- What are the signs and symptoms of iatragenic arterial gas embolism.
- Summary and Discussion.

Introduction: An iatrogenic air embolism is an uncommon phenomenon that can lead to catastrophic consequences. It is an uncommon complication of invasive vascular procedures. It occurs when air enters the vasculature from a procedure that violates either the venous or arterial circulation. When an air embolism enters the venous circulation, patients can develop hypotension, tachypnea, hypocapnia, pulmonary edema or cardiac arrest (1-3, 10). Whereas an arterial gas embolism can lead to confusion, lethargy, neurological deficits, cardiac arrhythmias/ischemia (1-3, 10). This case report discusses an iatrogenic air embolism within the left ventricle and left middle cerebral artery discovered 4 hours after placement of an automated implantable cardioverter defibrillator (AICD).

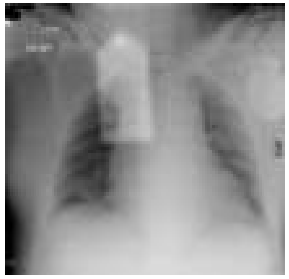
Case Report: A 55 year old male with past medical history was significat for coronary stent placement, coronary artery bypass graft, hypertension , dyslipidemia, chronic obstructive pulmonary. With a left ventricular dysfunction with an ejection fraction of 20-30% . Complained of right upper extremity weakness, shaking, and numbness after placement of a single chamber AICD at approximately 9:00am.

As the catheter was being introduced into the subclavian vein the patient took an inspitory gasp of air. The valve subsequently "fluttered," and the patient immediately desaturated. At the time patient was immediately put on 100% oxygen and symptoms quickly improved.

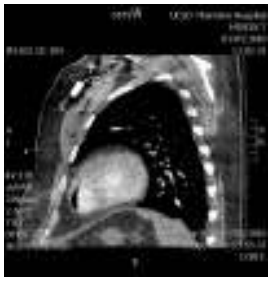
One hour after the procedure, the patient experienced numbness and tingling on his right hand and right foot. Neurology was consulted and a noncontrast CT scan of his brain was performed approximately an hour after the procedure; it was read as showing no pathology. An hour after the initial ct scan, the patient's had a worse pill-rolling tremor. A CT angiogram of head and chest four hours after presentation showed a "curvilinear density consistent with gas within two parietal branches of the left MCA (approximately 13:00)". The CT angiogram of the chest showed a similar density presumed to be gas in the nondependent portion of the left ventricle.

The patient arrived at HBO chamber approximately two hours after the initial call for HBO consultation. Upon arrival the patient was on his left side. He had his elbow flexed and was complaining of right sided upper arm weakness, shaking and numbness.

The patient was placed on 100% face mask and intravenous saline. Vital signs were withing normal limits and Glasgow coma score was 15. Patients' cranial nervers II-XII were intact. Patient had decreased sensation to light tough over the right hand and forearm. Patient was also hyperreflexive on the right pectoral/deltoid tendon/biceps and brachioradialis tendons (+3/5). Strength was also decreased with right shoulder abduction/adduction (+4/5), right elbow flexion/extension (+4/5), right wrist flexion and extension (+4/5). Gait was not assessed to minimize movement and decrease likelihood of dilodging gas from the left ventricle.

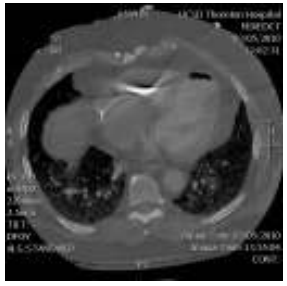


Portable chest x-ray



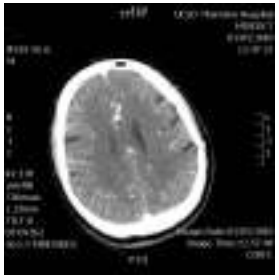
Sagital Chest CT

Gas seen in the left ventricle



Transverse Chest CT

Gas seen in the left ventricle



Transverse Head CT

curvilinear density consistent with gas within two parietal branches of the left MCA

Discussion: An iatrogenic air embolism can occur in all ages and during any invasive procedure. It does not discriminate between age, sex, height and weight of a person. Certain factors are needed for an air embolism to occur. First there has to be communication between an air source and the vasculature. Second there has to be a pressure gradient favoring passage of air into the circulation. Air can be pulled into an artery or vein by negative pressure. Air entering the venous system travels to the right heart and pulmonary circulation and may produce hypotension tachypenea, hypocapnia, pulmonary edema, cardiac arrest. An air embolism can enter the arterial circulation by several mechanisms: by direct introduction into an artery, a right to left intracardiac shunt (patent foramen ovale), an intrapulmonary shunt or incomplete filtration by the pulmonary capillaries. Air in the arterial circulation can produce subclinical symptoms it may also produce neurological deficits and cardiac ischemia/arrhythmia (1-3,10).

Prognosis varies widely and is based on how soon the patient can be treated with hyperbaric oxygen (HBO) at time of onset. The mortality rate for untreated air embolism is greater that 90%, but HBO therapy has reduced the rate to 7% with some having residual neurologic deficits (11).

The cardiologist recognized that possibility of air being introduced into the circulation. Recognition of the valve fluttering as the patient took a deep breath and the patient suddenly desaturating as the catheter was being introduced was an important sign that the patient quite possibly induced a high enough negative pressure gradient to force the valve to flutter.

After the procedure; the patient complained of symptomatic numbness and tingling in both the right upper and lower extremity. Neurology was consulted. Although the ct scan of the head has been shown to be highly sensitive for the detection of cerebral intraparenchymal gas (9), no apparent embolus was visualized. After the initial ct scan was performed, an echocardiogram was performed looking for possible air; considering that the patient experienced a sudden onset of cardiopulmonary decompensation (7,8).

The patients' numbness and tingling progressed to a pill-rolling tremor. Thus, a ct angiogram was performed and air was discovered in the left middle cerebral artery and left ventricle. The patient was immediately put on 100% nonrebreather mask, and kept at a left lateral position.

The patients' musculoskeletal strength subsequently improved to 5/5 in all extremities after his HBOT. The patient stated that his numbness and tingling improved after the HBOT.

The patient was reexamined and was immediately taken to radiology for a ct scan of his chest. Preliminary report was given by the radiologist, stated that no air was visualized in left ventricle.

Surprisingly, only a small amount of gas dislodged from the left ventricle and ascended into the neurovascular system. The patient's outcome would have been much different if the more of the air emboli progress into the neurovascular system. The patient would have presented with a more severe neurological manifestation such as alteration in consciousness, hemiparesis, hemianopsia, seizures, paresthesias, pupil asymmetry (4-6). It is postulated that due to the patient low ejection fraction (20-30%) the air with the ventricle did not progress into the cerebral vasculature. It's quite possible that the surface tension within the air bubble along with the hypodynamic left ventricle postponed the emboli from progressing.

This case is important not because of an iatrogenic air embolism due to an AICD placement, but because air was discovered within the hypokinetic left ventricle for a prolonged period of time. This case study serves as an example that not all late presenting neurologic complications are strokes or other manifestations other than an arterial gas embolism. This has an important application to dive medicine. The differential of and arterial gas embolism needs to be considered in divers as well as patients with low ejection fraction and delayed presentation.