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Atrial Fibrillation Post-Lung Lobectomy

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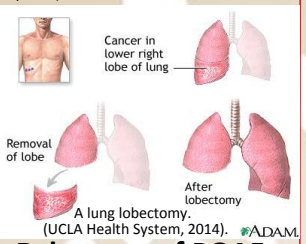
Introduction

According to the American Heart Association, atrial fibrillation affected 2.66 million individuals in the United States in 2010 and is expected to double by year 2050 (Sanoski, 2010, p. S11). Post-operative atrial fibrillation (POAF), a classification of atrial fibrillation (AF), can occur after thoracic surgery and is an ongoing source of morbidity and mortality (Imperatori et al., 2012). There is a 10-20% occurrence rate of POAF after non-cardiac surgery, such as a lung lobectomy, and a 40% incidence rate after a pneumonectomy (Omae & Kanmura, 2012, p. 429). Cardiovascular events such as heart failure, cerebral infarction, renal failure, and infection are significant complications associated with POAF (Omae & Kanmura, 2012, p. 429). Patients that develop POAF have a greater length of hospital stay, require more monitoring and care, and increased nursing support (Laidler & Rutherford, 2014, p. 274). The impact of POAF on patient outcomes has prompted zealous inquiries to seek the best measures for prevention and treatment of this complication after thoracic surgery (Omae & Kanmura, 2012, p. 429).



A lung lobectomy. (UCLA Health System, 2014).

Relevance of POAF

To optimize patient outcomes, it is imperative that nursing staff understand why post-operative lung lobectomy patients experience POAF, what puts them at risk for this dysrhythmia, and the pathogenesis of POAF. By learning about this potential complication, novice nurses will become more aware of clinical signs and factors to possibly prevent this arrhythmia and understand the rationale why specific treatments are used to manage POAF (Omae & Kanmura, 2012, p. 429). Overall, this new information will enable nurses to treat patients effectively and potentially decrease their morbidity, mortality, and hospital costs from POAF. In addition, nurses will be able to detect signs and symptoms of POAF early to prevent life-altering consequences to their patients (Sanoski, 2010, p. S11).

Case Study

A 76 year-old male, P.B., was admitted post right lower lobectomy for lung cancer associated with asbestos exposure and smoking. His past medical history includes hypertension and chronic obstructive pulmonary disease (COPD). On post-operative day (POD) one his vitals remained stable and his pain was managed adequately with intravenous opioids and oral medication. On telemetry, he remained in normal sinus rhythm. On POD two, his labs were within the normal range except his magnesium level was 1.3 mg/dL and was replaced intravenously. He had low urine output on night shift and was treated with 1 liter of normal saline. Later that afternoon, as he was getting out of bed, he became tachycardic, in the 140s, and appeared in atrial fibrillation. He had complaints of shortness of breath, dizziness, and felt his heart racing. An electrocardiogram was done and revealed atrial fibrillation. Why did his heart rhythm suddenly change into atrial fibrillation, status post a right lower lobectomy, when all along his rhythm was regular, his vital signs were stable, and his only cardiac history was hypertension?

Signs & Symptoms

Common Symptoms of POAF

- Shortness of breath, dizziness
- Heartbeat racing or palpitations
- Fatigue, generalized weakness
- Poor exercise intolerance
- Irregular pulse (Corell, 2012, p. 20)

Severe Signs and Symptoms of POAF

- Hypotension
- Chest pain or angina
- Decompensated heart failure

Irregular pulse, jugular venous pulsations, and variations in the strength of the first sound are clinical findings associated with POAF (January et al., 2014, p. 22). The initial examination of a patient with suspected POAF involves characterizing the pattern of the arrhythmia, such as acute-onset AF after surgery or paroxysmal form, lasting less than 7 days, or persistent atrial fibrillation which lasts longer than 7 days and both requiring termination by medications or electric cardioversion (Iwasaki, Nishida, Kato, & Nattel, 2011, p. 2264). To avoid further complications and patient compromise, practitioners must perform a rapid assessment, provide effective clinical management, and make a cardiology referral if necessary (Cotrell, 2012, p. 21).

POAF Pathophysiology

"Atrial fibrillation is defined as a tachy-arrhythmia characterized by predominately uncoordinated atrial activation with consequent deterioration of atrial function" (Contrell, 2012, p. 18). AF occurs when the electrical impulse is not initiated in the sinoatrial (SA) node and from a single re-entry circuit or by multiple re-entry circuits in the atria (Laidler & Rutherfold, 2014, p. 274). This fibrillating atrial activity does not adequately fill the ventricles and can decrease cardiac output by up to 20%, cause coronary artery ischemia, and possibly generate structural changes in the ventricles. The incomplete emptying of the atria leads to blood stasis and a possible clot formation, which is a major complication of AF. The presence of a clot in the left atria can increase the incidence of a cardio-embolic stroke and possible death (Nottingham, 2010, p. 281-282).

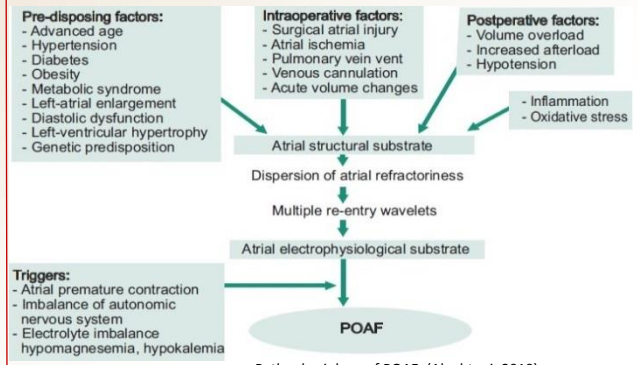
According to Chelazzi, Villa, and De Gaudio (2011), the electrophysiological mechanisms involved in POAF are not fully understood; however, the mechanisms are multifactorial (Laidler & Rutherford, 2014, p. 273). Researchers believe that patients who develop POAF may have already had an electrophysiological substrate for this arrhythmia before surgery. Mechanical manipulation of the heart and pericardium during thoracic surgery leads to local tissue trauma and an inflammatory response. Increased systemic sympathetic tone may result from this systemic inflammatory response in addition to the surgical trauma and pain. Circulating catecholamines may act on sensitized atrial myocardial cells causing a shortened refractory period and atrial reentry to produce POAF (Chelazzi, Villa, & De Gaudio, 2011). This local inflammation, and elevation in atrial pressure from post-operative ventricular straining may cause changes in atrial conduction, resulting in uncoordinated atrial activity (Contrell, 2012, p. 18). Inflammation from surgery may be associated with a rise in plasma concentrations of C-reactive protein. Both C-reactive protein and interleukin-6 are elevated in atrial fibrillation and may influence the development of POAF (January et al., 2014, p. 21)

The peak of sympathetic activation occurs within 24 hours post-operatively, thus the onset of POAF usually develops between 48 to 72 hours after thoracic surgery (Maesen, Nijs, Maessen, Allessie, & Schotten, 2012, p. 160).

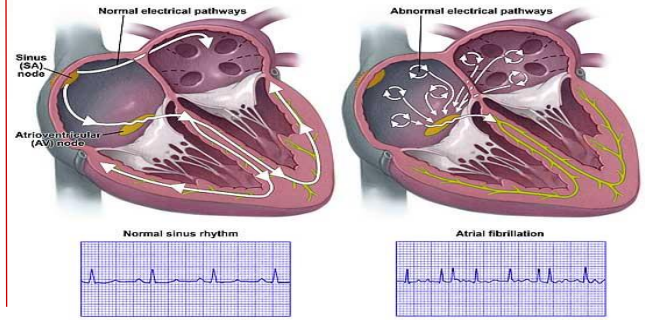
Pain alone, during the perioperative period, can trigger a sympathetic response which can create POAF. Pain increases sympathetic outflow and an imbalance between sympathetic and parasympathetic activity creates atrial ectopic beats and may cause POAF. Research indicates that adequate post-operative pain control is associated with a decreased occurrence of POAF. Thoracic epidural anesthesia (TEA) may reduce POAF because this produces a negative chronotropic effect on the heart, stronger coronary blood flow and myocardial oxygenation, and decreased sympathetic outflow from adequate pain control. Also, TEA can decrease myocardial sensitivity to circulating catecholomines, thus possibly decreasing the incidence of POAF (Chelazzi, Villa, & De Gaudio, 2011).

Hypovolemia, hypoxia, and anemia are also associated factors in eliciting new-onset POAF related to the increased sympathetic outflow. Hypovolemia and hypotension can occur from blood and fluid loss thus causing less venous return to the right atrium. This in turn decreases stroke volume and cardiac output and reduces tissue oxygen delivery. As a result, endogenous catecholamines are released and sympathetic hyperactivity occurs which may trigger POAF. In addition to hypovolemia, intraoperative hypoxia and anemia can occur which may cause ischemia to myocardial conduction tissue and atrial cells. This alteration can modify the cell's electric activities and cause supraventricular arrhythmias (Chelazzi, Villa, & De Gaudio, 2011).

Low levels of magnesium and potassium predispose changes in action potentials and the length of repolarization of the cardiac myocytes. Both conditions can induce POAF and are easily corrected by intravenous or oral supplements (Laidler & Rutherford, 2014, p. 275).



Pathophysiology of POAF. (Alqahtani, 2010).



Atrial Fibrillation. (Peninsula Heart Centre, 2014).

Significance of Pathophysiology

To provide effective treatment, it is imperative clinicians determine the underlying cause of the POAF, include associated cardiac and non-cardiac disease that predispose the individual to POAF, and assess the patient's thromboembolic risk (January et al., 2014, p. 22). By understanding what mechanism is causing the POAF such as surgical complications or acute precipitants, like pain or infection, practitioners can select the best intervention to correct this cardiac irregularity (Laidler & Rutherford, 2014, p. 275). There is a positive relationship between the mechanisms that trigger POAF and patients that have clinical risk factors that predispose them to POAF. Some of these clinical risk factors that cause atrial structural remodeling include the following: advancing age, structural heart disease such as hypertension or congestive heart failure, and left atrial enlargement. P.B.'s advanced age and his history of hypertension and COPD were all associated risks factors that may have predisposed him to develop POAF. (Maesen, Nijs, Maessen, Allessie, & Schotten, 2012, p. 168).

Aside from the pre-operative risk factors associated with POAF, this arrhythmia increases the risk of mortality and morbidity from stroke, heart failure, myocardial infarction, embolus formation, bleeding from anticoagulation, and hospital readmissions. Current research suggests that POAF can increase the length of the hospital stay (from 7 days versus 10 days for patients that experience POAF) and is related to a twofold increase in the occurrence of cerebral infarction (from 2.4% versus 5.3% in patients that have POAF) (Omae & Kanmura, 2012).

If the patient does not convert back into a normal rhythm within 48 hours, anticoagulation may be required due to the risk of developing a thromboembolic event. If POAF is left untreated, the incidence of experiencing a stroke is increased to five-fold (Laidler & Rutherford, 2014, p. 275). Clinicians should calculate the individual's risk of stroke to determine who is at greatest risk and will benefit from warfarin therapy versus those of who are at lowest risk and will benefit from aspirin therapy by using the $CHADS_2$ (Congestive heart failure, Hypertension, Age greater than 75, Diabetes mellitus, and prior Stroke or transient ischemic attack) score. (Nottingham, 2010, p. 283). To prevent further complications, it is imperative the ventricular rate be controlled and associated cardiac diseases be managed throughout one's post-operative course (Laidler & Rutherford, 2014, p. 275).

Nursing Implications

By clearly understanding the pathophysiology of POAF, clinical signs and symptoms related to this arrhythmia, and risks associated with this disease will enable nurses to remain calm and collective at the bedside if POAF should occur. This will enable them to provide competent, safe care while remaining supportive to the post-operative patient and family. In addition, this can lead to earlier detection of this arrhythmia and possibly prevent severe complications (Laidler & Rutherford, 2014, p. 275). Having a thorough understanding of the underlying disease process of POAF will encourage nurses to pay more attention to critical details such electrolyte abnormalities, signs of hypoxia, and post-operative pain, as these all can trigger POAF (Chelazzi, Villa, & De Gaudio, 2011).

An increased awareness of POAF by nurses will enable them to effectively educate their patients and families as to why this arrhythmia occurred and what to expect next during one's hospitalization. If anticoagulation is needed to prevent a stroke post-operatively, nurses will understand the implications of closely monitoring the patient for signs or symptoms of a stroke, clot, and maintain bleeding precautions (Laidler & Rutherford, 2014, p. 277).

Conclusion

P.B.'s advanced age, history of hypertension, COPD, smoking, and lung carcinoma put him at increased risk of possibly developing POAF (Cotrell, 2012, p. 20). His electrolyte levels and troponin were checked and all were within the normal range. He was given intravenous cardizem and within 24 hours converted back to NSR and made a stable recovery.

The mechanisms triggering and sustaining POAF are multifactorial and can be challenging for clinicians to manage. In order to effectively manage patients that develop POAF, it is imperative nurses have thorough assessment skills, adequately control patients' post-operative pain, pay close attention to patients' electrolyte levels, oxygenation and volume status, and predisposing risk factors associated with POAF (Chelazzi, Villa, & De Gaudio, 2011). Overall, this acquired knowledge of POAF will enable nurses to provide effective care to patients experiencing POAF by reducing their symptoms, optimizing their outcomes, and possibly reducing the financial burden on the health care system (Laidler & Rutherford, 2014).

References

Alqahtani, A. (2010). Atrial fibrillation post cardiac surgery trends toward management. [Photograph]. *Heart Views*. 11(2):57-63.

Chelazzi, C., Villa, G., & De Gaudio, A.R. (2011). Postoperative atrial fibrillation. *International Scholarly Research Network*, 2011, 1-10. doi: 10.5402/2011/203179

Cottrell, C. (2012). Atrial fibrillation part 1: pathophysiology. *Practice Nursing*, 23(1), 16-21.

Iwasaki, Y., Nishida K., Kato, T., & Nattel, S. (2011). Atrial fibrillation pathophysiology: Implications for management. *Circulation,* 11(124), 2264-2274. doi: 10.1161/circulationaha.111.019893.

Imperatori, A., Mariscalco, G., Riganti, G., Rotolo, N., Conti, V., & Dominioni, L. (2012). Atrial fibrillation after pulmonary lobectomy for lung cancer affects long-term survival in a prospective single-center study. *Journal of Cardiothoracic Surgery,* 7(4). doi:10.1186/1749-8090-7-4

January, C., Wann, L., Alpert, J., Calkins, H., Cleveland, J., Cigarroa, J., Conti, J., Ellinor, P., Ezekowitz, M., Field, M., Murray, K., Sacco, R., Stevenson, W., Tchou, P., Tracy, C.,& Yancy, C. (2014). 2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation.[published online ahead of print March 28, 2014]. *Circulation*. doi:10.1161/CIR.0000000000000041.

Laidler, S., & Rutherford, N. (2014). The nurse prescriber's role in management of postoperative atrial fibrillation. *British Journal Of Cardiac Nursing*, 9(6), 273-279.

Maesen, B., Nijs, J., Maessen, J., Allessie, M., & Schotten, U. (2012). Post-operative atrial fibrillation: A maze of mechanisms. *EP: Europace,* 14(2), 159-174. doi:10.1093/europace/eur208

Nottingham, F. (2010). Diagnosis and treatment of atrial fibrillation in the acute care setting. *Journal Of The American Academy Of Nurse Practitioners*, 22(6), 280-287. doi:10.1111/j.1745-7599.2010.00508

Omae, T., & Kanmura, Y. (2012). Management of postoperative atrial fibrillation. *Journal Of Anesthesia*, 26(3), 429-437. doi: 10.1007/s00540-012-1330-9

Peninsula Heart Centre. (2014) Atrial fibrillation [Photograph]. Retrieved from: http: phc.org.au/information/diseases/arrhythmias/atrial-fibrillation/

Sanoski, C. (2010). Prevalence, pathogenesis, and impact of atrial fibrillation. *American Journal Of Health-System Pharmacy*, 67(9), S11-6. doi:10.2146/ajhp100148

UCLA Health System (2014). Patient education: Lung cancer program at UCLA. [Photograph]. Retrieved from: http://lungcancer.ucla.edu/adm_therapy_pulmonary.html

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