Case Report

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Defecation Syncope: Two Cases of Post-Operative Cardiac Arrest

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Abstract

Introduction: The vasovagal response is a normal physiologic response that consists of sequential changes in blood pressure, heart rate, and contractility that resolves spontaneously in normal patients. This normal response can be problematic in patients with a compromised cardiovascular system who require surgery.

Case Summary: We report two cases of defecation syncope with cardiac arrest. Both patients had preexisting cardiovascular disease and no premonitory syncope symptoms. Both patients were returned to spontaneous circulation following initiation of ACLS protocol, but died later in the hospital course secondary to complications of cardiac arrest.

Conclusion: Promoting hydration, incorporating a stool softener into the medication regimen, and squatting during bowel movement may aid in reducing the risk for syncope and associated cardiac arrest with defecation.

Introduction

Mortality associated with syncopal events has been reported as early as the first century, A.D. Valerius Maximus of Rome wrote of the story of Coma, a bandit, who upon capture and interrogation, "covered his face, bent forward upon his knees and in the very sight of the highest power of the state through compression of his breath found the rest and security he desired" [1,2]. In the early half of the last century, modern medicine became familiar with the "notorious frequency of sudden and unexpected deaths of patients while using bed pans in hospitals", and related these deaths to physiologic changes that occurred during straining [2]. The first report on cardiac arrest associated with the Valsalva maneuver and defecation was published in 1968, and thereafter reports of defecation syncope appeared sparingly in the literature [3-5].

"Defecation syncope" describes the vasovagal response experienced while defecating that results in a loss of consciousness. The loss of consciousness results from bearing down to increase the pressure in the rectum. The increase in the pressure of the rectum calls for closure of the epiglottis, tightening of the diaphragm, tightening the muscles of the chest wall and stimulation of the parasympathetic nervous system.

The initial results of bearing down are an elevation in arterial pressure and bradycardia, followed by a rapid drop in blood pressure, and the subsequent decrease in blood flow to the brain. This is a normal response. However, in patients with a compromised cardiovascular system, the vasovagal response to defecation could put them at risk for an adverse cardiac event [6].

We describe our experience with two patients who suddenly went into cardiac arrest after having a bowel movement. We seek to provide a brief summary of the conditions leading up to their episode of cardiac failure.

Case Summary

We present two patients who suddenly went into cardiac arrest after having a bowel movement. The time required to restore to spontaneous blood flow was 15 minutes in patient 1 and 30 minutes in patient 2.

Our first patient is a 59 year-old woman with schizophrenia, dementia, COPD, congestive heart failure, and hypertension who was transferred from a nursing home and presented tachycardic with pain and swelling of her right arm. On physical exam, erythema, induration, blistering, and a shallow ulcer were noted over her right elbow, and laboratory studies revealed leukocytosis (with bandemia) and an elevated creatine kinase level. She was started on IV vancomycin and admitted with an initial diagnosis of cellulitis. On hospital day #1 after consultations with Cardiology, Infectious Diseases, and Plastic Surgery, and additional studies to include follow-up CBC with differential, x-rays, CT scan, EKG, and echocardiogram, her diagnosis was changed to paroxysmal ventricular tachycardia with marked ventricular arrhythmia, cardiomyopathy, and necrotizing fasciitis of the right upper extremity. Intravenous cefepime and clindamycin were added and she was taken emergently to the operating room for extensive debridement of the right upper extremity, carpal tunnel release, fasciotomies of the hand, of the forearm flexor and extensor compartments, brachial fasciotomy and arthrotomy of the right elbow joint.

Intraoperatively, tissue was debrided down to muscle fascia. The deeper muscle appeared to be viable and the majority of the muscle in the extensor and volar compartments of the forearm were preserved. The carpal tunnel and elbow joint were opened and aspirated, however no pus was encountered. The wound was then dressed with Betadine and saline-soaked Kerlix and she was transferred to the Surgical ICU in critical condition.

Post-operatively, the patient was started on vasopressin, levophed, and lidocaine drips. She was extubated on POD#1, weaned off pressors and started on a liquid diet on POD#2 and advanced to a soft diet on POD#3. She was transferred out of the SICU to a medical/surgical ward on POD#5. The patient was not on a stool softener, but had small bowel movements on POD#5 and 6. On POD#9, in the presence of nursing staff, the patient had a large bowel movement, immediately became anxious and irritable, and then was unresponsive. ACLS protocol was initiated and a return to spontaneous blood flow was attained after 15 minutes. The patient was transferred to the Medical ICU, where serial cardiac enzymes obtained were consistent with an acute myocardial infarction. She was extubated on POD#10 and remained in the ICU. On POD#18, the patient had three separate episodes of cardiac arrest with pulseless electrical activity and the decision was made by her family to withdraw care. No autopsy was performed.

Our second patient is a 64-year-old male with poorly controlled diabetes mellitus, peripheral vascular disease, and chronic osteomyelitis of the left calcaneus. The patient had undergone multiple debridements spanning several years, however he desired continued efforts toward limb salvage. Pre-operative cardiac evaluation included an echocardiogram with no vegetations visualized, normal left ventricular wall motion, and an ejection fraction of 60-65%, an EKG showing sinus rhythm with premature atrial complexes and a pharmacologic stress test interpreted as showing no increased perioperative risk of cardiac complication due to myocardial ischemia or left ventricular dysfunction. Pre-operative deep venous thrombosis screen was negative.

The patient was taken to the operating room for planned regional flap reconstruction. Based on intraoperative findings, a delayed medial plantar flap was created secondary to flap congestion. A 10x9cm split-thickness skin graft was harvested and placed within the midfoot and the medial plantar flap was inset over Xeroform gauze. The patient was awakened and transferred to the ICU without complication.

Post-operatively, he was placed on heparin, aspirin and a beta-blocker and had flap examinations every four hours. An oral diet was resumed on POD#1, and on POD#2, the patient was given a suppository for complaints of constipation. He was started on scheduled twice-daily stool softener on POD#3. On the morning of POD#4, the patient was supratherapeutic on heparin and the heparin drip rate was reduced according to protocol. Approximately three hours later, immediately following his first witnessed post-operative bowel movement, the patient complained of shortness of breath then became unresponsive—monitors showed asystole. ACLS protocol was initiated and the patient regained spontaneous circulation after 30 minutes, but did not regain consciousness. The patient was transferred to the Medical ICU with fixed and dilated pupils and a Glasgow Coma Scale score of 3. The post-arrest electrocardiogram showed marked ST abnormality, but cardiac enzymes were negative. Echocardiogram revealed borderline global hypokinesis of the left ventricle and reduced left ventricular systolic function with and ejection fraction of 45-50%. Electroencephalogram showed diffuse low voltage activity consistent with an anoxic event. CT scan of the brain revealed no intracranial hemorrhage, no extra-axial fluid collection, and no mass effect. On POD#10, withdrawal of care was instituted by the patient's family, and the patient expired. No autopsy was performed.

Discussion

The Valsalva maneuver can be divided into four phases: 1) the initiation of straining; 2) the period of continued straining; 3) the period immediately after release of straining; and 4) the subsequent period [3].

The normal circulatory response is also divided into four phases: 1) increased intrathoracic pressure with elevation of the systemic arterial pressure and slowing of the heart rate; 2) reduced venous return with sequential reduction and recovery of arterial pressure and reflexive tachycardia; 3) reduced intrathoracic pressure with further reduction of arterial pressure accompanied by persistent (and increased) tachycardia and 4) elevation of pressure and reflexive bradycardia [3,11,12].

Reports have shown high-grade atrioventricular block, nodal-type ectopic rhythms, other cardiac rhythm disturbances, decreased cerebral blood flow, and pulmonary embolism occurring in patients performing Valsalva [2,3,8]. The physiologic changes that occur may lead to a syncopal event, and in some cases to ventricular arrest [2,4]. Komatsu et al. reported that 65% of patients with defecation syncope had associated cardiovascular disease [7].

In our presented cases, the syncopal events were not preceded by premonitory defecation syncope symptoms, such as nausea, vomiting, abdominal cramps, or a strong urge to defecate with diarrhea. Although more common in patients with defecation syncope than in other forms of situational syncope (e.g. micturition syncope), these symptoms may be absent in nearly half of cases [7]. The cause of syncopal episodes in patients experiencing premonitory symptoms has not been well defined [5,7]. Both of the patients presented here have a history of cardiovascular disease and developed acute changes (i.e. irritability, anxiety, shortness of breath) immediately after defecation and immediately before cardiac arrest. Such symptoms have been reported with acute pulmonary embolism following defecation, however post-arrest studies in the presented cases do not support this diagnosis [9]. As reported by Kapoor et al. in over 50% of patients with defecation syncope, a clear etiology of the syncopal episode was not identified, however the disorder was associated with a high mortality [5].

The underlying commonality between these two patients is the series of physiologic events occurring with defecation that led to syncope and may have led to cardiac arrest. The exact mechanism of defecation syncope is not well understood, however, changes that occur during Valsalva are well known. Measures taken to reduce the physiologic response to Valsalva may thereby reduce the likelihood of an associated syncopal event.

Since vagal tone increases during the Valsalva maneuver, our recommendation is to avoid excessive effort during the bowel movement. Promoting hydration and incorporating a stool softener into the medication regimen may aid in achieving this goal [3,8]. Another risk reducing option is to squat over, instead of sitting on, a toilet seat while defecating. This technique has been shown to straighten the rectoanal anatomical angle, thereby reducing the expulsive effort required, and facilitating a safer bowel evacuation [6,10]. It is acknowledged that attaining the squatting position may be difficult for elderly or ill patients.

Conclusion

The condition of defecation syncope is associated with a high mortality, and in the majority of patients, an etiology cannot be identified. Both patients in this report had their surgical procedures performed without intraoperative complication, however in the days following surgery they had cardiac arrest during defecation. We recommend avoidance of straining during bowel movement by promoting hydration, incorporating stool softeners into the post-operative medication regimen, and when possible, squatting over the receptacle rather than sitting.

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