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Ruptured Aortic Aneurysm and Cardiac Arrest Associated with Spinal Anesthesia

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ALTHOUGH hemodynamic changes frequently occur during spinal anesthesia,[1] cardiac arrest during spinal anesthesia is extremely rare.[1-3] According to the closed claims analysis by Caplan *et al.*,[4] cardiac arrest during spinal anesthesia usually resulted from hypoxemia and hypercarbia associated with unappreciated respiratory insufficiency. Cardiac arrest during spinal anesthesia also has been reported without hypoxemia or obvious respiratory depression in patients with sick sinus syndrome or due to vasovagal response.[5-8] However, there are no previous reports of cardiac arrest and ruptured abdominal aortic aneurysm (RAAA) associated with spinal anesthesia. We report a case of cardiac arrest and RAAA during spinal anesthesia and discuss the pathophysiologic changes associated with cardiac arrest after spinal anesthesia in context with RAAA.

Case Report

A 65-yr-old, 77-kg man was scheduled for left inguinal hernia repair. He had presented with left groin pain and an 8-h history of nausea and vomiting. Medical history and physical examination were otherwise unremarkable. His preoperative laboratory results were normal except for a serum creatinine concentration of 2.1 mg/dl and a glucose concentration of 291 mg/dl. His hematocrit was 41%, his white blood count was 13,500/mm^3, and his platelet count was 289,000/mm^3. The preoperative electrocardiogram displayed left ventricular hypertrophy and was suggestive for anterior ischemia. However, the patient gave no history of angina, dyspnea, hypertension, or diabetes.

On arrival in the operating room, electrocardiograph, automated arterial blood pressure, and pulse oximetry measures were monitored. The arterial blood pressure was 130/85 mmHg, heart rate was 84 beats/min with sinus rhythm, and the room air hemoglobin oxygen saturation was 95%. After beginning a rapid infusion of crystalloid solutions, 2 mg midazolam was administered intravenously, and 3 l/min nasal oxygen supplementation was started. The patient was then turned to the left lateral decubitus position, and a subarachnoid block was induced with 11 mg hyperbaric tetracaine hyperbarically at the L_{2-3} interspace.

Approximately 2 min after the subarachnoid injection, while the patient was still in the left lateral decubitus position, he complained of abdominal and back pain. He was immediately placed in the supine position, at which time his arterial blood pressure was 168/116 mmHg, his heart rate 76 beats/min, and his sensory level at T_4. Within 2 min, he became agitated, vomited, and then became unresponsive. His heart rate abruptly decreased to 30 beats/min and was not responsive to 1 mg atropine intravenously. After suctioning the patient's airway, his lungs were ventilated *via* bag-valve-mask with difficulty. We administered 120 mg succinylcholine intravenously, and the trachea was intubated. Soon after intubation, ventricular fibrillation occurred, and chest compressions were initiated. After two doses of 1 mg epinephrine intravenously, defibrillation (200 J), and rapid crystalloid solution infusion, the patient responded with an increase in heart rate to 120 beats/min and a systolic blood pressure in the range of 70-80 mmHg. The working diagnosis for the cardiac arrest was circulatory failure secondary to a high spinal anesthesia.

An 8.5-French catheter was placed in the right femoral vein, and 1.5 l crystalloid solution was administered rapidly. His blood pressure increased to 140/80 mmHg. Once a pulse could be palpated, a 20 G was inserted in the right radial artery, and arterial blood gases were measured. At this time, the patient regained consciousness, became combative, and attempted to extubate his trachea. We administered 10 mg vecuronium with the careful titration of 4 mg lorazepam and 500 μg fentanyl intravenously for the next 10 min. The arterial blood gas results were: *p*H 7.30, $PaCO_2$ 31 mmHg, PaO_2 573 mmHg, base deficit 9.3 meq/l, and hematocrit 22%. Because the patient's hematocrit was significantly lower than his preoperative value, it was remeasured.

We considered transferring the patient to the intensive care unit, but decided to keep him in the operating room until the laboratory results were returned and further diagnostic studies were completed. During this period, the patient required intermittent boluses of crystalloid solution (200-300 ml) and dopamine infusion to maintain a

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systolic blood pressure of 100 mmHg. Because the second hematocrit was 20%, we requested the surgeons to perform a diagnostic peritoneal lavage, which was grossly positive for blood. Immediate exploratory laparotomy was performed, and a 9-cm ruptured infrarenal aortic aneurysm, which extended to both iliacs, was discovered. The patient underwent an aortoiliac bypass and a right groin exploration with distal embolectomy. The intraoperative course was otherwise uneventful. Postoperatively, the patient required hemodynamic support with dopamine and dobutamine infusions for 3 days and ventilator support for 7 days. On postoperative day 9, the patient returned to the operating room for an anterior gastrostomy and excision of a bleeding ulcer. The patient was discharged from the intensive care unit on postoperative day 14 and discharged home on postoperative day 19. At follow-up 1 yr later, the patient was functioning well.

Discussion

Rupture of an abdominal aneurysm is a catastrophic complication with a high mortality rate. Large aneurysms, as in this patient (9 cm), expand more rapidly and are more likely to rupture. An AAA of 6 cm poses a 50% risk of rupture within 5 yr, whereas one of 8 cm or more has a 75% risk.[9] The rupture of AAA may have occurred before or during the spinal anesthetic. It is possible that, in this patient, the incarcerated inguinal hernia masked the symptoms of a leaking AAA. Misdiagnosis of RAAA is a frequent occurrence in patients who seek treatment for a variety of symptoms that mimic other disease processes, including symptomatic inguinal hernia.[10-12] Recent widespread use of abdominal computed tomography scans have revealed presence of chronic contained RAAA in entirely asymptomatic patients.[13] The rupture may be contained and tamponade within the retroperitoneal space for a prolonged period. Investigators from our institution recently reported that they were unable to document a single patient during the 10-yr study who had an RAAA at operation that was not considered before surgery.[14]

Conversely, the rupture of the aneurysm may have occurred during the placement of the subarachnoid block. The increase in arterial blood pressure noted after the placement of subarachnoid block may have contributed to the rupture. The patients with AAA have degenerative changes in the structural matrix of the aortic wall and decreased vascular elasticity. Possibly, the combination of hypertension and a decrease in intraabdominal pressure (due to decreased abdominal muscle tone from spinal anesthesia) resulted in increased transluminal pressure sufficient to permit the aortic aneurysm to rupture. The complaint of abdominal and back pain after the subarachnoid injection may be suggestive of this mechanism. Finally, it is possible the aneurysm ruptured during external cardiac massage and resuscitation.

The cardiac arrest may have been the result of a high spinal block or RAAA or both. Hypotension, bradycardia, nausea, and vomiting are known to occur during spinal anesthesia.[1] Hypotension during spinal anesthesia results from decreased venous return due to peripheral pooling of blood and decreased cardiac output, from a decrease in systemic vascular resistance, or from a combination of both.[15,16] In addition, bradycardia due to preganglionic block of cardiac accelerator fibers or enhanced vagal tone due to decrease in the venous return to the heart or both may further decrease the cardiac output. The cause of nausea and vomiting observed in our patient is unknown. It is suggested that unopposed vagal activity, which occurs with sympathetic blockade and cerebral hypoxemia due to decreased perfusion, are the primary causes of nausea and vomiting during spinal anesthesia.[17,18]

The sympathectomy that accompanies spinal anesthesia is dependent on the height of the block and extends for 2-6 dermatomes above the sensory level.[19] A T_4 sensory block could have caused significant hemodynamic changes in this patient. In addition, inadequate compensatory circulatory mechanisms in the elderly may further accentuate the hemodynamic changes leading to cardiac arrest. Inadvertent upward extension of the motor blockade leading to respiratory insufficiency also may contribute to the cardiac arrest. In addition, hypoxia and hypercarbia associated with even modest respiratory insufficiency caused by the sedative drugs may accentuate the hemodynamic effects of spinal anesthesia.[4] However, our patient received only 2 mg midazolam intravenously before the subarachnoid injection, and he was awake and alert and complaining of abdominal and back pain immediately before the event. Furthermore, this patient was hypertensive just before the cardiac arrest, which suggests that the cardiac arrest was more likely to be from a cause other than sympathectomy due to spinal anesthesia.

If the patient did have a contained RAAA, the decreased abdominal muscle tone resulting from spinal anesthesia may have removed the tamponade effect of a contained RAAA and precipitated exsanguination within minutes. In addition, the sympathectomy due to spinal anesthesia would attenuate the normal compensatory response. Plasma catecholamine concentrations are significantly decreased in patients who have a spinal anesthesia level of T_6 or higher,[20] which can contribute to decompensation and circulatory failure after RAAA. The maximum decrease in blood pressure usually occurs 15-30 min after the subarachnoid injection.[1] Therefore, the abrupt onset of cardiac arrest in our

patient suggests presence of other etiologic factors, such as RAAA, responsible for the circulatory failure.

It was assumed that a high spinal anesthetic led to a cardiac arrest. However, a significant reduction in the hematocrit, compared with the preoperative value, suggested the possibility of significant blood loss. Although hematocrit is regarded as an unreliable indicator of severe bleeding, because time is needed for fluid shifts from the interstitium to compensate for loss of blood volume,[21] sudden exsanguination that might occur from RAAA can result in significant reduction in hematocrit. Because the chest x-ray did not show any fluid collection, it was thought that the blood loss was in the abdominal cavity. This prompted the diagnostic peritoneal lavage, which showed the presence of blood.

The mortality rate of RAAA continues to be 40–70%.[22,23] Significant hypotension, low hematocrit, and cardiac arrest are associated with a mortality rate of more than 70% in RAAA.[24,25] Patients with long diagnostic delay times had a lower mortality rate, probably due to less severe ruptures or a better ability to compensate for the blood loss.[10] The occurrence of this catastrophic event in the operating room may be an important factor responsible for the favorable outcome in this patient. However, Caplan *et al.*[4] found an extremely poor outcome after cardiac arrest during spinal anesthesia despite prompt initiation of cardiopulmonary resuscitation. Perhaps the early use of epinephrine and placement of femoral vein catheter for aggressive fluid resuscitation could have resulted in prompt recovery from the cardiac arrest and prevented an adverse outcome in our patient.

Recently, Longnecker[26] advocated increasing our responsibility to preoperative preparation and postoperative care of surgical patients. Saidman[27] proposed renaming the specialty of anesthesiology as perioperative medicine and pain management. The role played by our anesthesiology team, not only in the resuscitation, but also in the decision-making with regard to transport to the intensive care unit and diagnosis, is an example of how we could work our way toward playing the role of perioperative physicians.

In conclusion, this case provides an illustration of how interplay between the hemodynamic effects of spinal anesthesia and an RAAA can produce a cardiac arrest. Aggressive resuscitation with early use of epinephrine and expeditious diagnostic and therapeutic efforts contributed to the favorable outcome.

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Apparent Failure of a Precordial Magnet and Pacemaker Programmer to Convert a DDD Pacemaker to VOO Mode during the Use of the Electrosurgical Unit

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CARDIAC pacemaker failure in patients who are pacemaker dependent results in asystole, and can be catastrophic. A major concern is how electromagnetic interference (EMI) from the electrosurgical unit (ESU) will alter pacemaker function. The effects of EMI on pacemaker function are multiple. Electromagnetic interference, by itself, can reprogram some multiprogrammable pacemakers.[1–3] In addition, some pacemakers may automatically convert to the VOO (see table 1 for explanation of pacemaker code) or DOO mode.[4,5] Others may be totally inhibited.[4,5] If total inhibition occurs in a pacemaker-dependent patient, the result will be asystole. Pacemaker inhibition induced by EMI from the ESU generally responds to placement of a precordial magnet on the skin overlying the pacemaker generator. This effectively activates a magnetic reed switch within the pacemaker that converts the pacemaker to a VOO or DOO mode as long as the magnet overlies the generator unit. Atlee[4] recommends having the pacemaker reprogrammed to the VOO mode using a pacemaker programmer. In the VOO mode, the pacemaker is not expected to sense the EMI and, therefore, will continue to pace the heart asynchronously. We report a case of a pacemaker-dependent patient where EMI from the ESU produced total pacemaker inhibition, despite proper placement of a precordial magnet or use of a pacemaker programmer.

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Key Words: Heart: pacemaker function during surgery. Equipment: pacemakers; electromagnetic interference.

Case Report

The patient was a 70-yr-old man scheduled for resection of a carcinoma of the left neck. He had a DDDR pacemaker (Model 1254, Telectronics, Englewood, CO) placed for the treatment of complete heart block 3 yr before his current admission. He was currently pacemaker dependent. The pacemaker generator was placed in the left infraclavicular region and attached to endocardial ventricular (Cordis Core, Miami, FL, model 330-201) and atrial (Telectronics, model 330-801) leads. He had no subsequent problems related to his heart or pacemaker since its insertion. Regular clinic visits showed that his pacemaker was working appropriately and the intended programmed settings, in the DDD mode, were operational.

Physical examination revealed an arterial pressure of 150/70 mmHg and a paced heart rate of 70 beats/min.