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Case Report

Cerebral oximetry for early diagnosis of intraoperative type A aortic dissection during endovascular aortic repair: a case report

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A case was diagnosed with Stanford type A aortic dissection (TAAD) during endovascular aortic repair (EVAR), complicating with cardiac tamponade and asystole as a result. Intraoperative TAAD is rare but can be life threatening without prompt diagnosis and appropriate treatment. Its diagnosis requires highly clinical suspicion and appropriate diagnostic tools. However, its symptoms may be masked by general anesthesia, and the clinical presentation is often nonspecific. Thus, widely adopted screening tools, such as classic clinical triad of acute aortic dissection or aortic dissection detection risk score (ADD-RS), are not applicable for patients undergoing surgery, making a timely diagnosis of intraoperative TAAD challenging. Moreover, overuse of diagnostic tools can harm patients or interrupt the procedure. The use of perioperative monitoring tools, such as cerebral oximetry, may aid in establishing clinical suspicion and facilitate timely diagnosis for intraoperative TAAD. Routine use of cerebral oximetry can be considered for high-risk populations undergoing regular surgeries or surgeries with a high-risk of TAAD, such as EVAR.

Keywords: intraoperative type A aortic dissection, cerebral oximetry, endovascular aortic repair, general anesthesia

1. Introduction

Endovascular aortic repair (EVAR) stands as a prevalent therapeutic option for aortic pathologies, such as aortic aneurysms and chronic aortic dissections. In contrast to conventional open repair, EVAR is associated with lower perioperative morbidity and mortality. Nonetheless, EVAR still carries the potential for various perioperative complications. Among these complications, aortic dissection is one of the most lethal. During the EVAR procedure, arterial puncture is performed to establish vascular access for the placement of endografts. However, it poses the risk of access site complications such as hematoma, pseudoaneurysm, or even arterial dissection.(1) This is attributed to the compromised integrity of the arterial wall.

Severe arterial dissection is not common; however, it remains a potential complication during EVAR.(2) Stanford type A aortic dissection (TAAD) is one of the most severe complications of EVAR and can lead to hemodynamic compromise and even mortality. In addition to arterial puncture, various procedure-related factors such as the manipulation of catheters, wires, and devices, repeated balloon dilation, and graft oversizing may lead to aortic wall injury and result in TAAD. These factors are all associated with EVAR itself, and both the surgical and anesthesia teams must be aware of TAAD during EVAR. (3) According to current research, the incidence of retrograde TAAD in thoracic endovascular aortic

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repair (TEVAR) is approximately 1.7%.(2) Once it occurs, the mortality rate is exceedingly high. The other mechanisms of TAAD include aortic wall weakness, natural progression of the underlying disease. When TAAD occurs intraoperatively, clinical symptoms are often masked by anesthesia, leading to delayed or missed diagnosis, which can significantly worsen the prognosis. Currently, there is no effective screening tool or method to guide the intervention of diagnostic tools and therapeutic measures for patients undergoing surgery. Timely diagnosis only relies on highly clinical suspicion. During anesthesia, clinical suspicion is mainly based on intraoperative monitoring, such as abrupt hypotension.

Cerebral oximetry is device used to noninvasively monitor intracranial tissue oxygen saturation and enable perioperative evaluation of various physiological changes, such as anemia, low cerebral perfusion, low cardiac output, and mechanical obstruction of cerebral blood flow (CBF).(4) If a bilateral or unilateral reduction of ≥20% from baseline saturation values is noted, the aforementioned conditions should be ruled out. When used in combination with perioperative condition, cerebral oximetry can assist in achieving an accurate diagnosis for intraoperative emergencies.

Herein, we describe a case presenting with sudden and persistent intraoperative cerebral oxygen desaturation, ultimately diagnosed with TAAD. For acute aortic syndromes, both misdiagnosis and overtesting are key concerns. Under normal circumstances, if an awake patient presents with the clinical triads of acute aortic dissection or scores high on screening tools such as the aortic dissection detection risk score (ADD-RS), immediate consideration of aortic imaging is recommended. (5) However, this approach may not be applicable to patients undergoing surgery, as symptoms are masked by general anesthesia. Diagnostic tools, such as computed tomography (CT), are usually unavailable in the operating room. Therefore, another screening tool may be necessary for patients in the perioperative setting. Under such conditions, cerebral oximetry can be used to obtain an early warning of intraoperative TAAD, which can enable early and timely intervention of diagnostic tool. This facilitates early diagnosis and treatment, which may serve as a standardized assessment specific for high-risk patients undergoing surgery.

2. Case report

An 83-year-old man with a history of Stanford type B aortic dissection and abdominal aortic aneurysm (AAA) had an endoleak of a previously mounted stent graft, as evidenced on CT (Figure 1). He was admitted to our hospital and scheduled to undergo EVAR. He had hypertension, moderate to severe mitral and tricuspid regurgitation, permanent atrial fibrillation, and end-stage renal disease. He had undergone EVAR twice before for AAA.

In the operating room, general anesthesia began at 8:00. The bispectral index and cerebral oximetry were monitored to optimize perioperative management. Bilateral radial artery catheters were placed for real-time blood pressure monitoring. An 8-Fr central venous catheter was placed in the right internal jugular vein for possible blood transfusion or administration of vasopressors and inotropes.

The procedure began with the insertion of a pigtail catheter through the right femoral artery for intraoperative aortography. A branched stent was placed through the left subclavian artery to protect the celiac trunk and superior mesenteric artery blood flow. Arterial blood pressure monitoring was switched to the right radial arterial catheter during the rest of the procedure. At 11:40, displacement of the left subclavian artery sheath led to massive hemorrhage; the left regional oxygen saturation ($LrSO_2$) decreased markedly (from 42% to 36%), whereas the right

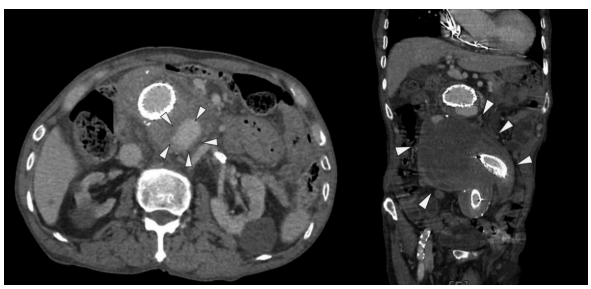


Figure 1. Preoperative image. In left image, preoperative contrast-enhanced abdominal CT indicated a type I endoleak over the left renal artery level with contrast extravasation (yellow arrowheads). The right image is a coronal view of the aneurysm sac (yellow arrowheads), with a maximum diameter of 15 cm.

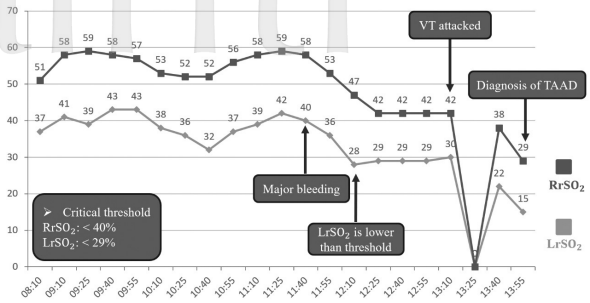


Figure 2. Trend of rSO2 values during surgery. The baseline LrSO2 was 37% (critical threshold: 29%), and the baseline RrSO2 was 51% (critical threshold: 40%). After the bleeding event at 11:40, the LrSO2 decreased to below the critical threshold at 12:10, and the RrSO2 gradually decreased. Even after appropriate treatment, the values did not return to the baseline levels. Following the VT event at 13:10, the bilateral rSO2 values decreased to below the critical threshold, indicating cerebral malperfusion. The rSO2 decline occurred 2 hours prior to the diagnosis of intraoperative TAAD.

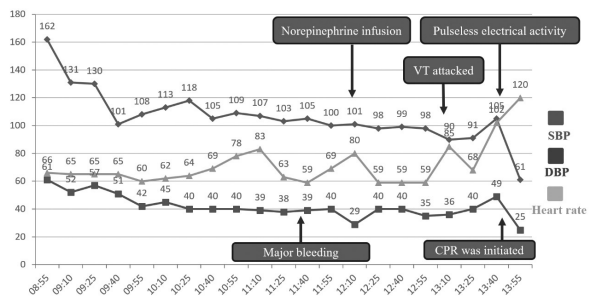


Figure 3. Trend of perioperative vital signs. Following anesthetic induction, the SBP gradually declined, maintaining within the range of 100-120 mmHg. Subsequently, at 11:40, a displacement of the left subclavian artery sheath led to significant hemorrhage, causing a reduction in SBP and an increase in heart rate. Subsequent to blood transfusion and the administration of norepinephrine (at 12:10), the SBP stabilized. However, conversely, the regional oxygen saturation value gradually declined. Asystole with PEA ultimately attacked at 13:50.

regional oxygen saturation ($RrSO_2$) decreased slightly (from 59% to 53%; Figure 2). Emergency refixation was performed. The cause of cerebral desaturation was deemed to be blood loss and anemia. Blood transfusion with 4 units of red blood cell was performed, and norepinephrine were administered for hemodynamic optimization.

After the treatment, perioperative vital signs gradually stabilized (Figure 3). However, the bilateral

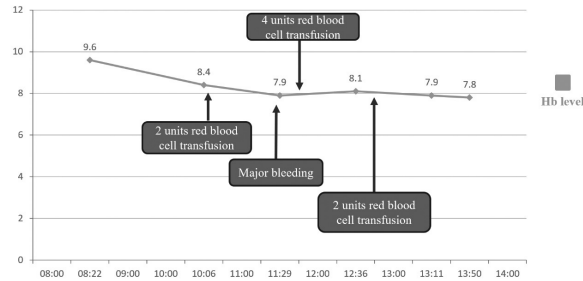


Figure 4. Trend of perioperative hemoglobin level (gm/dL). During the surgery, the patient's hemoglobin levels were continuously monitored through ABG analysis. At 10:06, the ABG results indicated perioperative anemia, prompting the administration of 2 units of concentrated RBCs. Subsequent to a major bleeding event at 11:40, an additional 4 units of concentrated RBCs were transfused, with the entire transfusion process completed by 12:30. However, subsequent ABG results at 12:36 revealed a hemoglobin level of 8.1 gm/dL, accompanied by a gradual decline in regional oxygen saturation value. This may suggest occult bleeding occurring outside the surgical field. Additional 2 units of concentrated RBCs were given and there was no further survey except ABG analysis. Eventually, the patient underwent emergency pericardiotomy, draining a minimum of 1000 ml of pericardial fluid, potentially representing the source of occult bleeding during the surgery.

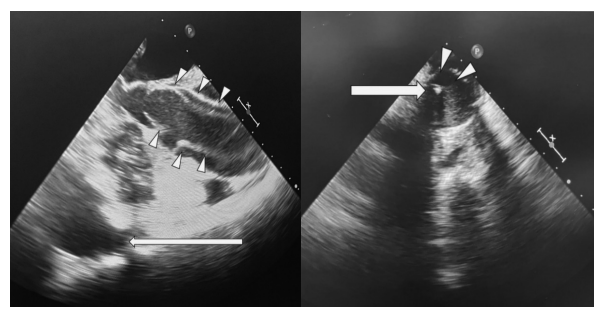


Figure 5. The left image is midesophageal long axis view from TEE, indicating type A aortic dissection (yellow arrowheads) complicated with massive pericardial effusion (yellow arrow), leading to cardiac tamponade. Dissection ranged from the sinus of Valsalva to at least the thoracic descending aorta. The right image is descending aortic short axis view, revealing dissection flap (yellow arrowheads) and guidewire used in procedure (yellow arrow).

regional oxygen saturation (rSO_2) continued to decrease, and at 12:10, the $LrSO_2$ decreased to the critical threshold (29%). FiO_2 was adjusted to 1.0, and mean arterial pressure was maintained at>65 mmHg through norepinephrine infusion (0.052–0.173 μg/

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kg/min). Arterial blood gas (ABG) analysis (Figure 4) revealed anemia (hemoglobin: 8.1 gm/dL). This explained why the bilateral rSO_2 did not return to baseline and continued to decline despite the blood transfusion. Additional blood products with 2 units were given. At 13:10, ventricular tachycardia was noted; cardioversion was performed, and a repeated ABG analysis revealed anemia (hemoglobin: 7.9 gm/dL) without other abnormal finding, indicating the patient remained refractory to blood transfusion. At 13:50, asystole with pulseless electrical activity occurred; transesophageal echocardiography (TEE) revealed TAAD complicated with cardiac tamponade (Figure 5). Emergency pericardiotomy was performed, and the amount of pericardial fluid was >1000 mL. Finally, the patient died at 14:55.

3. Discussion

TAAD is a surgical emergency, and the prognosis is poor if it is not promptly recognized and treated. An anesthesiologist should be aware of the potential TAAD among high-risk patient groups that have multiple predisposing factors.(6) There are several predisposing factors in this case, including severe aortic atherosclerosis, AAA and a previous history of Stanford type B aortic dissection. Additionally, arterial cannulation is a procedure-related predisposing factor. Therefore, vascular surgery itself is also a predisposing factor.

Intraoperative TAAD can be diagnosed through CT, TEE, and aortography.(6) However, excessive use of these instruments interrupts procedures and can harm the patient because of, for example, contrast and radiation exposure. Thus, there is a need for an efficient screening tool to guide the intervention of diagnostic tools. Traditionally, when patients present with high-risk clinical features, such as the clinical triad of aortic dissection: abrupt onset of thoracic pain with a sharp, tearing character, a variation in pulse and/or blood pressure, and mediastinal widening on chest radiograph, or get scores high in ADD-RS, there is a strong suspicion of TAAD. In such cases, a contrast enhancement CT is arranged for further diagnosis. However, the symptoms of TAAD, such as tearing pain, are masked by general anesthesia, and continuous monitoring through chest radiography is unattainable during surgery, making it challenging to timely diagnose TAAD. Moreover, contrast enhancement CT scans are typically only available in hybrid operating rooms. The traditional screening tool and diagnostic algorithm for acute aortic symptoms seems not applicable to patients undergoing surgery. Therefore, alternative screening tools and proper perioperative monitoring are needed to assist in the diagnosis of TAAD. There are several alternative tools or methods.

TEE itself is a diagnostic tool for TAAD and can be used for perioperative monitoring. TEE, as an imaging modality for diagnosing TAAD, has a sensitivity of up to 98%,(7) making it suitable for perioperative monitoring in high-risk patients. However, TEE is an invasive perioperative monitoring, and during EVAR surgery, TEE can frequently interrupt the procedure. Therefore, an appropriate screening method or tool to guide the timing of TEE intervention, in order to avoid frequent interruptions during surgery, is very important. As previously mentioned, acute aortic dissections can be identified based on the classic clinical triad. Approximately 96% of cases can be recognized based on it, enabling early intervention with diagnostic tools and facilitating early diagnosis.(8) Isolated blood pressure differential also strongly predicts TAAD with an 83% probability, making the measurement of non-invasive blood pressure (NIBP) in both upper limbs during the perioperative period seem feasible as a real-time monitoring tool and screening method. However, only 19-30% of TAAD patients present with pulse deficit,(9) making NIBP alone a low-sensitivity screening tool. Consequently, isolated blood pressure differential is not an optimal screening method and should be combined with other screening tools to enhance the sensitivity.

Compared to TEE and NIBP measurement of bilateral upper limbs, cerebral oximetry seems to be a favorable option, offering non-invasive and continuous perioperative monitoring. Moreover, cerebral oximetry can guide various intraoperative interventions, including initiating transfusion therapy, adjusting minute ventilation, and administering inotropes. These advantages make it superior to other screening or diagnostic tools. When cerebral oximetry values decline, obtaining NIBP measurements from

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bilateral upper limbs may serve as a substitute for the classic clinical triad and ADD-RS for patients undergoing surgery, serving as a high-risk predictor for intraoperative TAAD. Further advanced studies are required to clarify the sensitivity of combining blood pressure differential and cerebral desaturation as a screening method for TAAD.

The mechanism underlying the use of cerebral oximetry as a screening tool for TAAD involves CBF obstruction and cardiogenic shock, both of which originate from TAAD and lead to cerebral desaturation. According to research, approximately 30% of TAAD patients have common carotid artery dissection(10), and 10%-20% have cerebral malperfusion.(11) A significant correlation has been reported between mean middle cerebral artery flow velocity and rSO2, with blood flow obstruction causing decreased rSO2 values.(12, 13) Therefore, intraoperative TAAD can result to unilateral or bilateral reduction of rSO2 value. However, the cutoff value of cerebral desaturation indicating meaningful mechanical obstruction of CBF resulting from TAAD remains uncertain. Nevertheless, refractory cerebral desaturation after adequate treatment should raise a high suspicion of CBF obstruction according to current algorithm.(4)

Cerebral oximetry has already previously been used to assist in diagnosing intraoperative TAAD. (14, 15) Wang et al. reported a case of intraoperative innominate artery dissection during arch replacement and TEVAR.(16) A sudden reduction in the RrSO2 was noted during TEVAR, and cerebral desaturation persisted despite hemodynamic optimization. Subsequently, aortic dissection wasdiagnosed using emergency fluoroscopy. In their report, a unilateral or bilateral reduction in the rSO2 served as a warning sign of innominate artery dissection due to cerebral malperfusion. Intraoperative cerebral desaturation is generally managed on the basis of a widely adopted algorithm.(4) Once cerebral desaturation occurs, the mechanical obstruction of CBF, especially unilateral cerebral desaturation, must be ruled out initially. If cerebral desaturation persists despite systemic treatment and hemodynamic optimization, the presence of CBF obstruction should be reevaluated. Moreover, it is crucial to promptly rule out intraoperative TAAD among high-risk patients. Returning to this case, the patient presented with multiple predisposing factors for TAAD and underwent vascular surgery. During the procedure, sheath displacement occurred, potentially damaging the artery and leading to arterial dissection. The patient received concentrated RBC transfusion and norepinephrine administration, and the vital signs gradually stabilized. However, the cerebral oximetry value continued to decline despite appropriate treatment with FiO2 adjusted to 1.0, indicating other problems for insufficient oxygen supply to the brain. Following the algorithm, other potential causes should be ruled out, such as whether CBF obstruction occurred. Because sheath displacement occurred during the surgery, which could trigger arterial dissection or even TAAD, especially in such high-risk case, measuring NIBP in both upper limbs might reveal a blood pressure differential. At this point, TEE or aortography could have facilitated the early diagnosis of intraoperative TAAD. Cerebral desaturation occurs earlier than the onset of VT and hypotension by an hour, serving as an early warning sign of TAAD. Despite a delayed diagnosis, if cerebral oximetry had not been used as perioperative monitoring, there was no other early clinical feature of TAAD had been observed during the surgery. We reported this case, conducted a literature review, and recommend the application of cerebral oximetry as perioperative monitoring for patients at high risk of TAAD. Furthermore, the application of combining cerebral oximetry with bilateral blood pressure measurement can be a substitute for the classic clinical triad and ADD-RS in patients undergoing surgery, serving as a high-risk predictor for intraoperative TAAD.

4. Conclusion

Although the sensitivity and specificity of cerebral oximetry in detecting TAAD remain unclear, we recommend routine application of cerebral oximetry in high-risk patient group undergoing EVAR surgery. If a perioperative reduction in rSO2 greater than 20% compared to baseline is observed, along with the presence of blood pressure differential, TAAD should be highly suspected. Furthermore, if a patient present with a refractory response to cerebral desaturation

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treatment, even in the absence of blood pressure differential, as in this case, there should also be a high suspicion for TAAD. Prompt TEE or aortography should be introduced to rule out TAAD at this point, especially in high-risk procedures and patient groups.

Ethics Statement

This work complies with the ethical standards of the Declaration of Helsinki and its later amendments. Informed consent was obtained from the patient before performing the procedures.

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