Hand ischemia associated with profound hypotension and radial artery catheterization in a pediatric patient: A case report

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Direct arterial blood pressure monitoring via the radial artery is a relatively common method employed during the perioperative period. Complications attributed to radial artery catheterization are extremely rare but can include thrombosis, ischemia, infection, and aneurysm formation at the site of catheter insertion. This report describes an episode of hand ischemia in a pediatric patient. The patient experienced a brief period of hypotension secondary to blood loss. A pallor hand was noticed after the patient had been adequately resuscitated. Appropriate treatment was administered, and the patient was eventually discharged to home without any further complications.

Key words: **Arterial line, collateral circulation, direct blood pressure monitoring.**

Direct arterial blood pressure monitoring during the perioperative period is indicated when patients may be hemodynamically unstable during the surgical procedure or when frequent sampling of arterial blood gas tensions are indicated.[1,2] Arterial blood pressure monitoring is usually performed using the radial artery. The radial artery is accessed primarily because of its location and ease of entry. In addition, potential ischemic complications that may arise from cannulating the radial artery are reduced by a significant presence of collateral circulation to the hand. Other complications of direct arterial blood pressure monitoring are extremely rare but may include thrombosis, ischemia, infection, and aneurysm formation at the site of catheter insertion.[1,3]

The primary objective of monitoring the blood pressure with an arterial catheter is to recognize moment-to-moment changes in a patient's hemodynamic status. Hypovolemia from blood loss will decrease oxygen delivery to the tissues, and a compensatory peripheral vasoconstriction will occur. The presence of an arterial catheter during a hypotensive episode is beneficial in measuring the degree of hypotension, but also may contribute to promoting ischemia to the hand by serving as a mechanical obstruction to blood flow. Following is a case report of a pediatric patient who developed hand ischemia during an episode of hypotension with a radial artery catheter in place.

Case summary

A 14-year-old, 50-kg, 173-cm male presented for a posterior spinal fusion of T5 through L3 to correct a severe idiopathic adolescent scoliosis. His medical history was unremarkable. One month prior to surgery, he had donated 2 units of whole blood in anticipation of significant blood loss during the surgery. Complete blood count obtained preoperatively on the day of surgery revealed hematocrit of 34%, hemoglobin of 11.7 gm/dL, and platelets of 206,000. Baseline noninvasive blood pressure measurement was 115/65 mm Hg with a heart rate of 68 beats per minute. Somatosensory-evoked potential monitoring was initiated preoperatively and was within normal limits. A 20-gauge intravenous (IV) line was placed in the left hand, and a smooth IV induction was performed using standardized technique. Immediately after induction, an additional IV catheter was placed in the left antecubital vein using a 16-gauge catheter. An arterial line was placed percutaneously on the first attempt in the right radial artery with a 20-gauge angiocatheter. The presence of collateral circulation was not determined prior to the placement of the radial artery catheter. The appropriate arterial waveform was present, and the invasive monitoring pressures correlated with the noninvasive cuff pressures present in the same extremity.

The patient was then placed in the prone position on a Wilson frame. The Wilson frame is a positioning device used for posterior spinal surgeries. It consists of 2 curved chest bolsters that allow for improved chest expansion and decreased abdominal pressure in the prone patient. These bolsters also are adjustable to assist with spinal alignment. The patient's arms were padded and placed on armboards at a 35-degree angle.

The upper extremities were both within view of the anesthesia provider throughout the case.

Anesthesia was maintained using a combination of isoflurane, nitrous oxide, IV vecuronium, and intermittent IV fentanyl boluses. A total of 400 µg of fentanyl was administered during the first 30 minutes following the surgical incision. Cell salvage was initiated at the onset of the procedure. Approximately 30 minutes into the procedure, the surgeon requested a period of deliberate hypotension. A fentanyl infusion was then initiated at 2.1 µg/kg per hour, and the volatile anesthetic was titrated between an end-tidal concentration of 0.5 and 1.5% to maintain the mean arterial pressure (MAP) within the requested range of 50 to 60 mm Hg. The patient's arterial blood pressure stabilized (80s/40s mm Hg, MAP of 55-60 mmHg) 25% to 30% below baseline for approximately 90 minutes. The arterial waveform remained unchanged, and somatosensory-evoked potential monitoring remained similar to baseline.

Approximately 3 hours into the procedure, the patient's blood pressure dropped precipitously (60s/40s mmHg), and tachycardia became immediately present. No other electrocardiogram changes were noted. Overall, estimated blood loss up to this point was 1,800 mL, approximately 50% of the patient's total blood volume. Hemoglobin measured at this time was 8.3 g/dL. Urine output was 175 mL. Fluid replacements had consisted of 5,000 mL of of lactated Ringer's solution, 500 mL hetastarch solution, and 1,050 mL of cell salvaged blood. The arterial waveform dampened then disappeared. However, the noninvasive cuff pressures were consistently recording at 70s/50s mm Hg. The previously normal somatosensory-evoked potential monitoring was now interrupted. The fentanyl infusion was discontinued, the isoflurane decreased to an end-tidal concentration of 0.2%, and phenylephrine boluses in 50-µg alloquats were given to a total of 100 µg. This was followed by a phenylephrine drip at 50-100 µg/min that was infused over approximately 40 minutes. An additional 18-gauge IV catheter was placed in the right antecubital vein, and aggressive fluid administration was performed.

The fluid resuscitation period continued over the next 2.5 hours. An additional 1,500 mL of cell salvaged blood, 900 mL of autologous whole blood, 450 mL banked whole blood, and 500 mL of banked packed red blood cells were given. The arterial waveform reappeared after approximately 10 minutes, and a normotensive state was restored (MAP of 80 mm Hg) after 45 minutes. The approximate time period when the MAP was below 50 mm Hg was 10 minutes. Estimated blood loss at the conclusion of the case was approximately 5,900 mL. A total of 4,600 mL of lactated Ringer's solution, 2,600 mL of normal saline, 500 mL of hetastarch solution, 2,550 mL of cell saver blood, 900 mL of autologous whole blood, 500 mL of banked red blood cells, and 450 mL of banked whole blood were administered over the entire case. Total urine output was 365 mL.

The patient was transferred to the postanesthesia care unit where his right hand was noted to be cool and cyanotic. Despite a normal arterial waveform, the right radial pulse was not palpable, and capillary refill in the fingers was greater than 3 seconds. Blood was immediately aspirated from the arterial line to determine the absence of thrombosis and the presence of blood flow. This blood was later sent for analysis and revealed a pH of 7.18, P_{CO_2} of 42, P_{O_2} of 526, HCO_3^- of 15, and base excess of negative 13. Complete blood count revealed hemoglobin of 12.6 g/dL, hematocrit 36.3%, and platelets of 83,000. The arterial line was promptly removed, and digital pressure was applied in the standard fashion to prevent formation of hematoma. A warm compress was then applied to enhance vasodilation of blood vessels and improve collateral blood flow. Immediate improvement in the color was noted. A vascular surgeon was consulted and he found the right radial pulse; bilateral ulnar pulses were nonpalpable and absent via Doppler assessment. While the patient remained in the postanesthesia care unit, the vascular surgeon diagnosed a moderate ischemia secondary to a combination of profound hypotension and probable radial artery occlusion from the arterial line. The patient's progress was observed several times over the next 20 hours by the vascular surgeon. Within this timeframe, the patient regained a right arterial pulse, and his hand became warm and well perfused. No further intervention was required. The patient was discharged home on postoperative day 5 with complete resolution of the hand ischemia.

Discussion

Radial artery ischemia postcannulation has been attributed to (1) thrombosis formation at the site, (2) emboli originating from a proximal site, (3) excessive trauma from multiple attempts, (4) very long or large bore catheters, (5) prior vascular disease, (6) hyperlipoproteinemia, or (7) prolonged hypotension.[1,3] Distal vascular insufficiency often presents in the form of a pale, cold thenar eminence.[4,5] Singleton reported a case of radial artery spasm after cannulation with a 22-gauge catheter.[5] In this case report, the patient's hand remained pink throughout the anesthetic but was noted to be pale in the postanesthesia care unit and

remained pale for 30 minutes after decannulation of the radial artery.[5] A review of literature revealed multiple case reports of radial artery ischemia. However, only 1 case report demonstrated radial artery ischemia in a pediatric patient. Samaan reported an incidence of gangrene which eventually required amputation above the wrist in a 17-year-old male following radial artery cannulation for monitoring during open heart surgery.[6] However, the radial artery catheter was placed using a cutdown procedure, and the distal end of the radial artery was ligated. Samaan hypothesized that catheter interference during peripheral vasospasm induced by hypotension contributed to a state of poor blood supply to the extremity.[6]

A 1983 investigation prospectively examined 1,699 subjects undergoing radial artery catheterization.[7] The only subjects that suffered ischemia were those with multiple emboli or prolonged low cardiac output requiring high-dose vasopressors.[7] Our patient did require a phenylephrine infusion of approximately 8 mg titrated over 40 minutes to maintain his MAP in the low 50s mm Hg. As a direct-acting arteriole vasoconstrictor, phenylephrine could have contributed to our patient's ischemia.

Research investigations have determined that the Allen's test is not a predictive indicator of potential ischemia following radial artery cannulation.[1,3,7-9] The incidence of partial or complete occlusion of the radial artery after decannulation has been reported as high as 25% regardless of the presence of a negative pre-procedural Allen's test.[7] One investigator injected fluorescein through a radial artery catheter in subjects with a negative Allen's test.[8] After the injection, with the ulnar circulation intact, the radial artery was shown to perfuse only the thumb and thenar eminence. However, the occlusion of the ulnar artery resulted in the radial artery successfully perfusing the entire hand. The conclusion was that the Allen's test has no clinical ability to predict collateral perfusion of the hand.[8]

The suspected cause of this patient's ischemic episode can be attributed to a normal relatively small radial artery in a 50-kg, 14-year-old male; a 20-gauge radial artery catheter; and a period of profound hypovolemia requiring massive transfusion. During the period of hypotension, his peripheral blood flow was most likely shunted to the central core by profound peripheral vasoconstriction induced by vasopressors and hypotension. Arterial intima trauma, secondary to cannulation and significant decrease in peripheral blood flow during severe hypotension, leaves the artery primed for thrombus formation and spasm.[9-12] Extremities so affected will appear pale and feel cool to the touch. Capillary refill may be greater than 3 seconds, and pulses may be absent. When such symptoms of impaired perfusion are observed, initial treatment should consist of immediate removal of the catheter.[12]

If improvement of symptoms is not observed, aggressive treatment includes ipsilateral brachial plexus or stellate ganglion blockade to induce sympathectomy and subsequent vasodilation.[3,12] If thrombus formation is the suspected cause, anticoagulation with IV heparin of 5,000 units every 6 hours may be beneficial in improving blood flow.[12] If these measures are ineffective, surgical exploration should be promptly pursued.[3,12] Fortunately, our patient responded to immediate decannulation of the arterial catheter and application of warm compresses and did not require more aggressive treatment. Suspecting the possibility of, and searching for, peripheral ischemia during profound hypotension, may allow for early, conservative treatment and prevent permanent vascular damage.

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