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Massive bleeding during spine surgery in a patient with ankylosing spondylitis

Purpose: Ankylosing spondylitis is associated with pathophysiology that has important anaesthetic implications. We report a case where the sequelae of ankylosing spondylitis may have been responsible for massive bleeding during emergency spine surgery.

Clinical Features: A 69 yr old man with long standing ankylosing spondylitis sustained a complex fracture of the lumbar spine in a fall, and was scheduled for stabilization of the spine. Under general anaesthesia, prone positioning was difficult because of the extreme spinal deformity. During exploration, dilatation of epidural veins was encountered and sustained haemorrhage was encountered throughout the surgical procedure. Estimated blood loss was 17,000 ml which was replaced with 31 units of packed red blood cells, 3200 ml of salvaged blood, 18 units of fresh frozen plasma, 26 units of platelets, 1,000 ml of albumin and 9,000 ml of crystalloid.

Conclusions: Extreme deformity of the spine led to positioning difficulties that may have contributed to massive blood loss during complex spine surgery. Difficulties with placement in the prone position in patients with advanced ankylosing spondylitis should be anticipated.

Objectif : La spondylarthrite ankylosante est associée à la physiopathologie ayant d'importantes implications anesthésiques. Nous rapportons un cas où les séquelles de la spondylarthrite ankylosante peuvent avoir été responsables d'un saignement abondant pendant une chirurgie d'urgence de la colonne vertébrale.

Aspects cliniques : Un homme de 69 ans, atteint de spondylarthrite ankylosante depuis longtemps, a subi lors d'une chute une fracture complexe de la colonne lombaire pour laquelle on a programmé une stabilisation. Pendant l'anesthésie générale, l'importante déformation de la colonne a compliqué l'installation en décubitus ventral. Lors de l'exploration, on a dû faire face à la dilatation des veines périurales et à une hémorragie, qui s'est prolongée pendant toute la durée de l'intervention. La perte sanguine, estimée à 17 000 ml, a été compensée par 31 unités de globules rouges concentrés, 3 200 ml de sang récupéré, 18 unités de plasma frais congelé, 26 unités de plaquettes, 1 000 ml d'albumine et 9 000 ml de cristalloïdes.

Conclusion : L'importante déformation de la colonne vertébrale a compliqué l'installation du patient, ce qui a pu contribuer à des pertes sanguines abondantes pendant une chirurgie complexe de la colonne. Des difficultés d'installation en décubitus ventral sont à prévoir avec un patient hospitalisé souffrant de spondylarthrite ankylosante avancée.

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ANKYLOSING spondylitis (AS) is a chronic, progressive inflammatory disease of synovial joints which can lead to deformity of the spine and weight-bearing joints. For unknown reasons, progressive inflammation develops at the insertion of ligaments and joint capsules into bone (entheses).¹ The progressive inflammation of the ligamentous stability of the spine creates pain. The compensatory postural changes and osteoporosis lead to progressive ankylosis and kyphosis of the spine. The spinal deformity consists of squaring of the vertebral bodies, vertebral end-plate destruction, syndesmotic formation and kyphosis from osteoporosis and ventral vertebral body reabsorption with increasing frequency after the third decade.^{21,3}

Anaesthetic considerations for patients with ankylosing spondylitis include difficulty with positioning,⁴ difficult or impossible access to the airway,^{5,6,7,8} chronic central neuropathy,⁹ and increased technical difficulty with neuraxial anaesthesia.^{6,10,11,12} Although the deformity of AS presents positioning problems, we were unaware that positioning of an AS patient for urgent surgery may contribute to massive haemorrhage. We report the details of a case where the sequelae of AS contributed to massive blood loss during urgent, complex spine surgery.

Case report

The patient was a 69 yr old, 60 kg Caucasian man with a long-standing history of ankylosing spondylitis, manifest by disabling chronic back pain, "bamboo-spine", history of cervical fracture from minimal trauma treated with a six month course of halo-traction and progressive cervico-thoracic kyphosis. Three weeks before admission, he fell and noted a marked increase in his back pain. Radiological investigation was interpreted as normal. He remained in severe pain with new radicular symptoms and went to a different hospital, where radiological examination was also reported as within normal limits. He came to our institution where tomographic x-rays revealed a compression fracture of ventral aspect of the vertebral body at T₁₂, the most angulated area of the kyphosis. Magnetic resonance imaging (MRI) revealed encroachment of the bony elements at the thoraco-lumbar junction into the spinal canal with compression of the spinal cord. Surgery plans aimed at relieving cord compression and preventing progression of the kyphosis by decompression around T₁₂, complete lumbar spine osteotomy and fusion. Neurological monitoring with somatosensory evoked potentials was planned due to the high risk of spinal cord and nerve root injury.

Past medical history was positive for chronic hypertension, treated with hydrochlorothiazide 50 mg daily and captopril 150 mg daily, and coronary artery disease. Trans-thoracic echocardiography revealed good ventricular function with an ejection fraction of 55%. A persantine-thallium cardiac imaging test failed to detect any areas of inducible myocardial ischaemia. Previous surgery included subtotal colectomy with loop ileostomy and closure of the ileostomy, both performed under general anaesthesia initiated after awake, fibreoptic intubation. Preoperative haemoglobin and haematocrit were 13 g·dl⁻¹ and 39% respectively, and the prothrombin, partial thromboplastin time and platelet count were within normal limits.

He was premedicated with glycopyrrolate, metoclopramide and ranitidine prior to arrival to the operating room. Before induction of anaesthesia, a right radial arterial catheter and two 14 gauge intravenous catheters were placed. Nasal fibreoptic intubation was accomplished without difficulty after anaesthesia and vasoconstriction of the nares with lidocaine 2% jelly mixed with phenylephrine (1 mg phenylephrine/10 ml lidocaine jelly) and topical anaesthesia of the oropharynx and airway with nebulized lidocaine 4%. Transtracheal lidocaine injection was considered but not attempted due to anatomical abnormality, since the cricothyroid membrane could not be reliably identified due to the "chin-on-chest" deformity of the neck.

After confirmation of bilateral breath sounds, the patient was turned prone prior to induction of anaesthesia so that he could position himself in a manner which produced the least pain. Before induction, he was asked to identify any radicular pain that occurred during positioning so that cord compression could be avoided.¹³ Baseline SSEP readings were obtained in the prone position and were not changed from baseline. Optimum positioning (i.e. weight bearing by the upper body and pelvis with the abdomen hanging free) could not be achieved. Either a free-hanging abdomen was achieved with high ventilation pressures from chest compression, or minimal chest compression occurred with more than ideal upward pressure on the abdomen. With generous gel and foam padding, excessive pressure decreased. Once an acceptable position was achieved, anaesthesia was induced with 250 mg thiopentone, 200 µg fentanyl and 6 mg pancuronium to facilitate tracheal intubation. Anaesthesia was maintained with nitrous oxide in oxygen (60%/40%), isoflurane 0.2-0.4%, fentanyl at 1-2 µg·kg⁻¹·hr⁻¹ and neuromuscular blockade maintained with pancuronium. The incision site, T₈ to the sacrum, was infiltrated with epinephrine 1/500,000 in saline for haemostasis. Haemostasis was easily maintained by surgical means

until work on the bones of the spine began. Intravenous nitroglycerin was used intravenously whenever mean arterial blood pressure (MAP) was greater than 80 mmHg. When laminectomy and decompression of the spinal canal began, there was copious bleeding which could not be controlled. Large epidural veins were visible throughout the wound. The level of bleeding increased during osteotomy of the pedicles of L₃ and increased further after completion of the osteotomy of the L₃ vertebral body.

Heavy blood loss was sustained during the six hour instrumented fusion. An estimated blood loss of 17,000 ml was replaced with 31 units of packed red blood cells, 1,000 ml albumin, and 3,200 ml salvaged blood. When prothrombin, partial thromboplastin times exceeded control by more than 50%, fresh frozen plasma was administered. When platelet count was < 100,000, six units of pooled platelets were administered. As a result, there was administration of 18 units of fresh frozen plasma and 24 units of pooled platelets. Despite massive blood loss and prolonged surgery, normothermia was maintained with forced air heating blankets on the upper and lower body. Oliguria, despite reasonable MAP, was the indication for 25 g mannitol and dopamine at 3 µg·kg⁻¹·hr⁻¹. At the conclusion of surgery, he was taken to the Surgical Intensive Care Unit (SICU). Prothrombin, partial thromboplastin times and platelet count were normal when first measured after one hour. Urine output exceeded 1 ml·kg⁻¹ during the first hours and oliguria did not recur. The trachea was extubated on postoperative day (POD) one, but the patient remained in SICU for three days because of confusion. Weakness of the muscles innervated by the L₂ and L₃ nerve roots was identified, left greater than right, which was not surprising, since the alteration of the course of the spinal cord was maximal at this level. He achieved ambulation with a walker and was discharged to rehabilitation unit on POD 9. He was readmitted at six weeks with a haematoma, which was treated with incision, drainage and loose reapproximation, on three occasions during an 8-day hospitalization. He was discharged home with almost complete resolution of his L₂ and L₃ weakness.

Discussion

The details of this case highlight the potential for massive blood loss in the perioperative management of the AS patient. Although not apparent before surgery, a number of factors, some related and some unrelated to ankylosing spondylitis, predisposed our patient to the severe haemorrhage.

The first difficulty was in achieving the prone position. The ideal prone position allows the abdomen to

hang freely, and puts minimum pressure on the ventral thorax in order to decrease the impact of prone positioning on ventilation and thoraco-lumbar bleeding. Upward pressure on the abdomen displaces the diaphragm cephalad, increases intra-thoracic pressure, and decreases thoracic compliance and chest excursion. The net effect is decreased functional residual capacity (FRC) of the lungs and increased ventilating pressures necessary to achieve the same tidal volume. The net increase in intra-abdominal pressure decreases the flow within the inferior vena cava, which in turn increase flow via the azygous venous system. Part of the azygous system is the valveless epidural venous plexus. Anything which increases flow in the azygous system will increase the filling of the epidural venous plexus. Highly distended, large epidural veins could be seen in the wound regularly during the heavy blood loss. The valveless anatomy of the epidural venous plexus increases blood loss because any hole in a vein leads to bleeding from both directions.

Another feature which contributed to bleeding was the increased vascularity of the skeletal structures related to the advanced AS. The pathophysiology of AS is related to infiltration of granulation tissue into the bony insertion of ligaments and joint capsules, which leads to areas of intense fibroblastic proliferation, reactive bone formation and neovascularization.^{1,5} The long-term consequences of this pathophysiology are osteoporosis and massive heterotopic bone formation that increase bleeding with bony manipulation of the spine. The chronic inflammation, heterotopic ossification, osteoporosis and the vertebral body fracture present in this patient were responsible for large amounts of friable tissue that bled excessively during the dissection.

The chronic treatment of hypertension with a large dose of an ACE inhibitor also may have contributed to bleeding in this case. Therapeutic levels of ACE inhibitors produce peripheral vasodilatation and may inhibit the normal reflex vasoconstriction response to haemorrhage.¹⁴ The mild hypotension after induction of anesthesia in patients receiving ACE inhibitors is related to a reduction in afterload, and phenylephrine to increase afterload is sometime required for treatment.¹ Absence of compensatory vasoconstriction could have accentuated the already large potential to blood loss in this case.

The prolonged interval of blood loss further contributed to bleeding by inducing a dilutional coagulopathy. Dilution of both platelets and labile clotting factors created a coagulopathy which was confirmed by elevation of PT/PTT and decreases in the platelet count, all of which occurred and were documented in this case. Other features of massive transfusion which

contribute to derangement of coagulation, including hypothermia and acidosis, did not occur in this case, perhaps because of aggressive temperature support and volume replacement. The large volume of red cell salvage in this case could also have contributed to bleeding, since the returned product is red cells suspended in saline, and contains no platelets or clotting factors, further contributing to the dilutional coagulopathy. In some salvage systems, the returned product can contain small amounts of heparin, used as the salvage-anti-coagulant, which can contribute to coagulopathy, although this is unlikely in this case since we use a system which uses OR salvage and washing/concentration in the blood bank. With these systems, the heparin levels are routinely too low to measure, although that was not evaluated directly in this case.

In conclusion, we report a case of massive haemorrhage that occurred during spinal surgery in the prone position in a patient with AS. The pathophysiology of AS, which may have contributed to this bleeding, is discussed. This case documents further the difficulties of anaesthetic management of the patient with AS and suggests the need for good venous access, temperature support, the availability of massive transfusion devices, possible central line placement and excellent support from the hospital blood bank.

References

- 1 *Ball J*. Enthesopathy of rheumatoid and ankylosing spondylitis. *Ann Rheum Dis* 1971; 30: 213-26.
- 2 *Calin A, Elswood J*. The outcome of 138 total hip replacements and 12 revisions in ankylosing spondylitis: high success rate after a mean followup of 7.5 years. *J Rheumatol* 1989; 16: 955-8.
- 3 *Russell AS, Jackson F*. Computer assisted tomography of the apophyseal changes in patients with ankylosing spondylitis. *J Rheumatol* 1986; 13: 581-5.
- 4 *Simmons EH*. Kyphotic deformity of the spine in ankylosing spondylitis. *Clin Orthop* 1977; 128: 65-77.
- 5 *Sinclair JR, Mason RA*. Ankylosing spondylitis. The case for awake intubation. *Anaesthesia* 1984; 39: 3-11.
- 6 *Kumar CM, Mehta M*. Ankylosing spondylitis: lateral approach to spinal anaesthesia for lower limb surgery. *Can J Anaesth* 1995; 42: 73-6.
- 7 *Hains JD, Gibbin KP*. Fiberoptic laryngoscopy in ankylosing spondylitis. *J Laryngol Otol* 1973; 87: 699-703.
- 8 *Hyman SA, Rogers WD, Bullington JC III*. Cervical osteotomy and manipulation in ankylosing spondylitis: successful general anesthesia after failed local anesthesia with sedation. *J Spinal Disord* 1990; 3: 423-6.
- 9 *Calabro JJ*. Ankylosing spondylitis. Resident: and Staff Physician 1980; 73: 72-81.
- 10 *Schelew BL, Vaghadia H*. Ankylosing spondylitis and neuraxial anaesthesia: a 10 year review. *Can J Anaesth* 1996; 43: 65-8.
- 11 *Wittmann FW, Ring PA*. Anaesthesia for hip replacement in ankylosing spondylitis. *J R Soc Med* 1986; 79: 457-9.
- 12 *DeBoard JW, Ghia JN, Guilford WB*. Caudal anesthesia in a patient with ankylosing spondylitis for hip surgery. *Anesthesiology* 1981; 54: 164-6.
- 13 *Simmons EH*. The surgical correction of flexion deformity of the cervical spine in ankylosing spondylitis. *Clin Orthop* 1972; 86:132-43.
- 14 *Colson P, Saussine M, Séquin J, Cuchet D, Chaptal P-A, Roquefeuil B*. Hemodynamic effects of anesthesia in patients chronically treated with angiotensin-converting enzyme inhibitors. *Anesth Analg* 1992; 74: 805-8.