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Local Anesthetic Infusion Pump Systems Adverse Events Reported to the Food and Drug Administration

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THE purpose of this report is to characterize adverse event reports on continuous direct local anesthetic infusion into surgical wounds using infusion pump systems devised for this purpose. These pump systems typically consist of disposable, nonelectrical pumps or electromechanical pumps that deliver a continuous infusion at controlled rates for a specified duration of time. Postoperative pain may be managed by continuous direct infusion of anesthetic into a surgical wound. This technique is reportedly used in a variety of surgeries, *e.g.*, inguinal hernia repair,[1] upper abdominal surgery,[2] laparoscopic nephrectomy,[3] cholecystectomy,[4] knee arthroplasty,[5] and shoulder[6] and gynecologic operative laparoscopy.[7]

Infusion pump systems for anesthetic wound perfusion are regulated by the U.S. Food and Drug Administration (FDA) as medical devices. The FDA monitors the performance of regulated medical devices *via* a passive surveillance system.

Adverse events during direct local anesthetic infusion into surgical wounds, with an infusion pump system, have been reported to the FDA. These reports involve adverse events reported for surgeries performed at a variety of surgical sites, including orthopedic, gastrointestinal, podiatric, and others. Complications encountered with these infusion pump systems include tissue necrosis, surgical wound infection, and cellulitis. Following are examples of cases reported to the FDA and a summary of 40 injuries that occurred using direct local anesthetic infusion pump systems. These reports may represent sentinel events, *i.e.*, an early warning that is representative of a problem that is widespread, or alternatively, these may be isolated incidents.

Adverse Event Reports

The following are unedited reports from the Medical Device Reporting database.

Report 1

This report describes four patients. Bupivacaine and adrenaline were infused after total knee procedures. Two patients experienced full thickness sloughs that required plastic surgical intervention and a second procedure with a gastric flap. Two additional patients experienced partial thickness sloughs in the same area, which required split thickness grafts and prolonged nursing and rehabilitation.

Report 2

The pump was attached at the end of surgery for bunionectomy to a healthy 26-yr-old woman. The catheter was placed under the skin and attached to the infusion device (plain Marcaine [AstraZeneca Pharmaceuticals LP, Waltham, MA] was used). This device automatically delivers 2 ml/h continually until the pump is empty. The continuous infusion of this fluid over time caused swelling, pain, and blistering due to cell death. There was ischemic necrosis of the blister with a full-thickness loss of tissue (slough) down to the bone with a question of osteomyelitis. Wound culture was positive for a staphylococcal species. This patient required hospitalization and was sent for wound débridement and intravenous antibiotics. She may require skin grafting.

Report 3

The surgeon placed the pump during a gastrectomy with one regular catheter and one soaker catheter in a 77-yr-old man. A serious infection developed that seemed to originate at the catheter point of entry. The device was in place for approximately 4 days. A severe infection and dehiscence resulting in evisceration developed in the patient. This patient required intervention for a life-threatening adverse event and had to undergo repeated surgery.

Table 1 summarizes characteristics of 40 patients and adverse events from 34 reports to the FDA. These reports described a variety of infusion pump system models manufactured by three companies. Information on patient sex and age was usually not provided. Most surgeries were orthopedic (45.0%), typically total knee. The second most common procedure was for podiatric surgeries (20.0%), including bunionectomies, plantar fasciotomy, and others. The most commonly reported adverse event (42.5%) was tissue necrosis. The drug(s) infused were not reported in most cases (60%), but in the 16 reports that did specify, 9 were bupivacaine and adrenaline and 7 were bupivacaine only. In these 16 cases, necrosis was no more likely when both bupivacaine and adrenaline were reported than when bupivacaine alone was reported (exact test, $P = 0.20$). There were also numerous reports for wound infections and systemic infection.

Discussion

The FDA received 34 reports describing 40 patients with adverse events that occurred during the use of pump systems for direct anesthetic infusion into a sur-

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Table 1. Adverse Event Reports to the FDA for Disposable Continuous Infusion Pumps for Direct Anesthetic Infusion into Surgical Wounds

Description	n = 40	%n
Patient sex		
Male	5	12.5
Female	9	22.5
Not reported	26	65.0
Patient age		
20–40 yr	5	12.5
41–60 yr	4	10.0
61+ yr	3	7.5
Not reported	28	70.0
Surgical procedure		
Orthopedic	18	45.0
Podiatry	8	20.0
Gastrointestinal	6	15.0
Obstetrics–gynecology	2	5.0
Circulatory	1	2.5
Not reported	5	12.5
Adverse event reported*		
Necrosis	17	42.5
Surgical wound infection	15	37.5
Cellulitis	13	32.5
Infection†	10	25.0
Drug infused		
Bupivacaine only	7	17.5
Bupivacaine and adrenaline	9	22.5
Not reported	24	60.0

* More than one adverse event could be reported for each case. † Includes one case of toxic shock syndrome and one case of urinary tract infection, other infections not specified.

FDA = Food and Drug Administration.

gical wound. The most commonly reported complication was tissue necrosis, an adverse event almost never seen after normal procedures. The consequences of these adverse events were typically severe and required intervention and additional medical and surgical treatment. There were also numerous cases of wound infection reported, but these data do not permit us to assess the incidence of this adverse event, so it is not possible to determine whether these infections were above the expected background rate.

We reviewed the literature and found only one case series, published as a letter, that described complications associated with the use of pump systems for direct anesthetic infusion. This case series describes plastic surgery consultations for three patients referred by the same orthopedic surgeon after knee arthroplasties.[8] Two patients needed wound débridements and gastrocnemius flaps, and the third patient underwent repeated débridements and skin grafting. The likely cause of wound ischemia and necrosis was thought to be the infusion of bupivacaine with epinephrine. Reportedly, there was adequate drainage of the wound and no apparent compartment compression. The authors of the letter speculate that the adverse events were related to continuous infusion of epinephrine and subsequent vasoconstriction of the area. In adverse event reports to the FDA, some reports described similar adverse events with use of anesthetic infusion alone. These reports did not explicitly state that epinephrine was not used, and it is possible that epinephrine was used and was not mentioned in the report.

Limitations in Medical Device Reports or MedWatch data should be acknowledged. Device manufacturers, user facilities, and voluntary reporters submit reports to the FDA that are entered into an adverse events database. Accuracy and completeness of reports are not verified by the FDA. The reports do not establish a causal link between a device and an injury or patient problem. Because infection or necrosis may occur after surgery in the absence of continuous anesthetic infusion, it is not possible to definitively conclude that the reported necrosis or infections after surgery was due to infusion pump use, nor is there certainty that continuous infusion of bupivacaine or bupivacaine in combination with epinephrine caused these adverse events. Nevertheless, the reports provide a potentially important signal, suggesting the need for further investigation of the relation between use of pumps for direct continuous infusion of anesthetics and other drugs into surgical wounds and tissue necrosis, serious infections, or cellulitis.

Medical providers and consumers may report adverse events to manufacturers or directly to the FDA through MedWatch.‡§

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§ The FDA Safety Information and Adverse Event Reporting Program. Available at: http://www.fda.gov/medwatch/. Accessed December 9, 2003

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Acute Cardiovascular Instability during Percutaneous Ethanol Injection of a Hepatocellular Carcinoma under General Anesthesia

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HEPATOCELLULAR carcinoma accounts for 4.1% of all cancers, with an estimated 315,000 new cases reported per year. The time of diagnosis determines the type of treatment offered. Current therapeutic modalities include surgical resection, liver transplantation, local ablative techniques, and radiation and systemic therapy.[1]

Local percutaneous intratumoral ablative therapy with ethyl alcohol was first described by Livraghi *et al.*[2] in 1986. Percutaneous ethanol injection has traditionally been performed for hepatocellular carcinomas smaller than 5 cm, although in 1998, larger tumors were reported to have been treated during a single session under general anesthesia.[3] Acute complications seen after percutaneous ethanol injection include bleeding, hemoglobinuria, fever, and inebriation (particularly in nondrinkers). In addition, there have been reports of sudden hypotension immediately after percutaneous ethanol injection therapy.[4,5] Overall mortality associated with percutaneous ethanol injection is 0.6%, primarily as a result of the underlying illness.[4] We report a case of a patient who experienced sudden cardiovascular instability during intraoperative percutaneous ethanol injection and was successfully resuscitated.

Case Report

A 67-yr-old man presented with a 15 × 23 × 24-cm hepatocellular carcinoma involving the right lobe of the liver and a smaller mass in the distal right lobe. Further workup revealed no invasion of the inferior vena cava or portal and suprahepatic veins. Preoperative embolization of the right intrahepatic portal vein was performed to induce hypertrophy of the left hepatic lobe, in anticipation of possible resection of the tumor.

In addition to standard monitors, radial arterial and internal jugular central venous catheters were placed percutaneously. General anesthesia was induced with sodium thiopental, vecuronium, and fentanyl and was maintained with isoflurane in oxygen–air delivered *via* mechanical ventilation.

Laparoscopy was performed to determine the resectability of the tumor. The large size of the tumor and the presence of extensive intraabdominal metastasis made the tumor nonresectable. Ultrasound evaluation of the tumor showed multiple intrahepatic arteriovenous shunts. A suitable nonvascular area in the tumor was identified, and percutaneous ethanol injection was performed. Twenty grams absolute alcohol (20 ml ethanol, 100%) was injected over 20 s under ultrasound guidance. Immediately after the injection, the patient's blood pressure decreased precipitously from 120/80 mmHg to 60/40 mmHg. The electrocardiogram initially showed sinus tachycardia followed by profound sinus bradycardia at a rate of 30–40 beats/min. Twenty-five milligrams ephedrine, 1 mg atropine, and 200 μg phenylephrine were administered.

During cardiopulmonary resuscitation, end-tidal carbon dioxide ($ETCO_2$) was noted to be 12 mmHg. After approximately 1 min, the patient's blood pressure improved to 130/80 mmHg, and cardiopulmonary resuscitation was terminated. Blood gas analysis showed a mixed respiratory–metabolic acidosis (pH, 7.11; partial pressure of carbon dioxide [PCO_2], 68 mmHg; and base excess, −9.4 mEq/l). His blood alcohol level was 50 mg/dl (0.05 mg/%). Sodium bicarbonate was administered to correct the underlying metabolic acidosis, and minute ventilation was adjusted to compensate for the respiratory acidosis.

A repeat blood gas analysis performed 30 min later showed a normalized acid–base status. When hemodynamic stability was achieved, transesophageal echocardiography was performed, which revealed adequate biventricular function, and no wall motion abnormalities or echogenic masses were seen in the pulmonary artery. The patient remained hemodynamically stable and was successfully extubated at the end of surgery. He was then transferred to the surgical intensive care unit for further monitoring. No neurologic deficits were found, and his postoperative period was uneventful.

Discussion

The mechanism of ethanol-induced tumor lysis is due to dehydration of cytoplasmic proteins with subsequent cellular destruction, followed by endothelial cell necrosis and platelet aggregation in small blood vessels, eventually leading to ischemia of the neoplastic tissue.[1]

Routine blood sampling after intrahepatic injection reveals low concentrations of ethanol, suggesting at least partial absorption into the bloodstream. This increase is usually transient and devoid of significant hemodynamic abnormalities. Alternatively, the sudden entry of moderate to large doses of ethanol through the venous circulation may be associated with pulmonary vasoconstriction and increased right ventricular strain. In animal models, the acute intravenous administration of 0.5–1.5 g/kg absolute ethanol increased pulmonary vascular resistance and decreased right ventricular systolic function.[5,6]

A study in healthy physician volunteers showed a significant increase in pulmonary vascular resistance 30 min after the oral ingestion of 0.5 g/kg ethanol diluted to 15%, with return to normal values after 60 min.[5] Although the mechanism of ethanol-induced pulmonary vasoconstriction is not fully understood, there is some evidence that ethanol potentiates hypoxic pulmonary

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vasoconstriction.[7,8] Pulmonary vascular resistance can remain increased for up to 2 h after the ingestion of ethanol,[5] whereas the cardiovascular effects of ethanol seem to be dose dependent. Sarin *et al.*[9] reported a minimal change in pulmonary hemodynamics when using doses of 8–12 ml absolute alcohol for intravariceal sclerotherapy. However, in both animal and human models, doses of 0.5 g/kg or higher are associated with altered pulmonary and systemic hemodyamics.[5,10] The combined effect of an acute increase in pulmonary vascular resistance and negative inotropism can precipitate acute cor pulmonale in susceptible individuals. Patients with primary and secondary pulmonary hypertension may be especially sensitive to the pulmonary vasoconstrictive effects of large doses of intravenous ethanol.

In the absence of other plausible explanations and the temporal correlation with the intrahepatic injection of ethanol, we concluded that our patient's cardiovascular instability most likely was related to ethanol-induced pulmonary vasoconstriction and transient right ventricular dysfunction. Rapid institution of cardiopulmonary resuscitation, use of an indirect sympathomimetic to improve contractility, volume loading to improve ventricular end-diastolic volume, and the use of 100% fraction of inspired oxygen (F_{IO_2}) during resuscitation helped to ameliorate the detrimental effects of ethanol in this patient.

Oxygen supplementation has been shown to attenuate ethanol-induced pulmonary vasoconstriction in an animal model.[7] Hypercarbia, acidosis, and hypothermia should be aggressively treated because of their propensity to increase pulmonary vasoconstriction. Phenylephrine, however, should be used cautiously in this setting because it may exacerbate the existing pulmonary hypertension.[11] In the presence of systemic hypotension, restoration of coronary perfusion pressure with the use of an α_1-adrenoreceptor agonist may offset any effects on the pulmonary circulation and thus improve right ventricular function.[12,13]

In summary, acute pulmonary vasoconstriction should be strongly suspected in the presence of hemodynamic instability shortly after intrahepatic ethanol injection. Clinicians must institute measures to decrease pulmonary vascular resistance and improve right ventricular function immediately to minimize the possibility of cardiovascular collapse. In patients with preexisting pulmonary hypertension, caution should be exercised when using ethanol. Access to pulmonary vasodilators, pulmonary artery catheterization, or transesophageal echocardiography may also be helpful.

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Posterior Inferior Cerebellar Artery Infarction: An Unusual Complication of Posterior Spinal Fusion Surgery in an Adolescent with Idiopathic Scoliosis

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ADOLESCENT idiopathic scoliosis (AIS) is the most common form of scoliosis in the United States.[1] Surgical correction is required in some children.[1,2] Neurologic complications after scoliosis repair are infrequent but

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may be devastating and include complete or partial paraplegia, visual disturbances, and cranial and peripheral nerve injuries.[3] We present the case of a 13-yr-old girl with idiopathic scoliosis in whom a posterior inferior cerebellar artery infarction developed after posterior spinal fusion with instrumentation. Neuroimaging studies obtained postoperatively revealed a previously undiagnosed Chiari I malformation, which we speculate was an essential factor in the etiology of her cerebrovascular event.

Case Report

A 13-yr-old girl (weight, 45 kg; American Society of Anesthesiologists physical status I) with AIS (a right thoracic curve of 57° and a left lumbar curve of 70°) without neurologic symptoms and normal neurologic examination results underwent posterior spinal arthrodesis from T4 to L3 using dual rod fixation with hooks, sublaminar cables, and pedicle screws. General anesthesia was maintained with propofol (100–230 $\mu g \cdot kg^{-1} \cdot min^{-1}$) and remifentanil (0.1–0.9 $\mu g \cdot kg^{-1} \cdot min^{-1}$). Anesthesia and surgery times were 7 h, 9 min and 5 h, 53 min, respectively. Somatosensory and motor evoked potentials were monitored by a neurophysiologist. The operation proceeded uneventfully, with maintenance of mean arterial pressure between 50 and 80 mmHg, hemoglobin concentration of 8.7–12.8 g/dl, and stable somatosensory and motor evoked potentials. The patient's platelet count decreased from 435,000/mm^3 preoperatively to 231,000/mm^3 postoperatively. During the procedure, her estimated blood loss was 1,500 ml, and she received 3,800 ml lactated Ringer's solution, 500 ml cell saver blood, and 217 ml autologous packed erythrocytes. The patient was extubated while awake in the operating room. She was alert and comfortable, followed commands, and moved all four extremities in the recovery room. Postoperative analgesia was managed using patient-controlled analgesia with morphine (demand dose of 0.9 mg, lockout interval of 8 min, continuous infusion 0.9 mg/h, and a 1-h limit of 5.4 mg). On the first postoperative day, the continuous morphine infusion was discontinued and 4 mg ondansetron administered intravenously every 8 h was begun because the patient reported nausea and vomiting. On the second postoperative day, the patient reported occipital headache, generalized pruritus, and severe nausea and vomiting. The patient-controlled analgesia was changed from morphine to hydromorphone (demand dose of 0.2 mg, lockout interval of 8 min, and an hourly maximum of 1 mg) because these reports were attributed to the morphine. Approximately 7 h later, the patient became obtunded, requiring supplemental oxygen to maintain oxyhemoglobin saturation greater than 93%. An anesthesiology resident, believing the patient to be narcotized, administered 400 μg intravenous naloxone, and the demand-only hydromorphone patient-controlled analgesia was reprogrammed (demand dose of 0.1 mg, lockout interval of 8 min, and hourly maximum of 0.5 mg). The patient's mental status improved transiently. She had received 1.3 mg hydromorphone in the previous 7 h. Two hours after receiving naloxone, the patient was noted to be unresponsive, with bilaterally dilated and sluggishly reactive pupils and an absent gag reflex. Arterial blood gas analysis showed a pH of 7.45, a partial pressure of carbon dioxide (P_{CO_2}) of 40 mmHg, and a partial pressure of oxygen (P_{O_2}) of 81 mmHg in room air. Additional lab values included 129 mM Na^+, 156 g/dl glucose, a prothrombin time of 13.3 s, a partial thromboplastin time of 32.4 s, a platelet count of 231,000/mm^3, and 10 g/dl hemoglobin. The level of consciousness improved again with naloxone. Analysis of the patient-controlled analgesia hydromorphone syringe and pump revealed no concentration errors or malfunction in delivery. An emergency computerized tomography scan of the head revealed effacement of basal cisterns and hypodensity in the upper brainstem, consistent with edema and focal ischemia (fig. 1). The patient was transferred to the intensive care unit and treated with diuretics and corticosteroids. Magnetic resonance imaging of her brain (fig. 2) revealed an infarct in the left anterior inferior cerebellum in the distribution of the posterior inferior cerebellar artery. A 10-mm cerebellar tonsillar ectopia was also noted, consistent with Chiari I malformation (fig. 3). Vertebral artery dissection was ruled out by magnetic resonance arteriography. Neurophthalmologic examination revealed left sixth cranial nerve palsy and left hypotropia. The patient's condition improved rapidly over 24 h. Her only complaint at the time of discharge on the sixth postoperative day was diplopia. She is now more than 18 months out from her surgery and completely recovered without neurologic sequelae. Her thoracic and lumbar curves are being maintained at 23° and 12°, respectively.

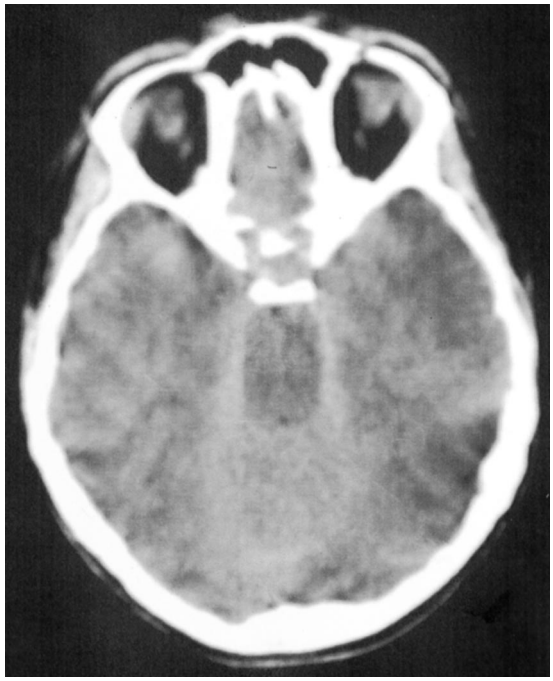

Fig. 1. Computerized tomography of the head revealed effacement of the basal cisterns and hypodensity of the upper brainstem.

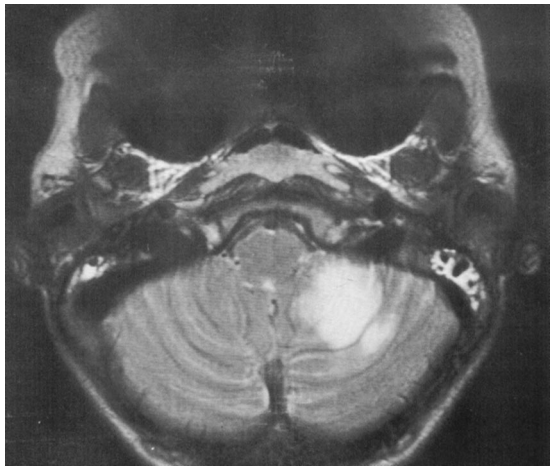

Fig. 2. Magnetic resonance imaging of the head showed a left anterior, inferior cerebellar infarct.

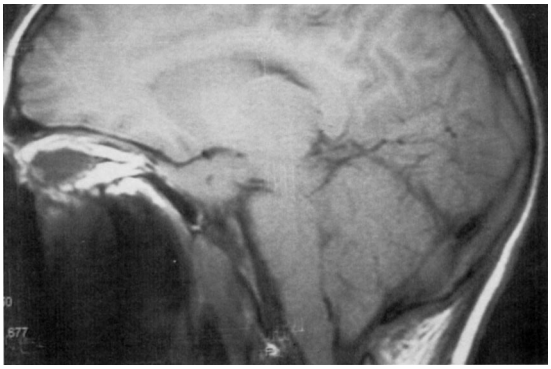

Fig. 3. Magnetic resonance imaging of the head showed a 10-mm cerebellar ectopia, consistent with Chiari I malformation.

Discussion

Stroke has not been previously reported after AIS repair. This patient had severe occipital headache, nausea, and vomiting, which we initially attributed to morphine. In retrospect, we believe these symptoms were signs of a left posterior inferior cerebellar artery infarct. This constellation of symptoms in a child after AIS repair warrants thorough neurologic evaluation.

Chiari I malformations can occur in association with a dural tear and cerebrospinal fluid leak.[4,5] However, we believe this condition preexisted in our patient because there was no dural tear in the operating room. Also, scoliosis is associated with spinal cord and posterior fossa abnormalities, including Chiari I malformation.[5–10] When Chiari I malformation and scoliosis coexist, some recommend the former be repaired by posterior fossa decompression first[6] because scoliosis may stabilize or improve. The indications for imaging the spinal cord and posterior fossa in children with scoliosis vary but generally include patients with abnormal neurologic signs, left-sided curves, high thoracic or cervical curves, painful curves, rapidly progressive curves, and curves that develop before the age of 10 yr.[9] This patient did not have any preoperative indications for magnetic resonance imaging.

The rate of ischemic stroke in the pediatric population is 7.8 in 100,000 children/yr.[11] The etiology of an ischemic stroke includes vertebral artery dissection, emboli, and coagulation abnormalities. This patient did not have clinical or laboratory evidence for a coagulopathy at any time during the perioperative period. Ischemic cerebellar stroke has been reported in a patient with Chiari I malformation.[12] Vertebral artery dissection was ruled out by magnetic resonance arteriography. Both fat and venous air pulmonary emboli have been reported in adolescents undergoing scoliosis surgery,[13–15] and cerebral microemboli were identified by transcranial Doppler in 11 of 13 children aged 13–17 yr who were undergoing surgical correction for scoliosis or kyphosis.[16] However, microembolization was not associated with neurologic sequelae in this report.[16] Although a basilar artery thromboembolic event cannot be ruled out, we speculate that mechanical factors in the perioperative period exacerbated the patient's Chiari I malformation with compression of her left posterior inferior cerebellar artery resulting in infarction. We believe that her symptoms of nausea, vomiting, and headache, which were present soon after surgery, were related to her infarction. Progressive somnolence, unconsciousness, and her ocular findings developed 48 h postoperatively as edema surrounding the infarction enlarged. The delay in diagnosis of her cerebellar infarction was due to her symptoms being the same as those that are commonly attributed to opioid analgesics (nausea, vomiting, and somnolence) and ondansetron (headache).

We present this case to highlight the association of occult posterior fossa abnormalities in children with AIS, to raise awareness of the rare possibility of exacerbating Chiari I malformations during AIS repair and the potential for posterior inferior cerebellar artery compression with cerebellar infarction, to raise awareness of the potential for significant thromboembolic events during scoliosis repair, and to stress the importance of a comprehensive neurologic evaluation in patients with severe occipital headache, nausea, and vomiting after AIS repair.

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Dysphagia, Obstructive Sleep Apnea, and Difficult Fiberoptic Intubation Secondary to Diffuse Idiopathic Skeletal Hyperostosis

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DIFFUSE idiopathic skeletal hyperostosis (DISH) is a common disease of the aging population, characterized by multiple axial and extraaxial involvement.[1] When the spine is affected, calcification and ossification along the anterior surface of several vertebrae can occur.[2] Reports that DISH of the cervical spine impairs management of the upper airway during anesthesia are rare. The authors describe a case of progressive dysphagia, new-onset obstructive sleep apnea, and difficult awake fiberoptic intubation due to anterior cervical osteophyte disease from DISH.

Case Report

A 55-yr-old man[1] (height, 177 cm; weight, 95 kg) was admitted with a 1-month history of progressive weakness and numbness of the lower extremities. His medical history was significant for controlled hypertension, progressive dysphagia for solids and liquids, and new-onset obstructive sleep apnea, consequently requiring continuous positive airway pressure of 12 cm H_2O. A recent sleep study showed a baseline apnea-hypopnea index of 42 (severe obstructive sleep apnea), with marked improvement to 4 when a continuous positive airway pressure of 11 cm H_2O without oxygen supplementation was instituted.

Preoperative cervical magnetic resonance imaging showed neural foraminal narrowing from C3–C4 to C6–C7, mild canal stenosis, and thecal compression at C6–C7. The patient was scheduled for C3–C7 laminectomies and fusion. Preoperative airway examination showed a two-finger breadth mouth opening, a 5-cm mentohyoid distance, and a Mallampati score of II. Neck flexion resulted in dyspnea, whereas neck extension was significantly limited secondary to severe pain. Awake fiberoptic intubation and positioning were planned because of findings on airway examination and the presence of early cervical myelopathy.

Sufentanil and midazolam were used for sedation because of marked preoperative anxiety. These agents were carefully titrated until the patient was somnolent but easily arousable and able to follow commands. His respiratory rate was maintained at greater than 10 breaths/min with an oxygen saturation greater than 95% on room air. Glycopyrrolate was administered preoperatively to decrease oral secretions; atropine was not used because of concerns of resultant tachycardia. Local anesthesia of the upper airway was accomplished with topical lidocaine and superior laryngeal nerve, glossopharyngeal nerve, and transtracheal blocks. Despite multiple attempts at oral fiberoptic laryngoscopy using the Williams airway, neither the epiglottis nor the vocal cords could be identified because of a combination of residual secretions and the unexpected presence of a large soft tissue mass extending from the posterior pharyngeal wall to the base of the tongue. The presence of a large anterior mass had not been detected during evaluation of the preoperative cervical radiographs. Direct laryngoscopy and light wand intubation was attempted without success. After multiple attempts at intubation, the decision was made to postpone the procedure, and the patient was admitted to the intensive care unit for observation because of concerns of airway edema.

The cervical radiographs were reevaluated. A large anterior osteophyte extending from C2 to C6—more pronounced at the C3–C4 level—was considered the culprit. The diagnosis of DISH was made (fig. 1).

The patient returned to the operating room a week later. He was premedicated with glycopyrrolate and atropine. After confirming the lack of oral secretions, local anesthesia of the upper airway was accomplished in a similar fashion. It was also decided to provide only minimal sedation to preserve full cooperation from the patient. Fiberoptic laryngoscopy confirmed the soft tissue mass. The epiglottis was noted to be deviated to the left of the midline, and the vocal cords could only be visualized briefly with deep inspiration. Intubation was difficult but accomplished successfully with a 7.0 endotracheal tube lubricated with lidocaine gel. Surgery proceeded uneventfully, and the postoperative course was uncomplicated.

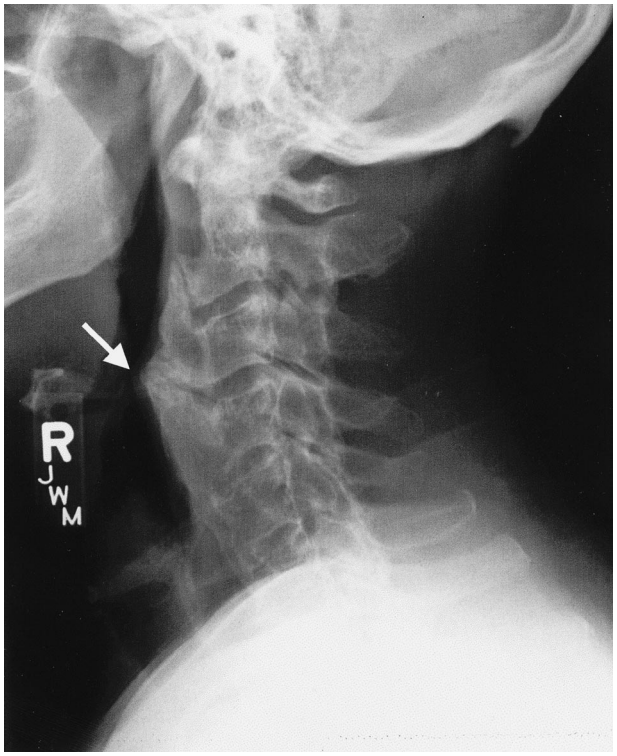

Fig. 1. Lateral C-spine radiograph showing extensive osteophytic formation from C2 to C6. The greatest protrusion occurs at C3–C4, resulting in significant narrowing of the upper airway (*arrow*).

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The patient returned 2 months later for anterior osteophytectomy. Awake fiberoptic intubation with minimal sedation and patient cooperation was performed successfully. Postoperatively, the patient had resolution of his dysphagia and improvement in his obstructive sleep apnea and continuous positive airway pressure requirements. Postoperative sleep studies were not obtained.

Discussion

Degenerative disease of the cervical spine is relatively common after the age of 40 yr and affects a significant number of patients after the seventh decade of life. The three most common causes of cervical osteophytosis are ankylosing spondylitis, DISH, and degenerative disc disease.

The axial distribution of DISH primarily involves the thoracic spine, followed in frequency by the lumbar and cervical spine. When the cervical spine is involved, coexisting ossification of the posterior longitudinal ligament is detected in approximately 50% of patients and may be responsible for spinal canal narrowing and neurologic deficits.[3] Cervical spine alteration is most common between the fourth and seventh vertebral bodies.

Extensive cervical hyperostosis that can be associated with DISH may result in symptoms related to compression, distortion, or both of the aerodigestive tract, including dysphagia, stridor, dyspnea, and obstructive sleep apnea.[4–7] Reports of difficult intubation are surprisingly rare. The presence of an enlarged cervical osteophyte may lead to edema of the laryngeal inlet, aggravating the mechanical obstruction.[8]

Our patient's cervical radiograph shows the presence of a large contiguous osteophyte involving the C2–C6 area. The apex of the osteophyte lies adjacent to the epiglottis, and the airway seems markedly narrow at this point, thus making fiberoptic visualization of the larynx and placement of the endotracheal tube difficult. Deep sedation during the initial fiberoptic intubation attempt prevented the patient from inspiring maximally, which was needed to optimally locate the laryngeal inlet. With subsequent fiberoptic intubation attempts, a lighter level of sedation was chosen, and the patient was instructed to inspire deeply during advancement of the fiberoptic scope, beyond the area of critical narrowing. In addition, more aggressive use of antisialogogue agents facilitated subsequent attempts. In patients with obstructive sleep apnea, adipose tissue deposits in the lateral walls of the pharynx, which is the most important site of collapse during deep sleep and when central nervous system depressants are used.[9] With deep sleep or sedation, upper airway muscle tone decreases and upper airway resistance increases, resulting in generation of subatmospheric pressure in the pharynx and ultimate pharyngeal collapse.[9]

The presence of excessively large cervical osteophytes, particularly those located adjacent to areas of normal esophageal fixation, may also impair swallowing.[10–12] There is also an increased risk for aspiration pneumonitis because of the altered anatomy of the hypopharynx.

Cervical dysphagia may improve with conservative therapy; however, the definitive management is surgical resection of the osteophyte. Obstructive sleep apnea has been described when large cervical osteophytes are present in combination with relaxation of the hypopharyngeal musculature in the supine position.[13] Continuous positive airway pressure devices relieve the obstruction caused by these large osteophytes during inspiration.

The presence of dysphagia, dyspnea, or sleep apnea, as in our patient, should alert the clinician to possible upper airway obstruction from large anterior cervical osteophytes. This mechanical distortion may complicate both direct laryngoscopy and intubation. Thorough perioperative preparation and radiographic imaging is necessary to manage this condition successfully.

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