Anesthesiology 2008; 108:325–8

Severe Brachial Plexopathy after an Ultrasound-guided Single-injection Nerve Block for Total Shoulder Arthroplasty in a Patient with Multiple Sclerosis

Matthew D. Koff, M.D., M.S., Jeffrey A. Cohen, M.D.,† John J. McIntyre, M.D.,‡ Charles F. Carr, M.D.,§ Brian D. Sites, M.D.‖*

DESPITE the known benefits of regional anesthesia for patients undergoing joint arthroplasty, the performance of peripheral nerve blocks in patients with multiple sclerosis (MS) remains controversial. MS has traditionally been described as an isolated disease of the central nervous system, without involvement of the peripheral nerves, and peripheral nerve blockade has been suggested to be safe.[1,2] However, careful review of the literature suggests that MS may also be associated with involvement of the peripheral nervous system, challenging traditional teachings. There is a paucity of evidence with regard to safety in using peripheral nerve regional anesthesia in these patients. This makes it difficult to provide adequate "informed consent" to these patients. This case report describes a patient with MS who sustained a severe brachial plexopathy after a total shoulder arthroplasty during combined general anesthesia and interscalene nerve block.

Case Report

A 65-yr-old right-hand-dominant man, American Society of Anesthesiologists physical status III, presented for a right total shoulder arthroplasty secondary to osteoarthritis. The patient's medical history was significant for hypothyroidism, benign prostatic hypertrophy, mitral valve prolapse, and MS. His medications included 40 mg pravastatin by mouth daily, 75 μg levothyroxine by mouth daily, and 15 mg oxybutynin (extended release) by mouth daily. The patient was allergic only to oysters, which had caused anaphylaxis in the past. Although without clinical changes for 2 yr, his MS was remarkable for bilateral lower extremity weakness (walker needed for ambulation) and the requirement for self–urethral catheterization.

After informed consent, the patient underwent an interscalene nerve block and general anesthetic. In the preanesthetic block room, sedation was provided with 50 μg intravenous fentanyl and 2 mg intravenous midazolam. An ultrasound-guided "single-shot" injection using an in-plane needle approach and nerve stimulation was performed. The injection was made at the mid-neck level at the nerve roots of the brachial plexus. The needle direction was in reference to the middle scalene muscle from the lateral toward medial direction. Three injections were made starting laterally on C5, then anteriorly on C5, and then medially to C5. The injections were made to create circumferential spread around the roots of the brachial plexus. The injection was performed as previously described.[3,4] A 50-mm, 22-gauge b-bevel (B. Braun Medical, Bethlehem, PA) was inserted in plane with the ultrasound beam during visualization of the roots of the brachial plexus on short axis. The needle (stimulating at 0.45 mA, 0.1-ms pulse duration, 2 Hz) was directed until it approached the outer edge of the C5 nerve root. The needle was not seen to penetrate the epineurium by our ultrasound image (fig. 1). After the demonstration of biceps contraction, an injection of 30 ml bupivacaine (0.5%), 1:400,000 epinephrine, and 50 μg clonidine was injected using a 10-ml Luer-Lok controlled stroke syringe. The local anesthetic was noted to surround the C5–C6 nerve roots. The needle was repositioned three times to generate complete coverage of the C5–C6–C7 roots. During the procedure, the patient experienced no discomfort, and there was not resistance to injection. The block was checked for success by the senior regional resident. This patient was noted to have partial sensory (to ice) blockade over the anterior shoulder (axillary nerve distribution C5) and partial motor (by strength testing) and sensory (to ice) blockade of the musculocutaneous nerve distribution 10 min after regional blockade.

After the induction and maintenance of general endotracheal anesthesia, the patient was placed in the beach chair position. Consistent with the sitting position, an episode of hypotension (77/46 mmHg, mean arterial pressure 56 mmHg) was noted after induction and patient repositioning to the sitting position. The patient initially required a total administration of 2 l incrementally of lactated Ringer's solution and a total of 15 mg ephedrine (in 5-mg dosing increments) to return to a mean arterial pressure greater than 70 mmHg. Intraoperatively, the patient's temperature ranged from approximately 35 to 36.4 centigrade. The arm was held in place by the Spider Arm Retractor (Tenet Medical Engineering, Calgary, Alberta, Canada). A Zimmer anatomic total shoulder system was used (Zimmer Inc., Warsaw, IN). During placement of the glenoid component, the arm was positioned in 35° of external rotation and 45° of abduction. The estimated blood loss was 400 ml, and the patient received 2,800 ml lactated Ringer's solution. Surgical time was 3 h 45 min. After emergence in the postanesthesia recovery unit, the patient was noted by nursing staff to have a dense motor and sensory block and was also noted to be comfortable for the first hour. The patient then began to report right arm pain that was described as burning in quality. It was rated as 5 out of 10 on a visual analog scale. A neurologic examination was performed by the operating orthopedic resident within 4 h postoperatively. At that time, the

This article is accompanied by an Editorial View. Please see: Hebl JR: Ultrasound-guided regional anesthesia and the prevention of neurologic injury: Fact or fiction? ANESTHESIOLOGY 2008; 108:186–8.

* Resident, ‖ Assistant Professor in Anesthesiology, Department of Anesthesiology, † Associate Chief in Neurology, Department of Neurology, ‡ Assistant Professor and Director of Spinal Imaging, Section of Neuroradiology, Department of Radiology, § Associate Professor in Orthopedics and Director of Sports Medicine, Department of Orthopedics, Dartmouth-Hitchcock Medical Center.

Received from the Department of Anesthesiology, Dartmouth-Hitchcock Medical Center, Lebanon, New Hampshire. Submitted for publication June 26, 2007. Accepted for publication September 25, 2007. Support was provided solely from institutional and/or departmental sources. Sonosite, Seattle, Washington, and Phillips, Atlanta, Georgia, provided ultrasound equipment for educational and research purposes.

Address correspondence to Dr. Koff: Department of Anesthesiology, One Medical Center Drive, Lebanon, New Hampshire 03756-0001. matthew.d.koff@hitchcock.org.

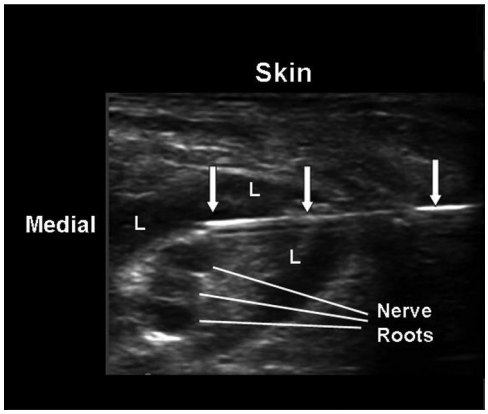

Fig. 1. Interscalene nerve block in our patient with multiple sclerosis. Image shows the C5–C7 ventral roots of the right brachial plexus. *Arrows* indicate the needle in plane with the ultrasound beam. The needle tip was completely visualized throughout the procedure. *L* indicates local anesthetic completely surrounding nerve roots, *i.e.*, "donut sign."

patient was again noted to have a dense motor and sensory block of the operative extremity, as would be expected 10 h after a successful regional blockade.

On postoperative day 1, the patient continued to have shoulder pain with a persistent flaccid motor block of his entire right upper extremity. This pain was exacerbated by shoulder and arm movement and not by neck movement or a Valsalva maneuver, as can be seen in cervical radiculopathy. A consultation by the neurology service on postoperative day 2 found sensation to temperature throughout dermatomes C4–T1, with absent light touch sensation in C6–T1. Vibration and joint position perception were absent throughout. A magnetic resonance image of the chest was performed on postoperative day 3, which demonstrated postsurgical changes without any evidence for compressive or avulsive pathology. However, it was diagnostic for brachial neuritis (fig. 2). High-dose methylprednisolone was initiated to treat a presumed autoimmune brachial neuritis. An electromyelogram performed on postoperative day 4 showed loss of the median and ulnar F waves. In addition, there was no voluntary recruitment of the following muscles: deltoid, triceps, biceps, brachioradialis, wrist extensors, and first dorsal interosseous. At this time, there was no evidence of active denervation in any of the muscles examined. On postoperative day 11, a complete paresis of the patient's entire arm persisted; an electromyelogram demonstrated active denervation of all muscles and no voluntary motor recruitment. This study demonstrated low-amplitude compound muscle action potentials of the median and ulnar motor nerves. Median ulnar and radial sensory nerve action potentials were absent. Electromyographic examination revealed active denervation in all of the muscles previously examined, with no voluntary motor recruitment (table 1).

A follow-up electromyelogram 3 months from the date of surgery showed improvement. There was reduced voluntary motor recruitment with evidence of reinnervation in all of the muscles that were previously examined. The patient's unaffected limbs were tested, and studies of the radial and sural sensory nerves and ulnar and peroneal motor nerves with F waves yielded normal results. Nerve fiber loss can still be significant despite normal nerve conduction study results. Therefore, a normal electromyelogram does not completely rule out subclinical peripheral neuropathy.

At 8 months postoperatively, the patient continued to have significant range of motion and strength deficits. His distal hand function remained limited secondary to stiffness from the prolonged neurologic recovery. Range of motion at the wrist, metacarpophalangeal joints, proximal interphalangeal joints, and distal interphalangeal joints were significantly limited, with approximately 50% loss of motion at each level. The patient also continued to have visible isolated muscle atrophy of proximal musculature, including the pectoralis major and pos-

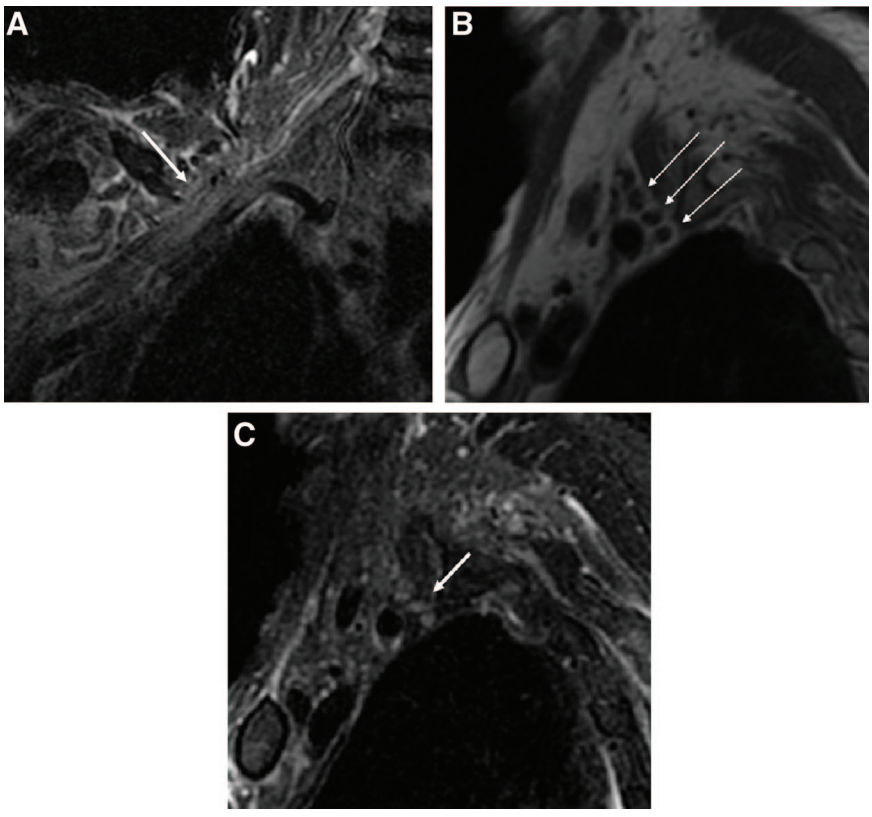

Fig. 2. (*A*) T2 coronal image demonstrating increased signal intensity of the right brachial plexus (*arrow*). (*B*) Sagittal T1 image demonstrating swelling of the brachial plexus (*arrows*) posterior and superior to the subclavian artery flow void. (*C*) Sagittal T2 image demonstrating increased girth and increased signal of the brachial plexus (*arrow*) posterior and superior to the subclavian artery flow void.

Table 1. Needle Electromyography of Patient on Postoperative Day 11

	Spontaneous Activity			Volitional Activity		
Muscle	Fibrillations	+Waves	Polyphasic	Amplitude	Duration	Recruitment
Deltoid, right	2+	2+	Increased	Increased	Increased	Single
Biceps, right	1+	1+	No	Voluntary	Motor	Units
Triceps, right	1+	1+	No	Voluntary	Motor	Units
Brachioradialis, right	1+	1+	No	Voluntary	Motor	Units
FDI, right	0	0	No	Voluntary	Motor	Units
FPL, right	1+	1+	No	Voluntary	Motor	Units
EDC, right	0	0	No	Voluntary	Motor	Units
Trapezius, right	0	0	0	Normal	Normal	Full

EDC = extensor digitorum complex; FDI = first dorsal interossei; FPL = flexor pollicis longus.

terior deltoid. His final diagnosis was an inflammatory brachial neuritis (IBN).

Discussion

Multiple sclerosis is described as a chronic disease of the central nervous system that usually begins in young adults. Pathologically, MS is characterized by multiple areas of central nervous system white matter inflammation, demyelination, and glial scarring or sclerosis.[5] The clinical course of MS varies from a benign, largely symptom-free disease to a rapidly progressive and disabling disorder. The etiology of MS is likely due to autoimmune mechanisms, possibly triggered by infectious and other environmental factors in genetically susceptible individuals.[6]

Controversy exists in providing regional anesthesia to patients with neurologic diseases. The "double-crush" phenomenon suggests that patients with preexisting neural compromise may be more susceptible to injury at another site when exposed to a secondary injury.[7] The performance of a neuraxial technique in patients with preexisting central nervous system disorders may increase the risk of a double-crush phenomenon.[8] In contrast to a spinal or epidural block, a peripheral nerve block in MS patients is theoretically attractive because the neural pathology is presumed to be located in the central nervous system. However, this association seems to be incomplete and is based on the fact that the clinical involvement of the peripheral nervous system in MS patients has traditionally been ignored by modern textbooks. This is despite the fact that the description of this link dates back a half century.[9] Importantly, this conventional teaching is also present in the anesthesia literature.[1,2,10–13] Careful assessment of the literature reveals that multiple recent studies have shown the existence of subclinical peripheral neuropathy in some patients with MS.[14–18] Pogorzelski *et al.*[14] noted both sensory and peripheral motor nerve lesions of a demyelinating-axonal character. They also noted that sensory abnormalities were more pronounced than motor ones. Another study found electrophysiologic abnormalities in the 14.7% of all peripheral nerves examined (n = 244) in patients with MS.[17] This is well above the reported prevalence of 2.4% in the general population. In the elderly, the prevalence is reported to be as high as 8%, mostly due to diabetes mellitus.[19] Hughes *et al.*[20] described an association of a demyelinating peripheral neuropathy in MS patients. Other inflammatory demyelinating diseases exist that have both central and peripheral components, such as chronic inflammatory demyelinating polyneuropathy.[21]

Patients with underlying peripheral neurologic disorders may be more susceptible to nerve injury with the use of regional techniques.[22] Despite testing modalities such as electromyography and magnetic resonance imaging, it may be difficult to differentiate between multiple etiologies, including direct trauma during the regional procedure, neurotoxicity from local anesthetics (and additives), and patient positioning, such as extreme abduction and external rotation, which has been implicated in surgical stretch injury of the brachial plexus. All of these could occur in a patient undergoing total shoulder replacement. The other confounding variable in diagnosing the etiology of a postoperative neurologic deterioration is that the clinical course of MS may be exacerbated from many nontraumatic-related reasons, such as hyperthermia, electrolyte abnormalities, stress, and pain.

Brachial plexus injury after total shoulder arthroplasty has been estimated at 2.8%.[23] To our knowledge, this is the first report of an IBN after total shoulder replacement in a patient with MS. This is also the first report of IBN in a patient using an ultrasound-guided regional anesthesia technique. Brachial plexus injury after interscalene nerve blockade has been previously described.[24] IBN has also been reported to occur in patients during treatment for MS.[18] IBN is a well-recognized clinical syndrome characterized by brachial pain followed by patchy atrophy of muscles in the shoulder girdle and arm innervated by individual branches of the brachial plexus.[25–27] Postsurgical IBN has not been widely recognized since Parsonage and Turner's original description.[27]

In summary, we report a case of a severe brachial plexus injury that occurred in a patient with MS after a

total shoulder replacement during combined general anesthesia and interscalene nerve block. Although the mechanisms of this injury are unclear, the potential preexisting pathology of the peripheral nervous system may have contributed. It is possible that this patient preoperatively had an occult peripheral neuropathy, and his underlying MS predisposed him to development of a peripheral autoimmune injury leading to a brachial neuritis. The individual decision to perform peripheral regional anesthesia in a patient with MS must rest on the perceived benefits of avoiding non–opioid-based analgesia and/or avoiding general anesthesia. Anesthesiologists should recognize that the peripheral nervous system may also be abnormal in patients with MS.

References

1. Horlocker TT: Concurrent medical problems and regional anesthesia, Regional Anesthesia and Analgesia. Edited by Brown DL. Philadelphia, WB Saunders, 1996, pp 423–45
2. Jankowski CJ: Neuraxial anesthesia techniques, Textbook of Regional Anesthesia. Edited by Raj PP. Philadelphia, Churchill Livingstone, 2002, pp 829–52
3. Chan VW: Applying ultrasound imaging to interscalene brachial plexus block. Reg Anesth Pain Med 2003; 28:340–3
4. Sites BD, Brull R: Ultrasound guidance in peripheral regional anesthesia: Philosophy, evidence-based medicine, and techniques. Curr Opin Anaesthesiol 2006; 19:630–9
5. Love S: Demyelinating diseases. J Clin Pathol 2006; 59:1151–9
6. Sadiq SA: Multiple sclerosis, Merritt's Neurology, 11th edition. Edited by Rowland LP. Philadelphia, Lippincott Williams & Wilkins, 2005, pp 941–63
7. Hebl JR, Horlocker TT, Pritchard DJ: Diffuse brachial plexopathy after interscalene blockade in a patient receiving cisplatin chemotherapy: The pharmacologic double crush syndrome. Anesth Analg 2001; 92:249–51
8. Hebl JR, Horlocker TT, Schroeder DR: Neuraxial anesthesia and analgesia in patients with preexisting central nervous system disorders. Anesth Analg 2006; 103:223–8
9. Wilson S: Neurology. Baltimore, Williams & Wilkins, 1940, p 170
10. Dierdorf SF, Walton JS: Anesthesia for patients with rare and coexisting diseases, Clinical Anesthesia, 5th edition. Edited by Barash PG, Cullen BF, Stoelting RK. Lippincott Williams & Wilkins, Philadelphia, 2006, pp 502–28
11. Naguib M, Lien CA: Pharmacology of muscle relaxants and their antagonists, Miller's Anesthesia, 6th edition. Edited by Miller RD. Philadelphia, Elsevier Churchill Livingstone, 2005, pp 481–572
12. Schneider KM: AANA Journal course: Update for nurse anesthetists—an overview of multiple sclerosis and implications for anesthesia. AANA J 2005; 73:217–24
13. Stoelting RK, Dierdorf SF: Diseases of the nervous system, Anesthesia and Co-existing Disease, 4th edition. Edited by Stoelting RK, Dierdorf SF. Philadelphia, Churchill Livingstone, 2002, pp 233–98
14. Pogorzelski R, Baniukiewicz E, Drozdowski W: Subclinical lesions of peripheral nervous system in multiple sclerosis patients [in Polish]. Neurol Neurochir Pol 2004; 38:257–64
15. Poser CM: The peripheral nervous system in multiple sclerosis: A review and pathogenetic hypothesis. J Neurol Sci 1987; 79:83–90
16. Rubin M, Karpati G, Carpenter S: Combined central and peripheral myelinopathy. Neurology 1987; 37:1287–90
17. Sarova-Pinhas I, Achiron A, Gilad R, Lampl Y: Peripheral neuropathy in multiple sclerosis: A clinical and electrophysiologic study. Acta Neurol Scand 1995; 91:234–8
18. Walker M, Zunt JR, Kraft GH: Brachial neuropathy after immunosuppression and stem cell transplantation for multiple sclerosis. Mult Scler 2005; 11:90–1
19. Hughes RA: Peripheral neuropathy. BMJ 2002; 324:466–9
20. Hughes R: Demyelinating neuropathy, Handbook of Clinical Neurology. Edited by Koetsier J. Amsterdam, Elsevier Science Publishers, 1985, pp 605–27
21. Koller H, Kieseier BC, Jander S, Hartung HP: Chronic inflammatory demyelinating polyneuropathy. N Engl J Med 2005; 352:1343–56
22. Blumenthal S, Borgeat A, Maurer K, Beck-Schimmer B, Kliesch U, Marquardt M, Urech J: Preexisting subclinical neuropathy as a risk factor for nerve injury after continuous ropivacaine administration through a femoral nerve catheter. ANESTHESIOLOGY 2006; 105:1053–6
23. Lynch NM, Cofield RH, Silbert PL, Hermann RC: Neurologic complications after total shoulder arthroplasty. J Shoulder Elbow Surg 1996; 5:53–61
24. Walton JS, Folk JW, Friedman RJ, Dorman BH: Complete brachial plexus palsy after total shoulder arthroplasty done with interscalene block anesthesia. Reg Anesth Pain Med 2000; 25:318–21
25. Fibuch EE, Mertz J, Geller B: Postoperative onset of idiopathic brachial neuritis. ANESTHESIOLOGY 1996; 84:455–8
26. Gonzalez-Alegre P, Recober A, Kelkar P: Idiopathic brachial neuritis. Iowa Orthop J 2002; 22:81–5
27. Malamut RI, Marques W, England JD, Sumner AJ: Postsurgical idiopathic brachial neuritis. Muscle Nerve 1994; 17:320–4

Anesthesiology 2008; 108:328–9

Ethanol-induced Coma after Therapeutic Ethanol Injection of a Hepatic Cyst

Anne Wernet, M.D., Annie Sibert, M.D.,† Catherine Paugam-Burtz, M.D.,* Arnaud Geffroy, M.D.,‡ Sebastian Pease, M.D.,* Jacques Belghiti, M.D.,§ Valérie Vilgrain, M.D.,‖ Jean Mantz, M.D., Ph.D.#*

HEPATIC cyst is a common congenital malformation, the incidence of which varies from 0.1% to 4.5%.[1,2] Hepatic cysts are most often asymptomatic. Clinical symptoms comprise abdominal compression revealed by abdominal pain, gastric satiety, vomiting, biliary compression with jaundice, dilatation of biliary ducts or cholestasis, and vascular compression of the inferior vena cava or hepatic vessels.[3] Cyst sclerotherapy may be required in such cases, as well as in intracystic hemorrhage. Sclerotherapy is usually performed by percutaneous ethanol injection *in situ* into the cyst. Such therapy is currently recommended for the treatment of symptomatic hepatic cyst, because of its efficiency and the absence of reported severe complications.[4] Here, we report the original case of a patient who demonstrated ethanol-induced coma requiring mechanical ventilation after ethanol injection of a symptomatic hepatic cyst.

* Staff Anesthesiologist, # Professor and Head, Department of Anesthesia and Intensive Care, † Staff Radiologist, ‖ Professor and Head, Department of Imaging, § Professor and Head, Department of Hepatobiliary Surgery, Beaujon University Hospital, Assistance Publique des Hôpitaux de Paris, Clichy, France. ‡ Staff Anesthesiologist, Department of Anesthesia and Intensive Care, Bichat-Claude Bernard University Hospital, Assistance Publique des Hôpitaux de Paris, Paris, France.

Received from the Department of Anesthesia and Intensive Care, Beaujon University Hospital, Assistance Publique des Hôpitaux de Paris, Clichy, France. Submitted for publication August 8, 2007. Accepted for publication September 19, 2007. Support was provided solely from institutional and/or departmental sources.

Address correspondence to Dr. Wernet: Service d'anesthésie et de réanimation chirurgicale, Hôpital Beaujon, Assistance Publique des Hôpitaux de Paris, 100 boulevard du Général Leclerc, 92110 Clichy, France. anne.wernet@bjn.aphp.fr.

Case Report

A 69-yr-old woman (168 cm, 65 kg) was admitted to the recovery room after ethanol injection of a hepatic cyst performed during general anesthesia. Her medical history included arterial hypertension. Medication was bisoprolol. She reported no alcohol consumption. Intracystic hemorrhage of a 22 × 20 × 15-cm hepatic cyst located to the right lobe occurred 3 weeks before admission and led to a decision to treat the cyst by *in situ* ethanol injection. General anesthesia was provided by continuous infusion of intravenous propofol while the patient was spontaneously breathing an oxygen–air mixture (6 l/min; fraction of inspired oxygen [F_{IO_2}], 0.5) delivered *via* a facemask tightly connected to the face. The patient was monitored with an electrocardioscope, a noninvasive blood pressure device, a pulse oximeter, and an end-tidal carbon dioxide measurement device. The procedure was performed by an experienced radiologist, under sonographic guidance. Cystic puncture was performed with a pigtail catheter, and 3,500 ml fluid was evacuated. Postevacuation opacification ruled out communication between the cyst and the biliary tree, and 240 ml ethanol, 95%, was injected into the cyst cavity. The patient was then positioned alternately on left and right lateral decubitus to allow ethanol to reach the maximum area of the cyst cavity. Fifty minutes later, the same quantity of liquid was removed from the cyst, and the procedure ended uneventfully. The total dose of propofol delivered to the patient was 210 mg. No additional anesthetic or opioid was administered during the procedure. The patient was able to properly respond to verbal command and was discharged to the postanesthesia care unit. Shortly after arrival in postanesthesia care unit, the patient developed lethargy and became unresponsive. Her breath smelled of alcohol. Consciousness rapidly deteriorated and was followed by a coma scored as 3 on the Glasgow Coma Scale. The trachea was intubated, and mechanical ventilation was initiated (F_{IO_2}, 0.4; tidal volume, 650 ml; respiratory rate, 12 breaths/min). An ethanol-induced coma was suspected and confirmed by measurement of the patient's blood ethanol level, which was 3.10 g/l. The patient progressively recovered satisfactory consciousness and was extubated 11 h after the procedure. Her ethanol blood levels were 1.88 g/l at hour 7 and 0.27 g/l at hour 15 after the procedure. The patient was discharged uneventfully from the institution 2 days later.

Discussion

We report here a massive ethanol intoxication leading to coma after ethanol sclerosis of a hepatic cyst. To our knowledge, this is the first description of severe ethanol-induced coma after ethanol injection of a hepatic cyst.

Mild alcoholemia-related clinical signs after hepatic cyst alcoholization have been scarcely published, and no alcoholemia-related morbidity has been described. Maximal ethanol blood levels up to 1.02 g/l have been reported 1 h after the procedure.[5,6] Hepatic cysts are avascular tumors. Systemic absorption of ethanol may therefore have occurred *via* two pathways. At first, ethanol could have entered biliary ducts and then the gut *via* transmural absorption by mesenteric blood vessels. However, the demonstration of absence of communication between the hepatic cyst and biliary ducts after opacification likely rules out such a scenario in our case. Similarly, the delayed onset of symptoms, with respect to the time of ethanol administration, is hardly consistent with an accidental vascular injection. More likely, ethanol was directly absorbed through the cyst wall formed by an epithelium which resembles biliary epithelium and a stroma, made of a thin layer of connective tissue.[7] The giant size of the cyst, the large volume of ethanol used,[8] and the long time in contact surely contributed to this unusually high absorption rate. The alcoholic smell of the patient's breath was rapidly detected postoperatively, supporting ethanol as the cause of the coma. The diagnosis was further confirmed by measurement of the ethanol blood level. The rapid decrease in ethanol blood level after the procedure was consistent with the fact that excessive ethanol absorption had occurred both intraoperatively and in the early postoperative period.

Conclusion

Ethanol-induced coma must be considered in the absence of recovery, or deterioration of consciousness after apparently normal awakening, after ethanol injection of a hepatic cyst performed during general anesthesia. Anesthesiologists as well as radiologists should be aware of this rare but potentially life-threatening complication. A limited volume of injected ethanol is warranted. Ethanol levels should be assessed in the early postoperative stage.

References

1. Bruneton JN, Eresue J, Caramella E, Drouillard J, Roux P, Fenart D: Congenital cysts of the liver in echography [in French]. J Radiol 1983; 64:471–6
2. Caremani M, Vincenti A, Benci A, Sassoli S, Tacconi D: Echographic epidemiology of non-parasitic hepatic cysts. J Clin Ultrasound 1993; 21:115–8
3. Azizah N, Paradinas FJ: Cholangiocarcinoma coexisting with developmental liver cysts: A distinct entity different from liver cystadenocarcinoma. Histopathology 1980; 4:391–400
4. Montorsi M, Torzilli G, Fumagalli U, Bona S, Rostai R, De Simone M, Rovati V, Mosca F, Filice C: Percutaneous alcohol sclerotherapy of simple hepatic cysts: Results from a multicentre survey in Italy. HPB Surg 1994; 8:89–94
5. Leinonen A, Siniluoto T, Päivänsalo M, Karttunen A, Kairaluoma M, Suramo I: Percutaneous aspiration and ethanol sclerotherapy of symptomatic hepatic cysts. Eur Radiol 1993; 3:213–8
6. vanSonnenberg E, Wroblicka JT, D'Agostino HB, Mathieson JR, Casola G, O'Laoide R, Cooperberg PL: Symptomatic hepatic cysts: percutaneous drainage and sclerosis. Radiology 1994; 190:387–92
7. Balli M, Zhao M, Zimmermann A: Polycystic liver disease: Immunohistochemical characterization of cyst epithelia and extracellular matrix. Contrib Nephrol 1995; 115:127–33
8. Bean WJ, Rodan BA: Hepatic cysts: Treatment with alcohol. AJR Am J Roentgenol 1985; 144:237–41