Undersea Biomedical Research, Vol. 19, No. 3, 1992

Management of herniated intervertebral disks during saturation dives: a case report

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Stevens DM, Caras BG, Flynn ET, Dutka AJ, Thorp JW, Thalmann ED. Management of herniated intervertebral disks during saturation dives: a case report. Undersea Biomed Res 1992; 19(3):191–198.—During research saturation dives at 5.0 and 5.5 atm abs, 2 divers developed an acute herniation of the nucleus pulposus of the L5-S1 intervertebral disk. In both cases the pain was severe enough to require intravenous morphine or intramuscular meperidine. Although the symptoms presented by these divers are frequently considered to be an indication for immediate surgical consultation, we decided that emergency decompression posed an unacceptable risk that decompression sickness (DCS) would develop in the region of acute inflammation. In both cases strict bedrest and medical therapy were performed at depth. In the first case, 12 h was spent at depth before initiating a standard U.S. Navy saturation decompression schedule with the chamber partial pressure of oxygen elevated to 0.50 atm abs. In the second case, a conservative He-N_2-O_2 trimix decompression schedule was followed to the surface. In both cases, no initial upward excursion was performed. The required decompression time was 57 h 24 min from 5.5 atm abs and 55 h 38 min from 5.0 atm abs. During the course of decompression, the first diver's neurologic exam improved and he required decreasing amounts of intravenous narcotic; we considered both to be evidence against DCS. The second diver continued to have pain and muscle spasm throughout decompression, however he did not develop motor, reflex, or sphincter abnormalities. Both divers have responded well to nonsurgical therapy.

decompression sickness
inflammation
herniated nucleus pulposus
morphine
previous injury

While working on an underwater cycle ergometer during He-O_2 research saturation dives at 5.0 and 5.5 atm abs, 2 divers developed lower back pain, and one had accompanying sensory and motor abnormalities characteristic of an acute herniation of the nucleus pulposus (HNP) of the L5-S1 intervertebral disk. When the divers' condition deteriorated, we had to develop a method to bring them to the surface as rapidly as possible (in case surgery was needed) without incurring damage to the already-injured area from decompression sickness (DCS). Information in the litera-

ture indicated that DCS might be more likely to develop in the region of acute inflammation than in normal tissue (1, 2). We were also concerned that if DCS should occur, its symptoms might be difficult to evaluate due to the existing neurologic deficits from compression of nerve roots by the HNP and because narcotic analgesics were being used. This report describes our management of these cases.

CASE HISTORIES

Case 1

On Dive Day 4 of a research-oxygen saturation dive in a Man-Rated Chamber Complex (MRCC), a 33 yr-old black male, U.S. Navy diver developed acute lower back pain with radiation to both buttocks while riding a cycle ergometer immersed in a wet pot at a chamber pressure of 5.5 atm abs [equivalent to 150 feet of seawater (fsw)]. The diver had a history of episodic, mild lower back pain that never required hospitalization. The previous episodes had responded to conservative treatment with anti-inflammatory and muscle relaxant medication. He was treated successfully 5 mo. earlier for type II DCS, which included hypesthesias in the distribution of the left L5-S1 dermatome following a 60-fsw He-O_2 dive for 120 min. Although he did not report lower back pain at that time, similarities in the type and distribution of sensory changes to the case reported here further suggested a predisposition to DCS in the L5-S1 area. With this episode, the diver was able to climb out of the wet pot and remove his diving gear, but these maneuvers caused him obvious pain. Once in the dry chamber, it was noted that the pain had increased in the center of his lower back and radiated down his left buttock and leg to a level below the knee. The right buttock was no longer painful. Neurologic examination showed a significant hypesthesia of the left buttock, left leg, and left foot without a radicular pattern. Sensory perception of pinprick and light touch were normal. Motor strength of the left anterior tibialis and left gastrocnemius muscles was 4/5 (Medical Research Council Scale). Plantar responses were flexor. Straight-leg raising (SLR) was tolerated to 5° on the left and 20° on the right, with a positive Leseague's sign on the left. The impression of the diving medical officer and a neurology consultant at that time was HNP, although the level was unclear. The diver was treated with oral ibuprofen (800 mg every 8 h) and diazepam (5 mg every 6 h) and restricted to his bunk with sanitary privileges. Emergency decompression was not considered to be indicated and chamber depth was maintained.

Forty-eight hours after the onset of symptoms, the pain had decreased to 50% of its peak. A Chatillon spring strain gauge was used to measure the strength of ankle dorsiflexion. The diver could hold against 90–100 lb of force on the right and 38–45 lb on the left. SLR was tolerated to 10° and 20° on the left and right, respectively. After strain gauge testing the diver reported increased lower back pain for which the dose of diazepam was increased to 10 mg every 6 h. Ibuprofen was discontinued, and the diver was started on oral dexamethasone (4 mg every 6 h). Ulcer prophylaxis was provided by using antacids and ranitidine (150 mg orally every 12 h).

The lower back pain improved slightly each day until 82 h after the onset of symptoms. While moving to use the sanitary system, the diver experienced an acute increase in pain from "2/10" to "10/10", with "10" being the most severe pain

possible. The diver described this pain as located in the center of his back now radiating down both legs to the feet. He also complained of increasing lower extremity muscle weakness. A neurologic exam (performed by one of the other divers while observed by a medical officer) revealed decreased perception of pinprick of both lower extremities medially from thigh to foot, motor weakness (4/5) of dorsiflexion and plantar-flexion of both the right and left ankles, bilateral patellar hyperreflexia, and flexor plantar responses. The anal wink and bulbocavernosus reflexes were intact. Rectal tone was normal and the cremasteric reflex was normal on the left but absent on the right. Bowel function remained normal, but the diver stated that he was unable to sense bladder fullness and, although able to void, did not feel relief with voiding. The progression of symptoms was considered to be evidence of increased compression of spinal nerve roots by the HNP.

At that time we had to develop the best way to bring the diver to the surface as soon as possible because he might need surgical treatment that could not be provided in the chamber. We considered an emergency decompression, but decided that there was too great a risk of DCS, which could cause worse damage in the region of acute inflammation. We therefore decided to postpone decompression for 12 h to allow further stabilization. He was given diazepam (10 mg every 6 h), dexamethasone (4 mg every 6 h), ranitidine (150 mg twice per day), and Percocet (oxycodone 5 mg and acetaminophen 325 mg per tablet; two tablets every 4 h). The diver was kept at strict bedrest. When it became apparent that Percocet was inadequate to control pain, a diving medical officer was locked into the chamber to help care for the diver. An i.v. line was established and a 1-mg dose of morphine sulfate was administered with no adverse reactions. The drug was administered in 2–5-mg doses as needed for the remainder of the dive.

Twelve hours after the acute exacerbation, the chamber partial pressure of oxygen (PPO_2) was increased to 0.50 atm abs to promote off-gassing, and decompression was started following a standard U.S. Navy saturation schedule (Table 1) (3). An allowable upward excursion to 94 fsw was eliminated, and the linear schedule was followed from 150 fsw. Before leaving the bottom, the diver's neurologic exam showed weakness of the left anterior tibialis and gastrocnemius muscles. The reflexes were symmetric, and the toes responded with flexor plantar responses. SLR was tolerated to 20° on the left and 30° on the right. Laseague's sign remained positive on the left side.

Decompression lasted a total of 57 h 24 min and was uneventful. There was improvement of the neurologic exam and a steadily decreasing need for i.v. morphine analgesia. Bowel and bladder function were normal and bladder sensation returned to normal. These findings were taken as evidence against any occurrence of DCS, and the patient reached the surface according to schedule. He was hospitalized immediately. A computed tomography (CT) scan showed a midline HNP of the L5-S1 intervertebral disk with minimal displacement of the thecal sac or spinal cord roots (Fig. 1). The diver responded well to a 5-day treatment course of anti-inflammatory medications and bedrest. He was discharged with a program for weight loss, physical therapy, and anti-inflammatory medications. He has returned to work as a diving supervisor and reports minimal lower back pain without neurologic symptoms.

Case 2

On Day 4 of a helium-nitrogen-oxygen (49% He:39% N_2:12% O_2) experimental saturation dive, a 30-yr-old diver had onset of right buttock pain. This occurred during

TABLE 1
DECOMPRESSION PROFILE OF CASE 1

Dive Day	Time	Depth, fsw Comments	Rate of Ascent
9	0700	150	5 FPH
9	1400	115	0 FPH
	rest period (1400–1600)		
9	1600	115	5 FPH
	resume travel		
9	1900	100	4 FPH
9	2400	80	0 FPH
	overnight hold (2400–0600)		
10	0600	80	4 FPH
	resume travel		
10	1330	50	3 FPH
10	1400	49	0 FPH
	rest period (1400–1600)		
10	1600	49	3 FPH
	resume travel		
10	2400	25	0 FPH
	overnight hold		
11	0600	25	3 FPH
	resume travel		
11	1300	4	0 FPH
	80-min hold		
11	1420	4	1 FPM
	resume travel		
11	1424	0	
	surface		

Key: FPH = feet per hour; FPM = feet per minute.

decompression, approximately 1 h 20 min after an upward excursion from 132 to 116 fsw, and was reported by the diver about 30 min after onset. The character of the pain was a burning ache with intermittent radiation to the right lateral leg, but had no effect on the thigh. The buttock pain gradually worsened and the radiating pain became constant. Sensory and motor exams were normal. Based on the immediately available history, DCS could not be ruled out, and the decision was made to return to storage depth and place the patient on a hyperoxic treatment gas (two 20-min periods at 2.3 atm abs O_2 and three 20-min periods at 2.7 atm abs O_2).

The patient's pain continued to worsen during treatment; however, he was able to provide a more detailed history. He recalled a brief twinge of pain several hours earlier upon completing a 25-min cycle ergometer ride at a workload of 150 W. This pain lasted several seconds and resolved completely. The current pain had an acute onset after a violent cough, contrary to the initial history of a gradually developing pain. Subsequent coughing was accompanied by worsening pain and constant radiation of pain to the leg. Past history revealed an episode of left-sided sciatica approximately 6 yr earlier. Lumbo-sacral spine x-rays were negative at that time, and the

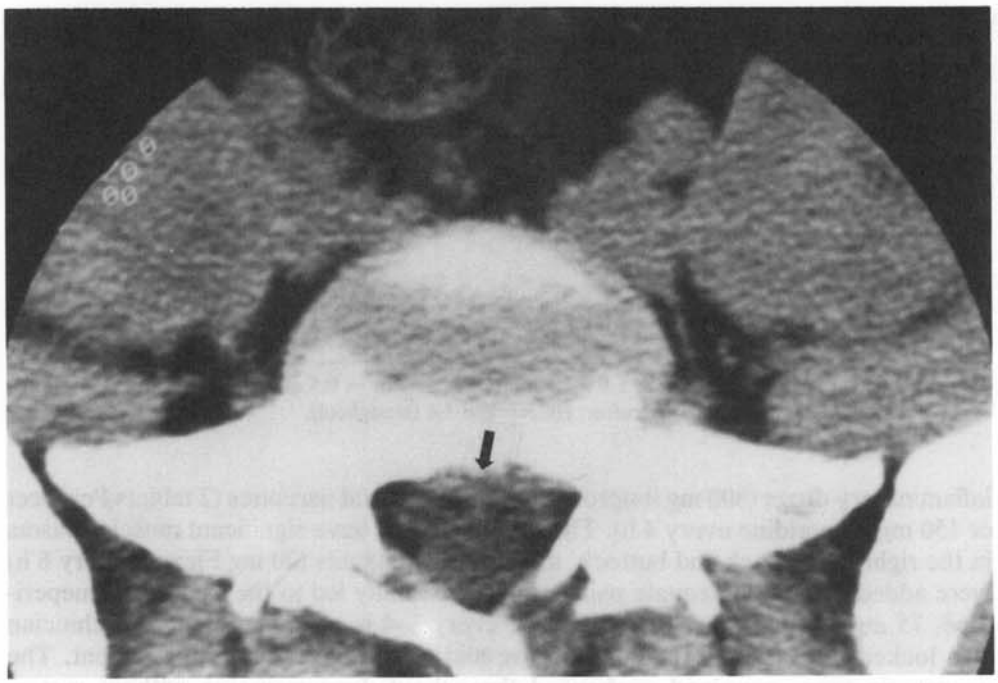

Fig. 1. Representative image from the CT scan of case 1. *Note* the midline herniation of the L5-S1 intervertebral disk (*arrow*).

symptoms completely resolved with physical therapy. There were no sequelae and the patient was very active without further complaints of lower back pain or radicular symptoms.

With this episode, the sensory and motor exams remained normal. The patient had a positive Leseague's sign on the right, with exquisite pain on SLR to a 10° elevation. This examination was conducted by another member of the dive team under the instruction and observation of a physician. The onset and response to treatment of the diver's symptoms were not consistent with the working diagnosis of type II DCS. The history and physical examination led us to consider these symptoms as a manifestation of an acute herniation of the L5-S1 intervertebral disk. The diver was placed at strict bedrest, and analgesics were provided as needed. Decompression commenced according to a well-tested He-N_2-O_2 trimix decompression schedule. During previous tests of this schedule an initial upward excursion was allowed from 132 to 116 fsw. This was eliminated in the current case, and the decompression was lengthened to travel at 20 min/1 fsw between these depths (Table 2). The total decompression time was 55 h 38 min. This modified schedule was 7 h 2 min longer than a standard U.S. Navy He-O_2 saturation decompression schedule. The schedule (including the initial upward excursion) had been previously tested with 11 man-dives without incidence of DCS. Also, 20 man-dives with less conservative decompression schedules (total decompression time equaling 41.2–44.5 h) resulted in two cases of DCS. It was the consensus that the chosen schedule was sufficiently conservative to minimize the risk of DCS while not unduly delaying definitive care for the patient. Measures to control the subject's pain were quickly advanced from nonsteroidal anti-

TABLE 2
DECOMPRESSION SCHEDULE IN CASE 2[a]

Depth fsw	Ascent Rate
132–100	20 min/fsw
99–50	24 min/fsw
49–30	26 min/fsw
29–20	30 min/fsw
19–3	36 min/fsw
3	hold for 108 min
3–surface	1 min

[a]All in steps of 1 fsw until last 3 fsw. (Po_2 = 0.5 ata until 26 fsw 28–30% thereafter; He:N_2 ratio 5:4 throughout).

inflammatory drugs (800 mg ibuprofen every 8 h) to oral narcotics (2 tablets Percocet or 150 mg meperidine every 4 h). The diver began to have significant muscle spasms in the right lower back and buttock, and muscle relaxants (20 mg Flexeril every 6 h) were added. Lack of adequate pain control eventually led to the use of i.m. meperidine, 75 mg, and 50–75 mg hydroxyzine every 3–4 h. A diving medical technician was locked in at 23 fsw for this purpose and to further examine the patient. The neurologic exam remained unchanged throughout decompression without motor, sensory, reflex, bowel, or bladder abnormalities. There were no acute exacerbations of pain during decompression.

The morning of surfacing, the patient had some relief of his pain. Examination on the surface by the attending physician revealed mild weakness (4/5) of the right extensor hallucis longus; however, the tibialis anterior strength and the rest of the motor examination were normal. All of the sensory modalities were intact. Sphincter tone and bladder function were normal. The diagnosis of a symptomatic herniated intervertebral disk with acute right L5 radiculopathy was confirmed by magnetic resonance imaging (MRI) (Fig. 2). The diver is currently responding well to conservative therapy.

DISCUSSION

Diver's activities, such as underwater repairs or hoisting equipment over the side in rough seas, may require lifting in awkward positions. Such work can result in physical injury including HNP. Two problems complicate the ability to provide appropriate medical care to the diver afflicted with this injury. First, it may be impossible to distinguish symptoms of DCS from those of acute HNP with compression of the spinal nerve roots. Two of the previously referenced cases illustrate the role that this diagnostic dilemma can play in delaying the recompression treatment for DCS (2). The second problem arises when it is necessary to decompress the diver with an acutely inflamed spinal nerve root due to HNP occurring while at depth.

Since previous injury may be a predisposing factor in the development of DCS (1, 2), what precautions can be made to minimize the risk of DCS? Our first diver's previous history of type II DCS, including sensory symptoms referable to the L5-S1

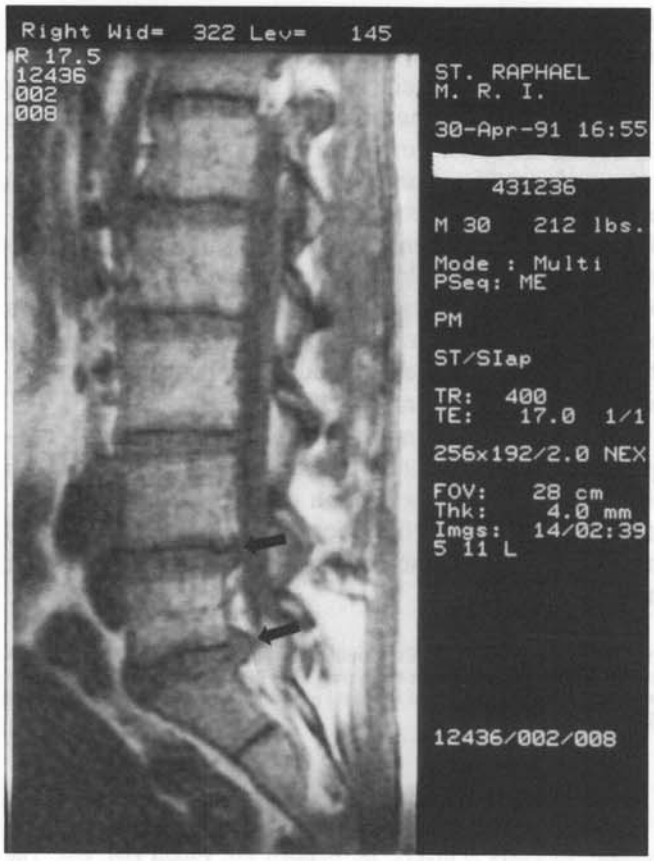

Fig. 2. Representative image from the MRI scan of case 2. *Note* the herniation of the L4-L5 and L5-S1 invertebral disks (*arrows*).

spinal nerve root distribution, further suggested a predisposition to DCS in this anatomic area. Both of these concerns are considered in the context of this case.

In these cases, divers with minimal prior histories of lower back pain developed symptoms of an acute HNP while at depth. Initial treatment of HNP usually consists of a trial of conservative measures: bedrest and anti-inflammatory and antispasmodic medications. Institution of these measures at depth were initially successful in decreasing the symptoms of the first diver and allowed the saturation dive to continue. Without insistence on absolute bedrest, however, this diver experienced an acute exacerbation of symptoms while moving to use the sanitary system. We would recommend enforcing strict bedrest in any future cases of HNP at depth.

Due to increasing neuromuscular deficits and unrelenting muscle spasm and radicular pain, we felt there was some urgency to bring these divers out of the chamber for complete neurosurgical evaluation. In the first case, after considering the increased threat that DCS posed to the damaged and inflamed tissue, we decided to give anti-inflammatory medications and prescribe complete bedrest for 12 h before starting decompression. Decompression was accomplished utilizing standard U.S. Navy saturation decompression tables (3). In the second case, a conservative, well-tested trimix saturation decompression profile was available. Decompression was started immediately in case 2. In both examples, an allowable upward excursion was eliminated due to the perceived additional risk of injury in the inflamed area. We felt that

the resulting additional decompression time was justified on this basis. We would not recommend upward excursions in this situation, although we cannot say whether adverse effects would necessarily result. The chamber's PPO_2 was increased to facilitate off-gassing during decompression in the first case. The diver could have breathed an even higher PPO_2 during the latter portion of decompression; however, repeated neurologic exams showed continued improvement with time and extra oxygen was not considered necessary. Experience with the trimix saturation decompression schedule suggested that additional oxygen was not necessary for safe decompression using this algorithm. The normal neurologic exam in diver 2 and improvement in the first diver's neurologic status and during decompression, supported by a decreasing need for i.v. morphine or i.m. meperidine, were considered evidence against the possibility of DCS. If there had been a sudden clinical deterioration, despite complete bedrest, we had planned to treat for DCS.

These cases demonstrate the successful decompression of two divers with confirmed, acute HNP occurring at depth using a standard U.S. Navy schedule with an elevated chamber PPO_2 or an He-N_2-O_2 trimix saturation decompression schedule. These cases also demonstrate the use of narcotics (i.v. morphine and i.m. meperidine) in the saturation environment, where their efficacy and potency seem to be similar to that experienced at the surface.

This work was supported by the Naval Medical Research and Development Command Work Unit no. 63713N M0099.01A-1052. The opinions and/or assertions contained herein are the private ones of the authors and are not to be construed as official or reflecting the views of the Navy Department or the naval service at large.—*Manuscript received September 1991; accepted February 1992.*

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