SAFE LIMITS: AN INTERNATIONAL DIVE SYMPOSIUM
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SEVERAL CASE STUDIES FROM GENERAL PRACTICE

Peter Chapman-Smith

A common but misleading case of decompression illness

This 29 year old clerk presented in my general practice surgery on a Tuesday morning. Two days earlier, he had been scuba diving for crayfish off the Hen and Chicken Islands near Whangarei. A regular diver, although not following any named tables, he utilised and had faith in an Aladin dive computer. The conditions at sea were surgy, his buddy being seasick. He had dived 3 or 4 times from midday, into decompression times for the first time. Following diving to 25 m for 38 minutes, he ascended. 20 minutes at 3 m at the end of this time, with 10 minutes on the surface during this shallow period. After a 2 hour surface interval, a second dive to 12 m for 40 minutes with a 3 m stop for 10 minutes followed.

He had experienced nausea, and chest tightness on that day. The next morning he was lethargic, just not himself, and was anxious.

In the past he had had a significant motor vehicle accident (MVA) 10 years before, sustaining multiple fractures, was on no medications and he was evidently allergic to Penicillin. Both shoulders had been reconstructed".

His detailed examination findings were quite normal, and he was reassured and sent home with the offer to return if not settling.

He came back 3 days later, with persistent symptoms. Still nauseated, with lethargy, tightness in his chest, now unsteadiness, and a niggly pain in his right shoulder. He had been to the chiropractor the day before for treatment on his own initiative.

Full neurological examination was still normal, including a sharpened Romberg test. His peak flow was 610 l/min, and there were no chest or cardiac signs.

A provisional diagnosis of decompression illness was made, and he was referred to Auckland for a trial of recompression therapy. Treated with a single treatment, on oxygen at 10 m for 60 minutes then a 30 minutes ascent, all signs and symptoms were relieved immediately according to the Naval Hospital report. His 100-7 test improved from 60 to 40 seconds, and some degree of left hip weakness of extension and flexion disappeared (previously ascribed to his old MVA). This was considered to be multifocal neurological non-progressive decompression illness.

After 24 hours he was well and was discharged to go home, with a 1 month ban on scuba diving. Reviewed at my rooms 1 week later with his wife, he reported some ongoing chest tightness, had tried Voltaren but felt this had not helped.

At follow up 1 month after the event, he was having difficulty coping with stress, was having mood swings, tending to cry and was labile in his mood. Memory was fine and he had not been diving.

His case was chosen to highlight a very common description of decompression illness, that is persistent tiredness, with patient insight that all is not well. "I am just not right!" is a common description. Examination can be often be quite unfruitful. A clear history, the ongoing nature of the problems, and complete resolution of symptoms with a trial of recompression treatment often confirms what is suspected.

Multiple presentations of decompression illness

A 19 year old male suffered from musculoskeletal decompression illness in July 1993 in New Zealand, presenting with a 3 day history of fleeting joint discomfort and paraesthesiae, and a one day history of upper limb weakness and lethargy. The previous day he had been scuba diving with a conservative profile, to 12 m for 35 minutes, then making a rapid ascent at the end of the first dive to assist his buddy experiencing loss of buoyancy control. After 1 hour and a quarter on the surface, he dived again to 8.5 m for 35 minutes chasing those elusive crayfish, but made 3 ascents during the dive. After diving he was well.

One day later, he became aware of some slight fleeting joint discomfort and tingling affecting his right wrist, knee and hip. Unconcerned, he went fishing for the day. That night his arms were much less strong, tiredness was more so, with increased paraesthesiae.

A PADI diver, with 110 logged dives to this date, and no other medical or diving history of note, he had been welding and using a power saw the month before this.

Examination findings were remarkably normal, with a soft flow murmur noted, but no neurocognitive deficit. The murmur disappeared before his hospital discharge.

With typically vague symptoms, and affected joints 24 hours after diving, decompression illness was presumed. A single recompression and IV fluids were given. There were 2 extensions to the table at 18m before decompression. All his symptoms completely resolved by the next morning. He was advised not to fly or dive for 1 month.

4 months later he came for a commercial diver's medical. A trained scuba diver for 3 years, he had a past history of tonsillectomy and grommets. When examined, a systolic murmur and early diastolic murmur were noted. He admitted to very long standing ongoing enuresis 2-3 times weekly. His mid-stream urine was normal, as was an echocardiogram. A 2D colour doppler echocardiogram was recommended but not done. He was reviewed by a physician who diagnosed a trivial flow murmur only, with complete right bundle branch block noted, as well as posterior fascicular block on an ECG.

He came back 2 months later unwell, with myalgia, photosensitivity, and feeling just not right. He felt lethargic, forgetful, unwell and was aware of poor mental concentration. Over Christmas he had been diving a lot, well within accepted times. Mostly these were single daily dives at the Poor Knights Islands, between depths of 10 and 25 m. 20-25 minute bottom times were average, with the longest exposure to nitrogen being a dive 1 month earlier to 23.5 m for 38 minutes.

Now he was 10 days post dive, with aching shoulders, knees and near the joints he felt some pain. Short term memory was reduced, and blood tests were expectedly normal. A lean and reserved young man, his detailed neurological examination was normal. After discussion he was transferred to the Royal New Zealand Navy Hospital (RNZNH) for recompression. On arrival he was noted to have poor attention, but no abnormal signs were detected in his cardiovascular and neurological systems.

Presuming chronic static constitutional decompression illness, a trial of pressure (Table 62) was conducted. The following day his feelings of malaise and lethargy were gone. 24 hours later he went home well.

Reviewed 1 week later, all of his symptoms were gone subjectively. Mentally more acute, and well coordinated, he was advised against flying and diving for one month. A further month passed, he reported no symptoms at all, and was encouraged not to pursue his planned career of commercial diving.

Interestingly, his mother came to advise me that he was born at 38 weeks, with low oestriols, and his childhood milestones were normal. Recurrent tonsillitis and otitis media were childhood illnesses, but he continued to do well at school. At the age of 11 years, he was labelled as a minor dyslexic, although with a reading age of 7 and a half years. His short term memory failed him, and his mother insisted that his work efficiency improved when under pressure. Over the years she reported purchasing many tubes of "Deep Heat", a heat rub, for frequent complaints of vague musculoskeletal pains.

Interestingly, when in Vanuatu, diving with his father 5 months earlier, decompression profiles were chosen on 5 days with a maximum depth of 58 m. There were 75 hours without diving before flying out. His first episode of diving illness was to present 2 weeks later. His father had made similar dives, and was well at this time.

When he became unwell 5 months later, his father also became quite feverish and unwell within hours. They both experienced chills, rigors, and malaise for about 18 hours.

Clearly he was advised not to undertake a career of commercial diving at this point. A full recovery was noted but an informed consent approach was taken about further recreational diving.

Five months after his second treatment at RNZNH, he reported daily hip and knee pain, with onset one hour after rising. At times severe, he stated he could not stand for more than an hour without the need to sit or lie down. A depressed mood prevailed, with interest shown to exclude tropical diseases such as dengue fever. Antibodies for this were negative, and other bloods were normal.

He demonstrated 2 apparent separate episodes of decompression illness 6 months apart, the first after more provocative profiles, and an excellent response to recompression first aid with relapsing ongoing musculoskeletal pain within months. Depression colours the picture, a not uncommon scenario.

Recurrent constitutional decompression illness

This 47 year old self employed mechanic presented with his third episode of decompression illness requiring recompression in January 1993. He had been successfully treated with recompression in 1977 and 1989. At this time he was advised not to dive again, but had done many dives since, always completing a 3 m stop.

He had been chasing crayfish at 5 p.m. two days earlier, his maximum depth was 16 m with a bottom time of 41 minutes. In good, calm sea conditions, he has sat on a rock at 6 m to de
compress until his gauge showed 150 psi. A slow ascent followed to the surface as usual.

He returned to work to complete a job, and felt well. Two hours after the dive he noted an earache on the left. There were no other ear symptoms, and he took an aspirin. The next morning he awoke with earache still, but in both ears and with pain in both legs also. All over his skin, he was aware of altered sensation. His ears felt blocked, and he knew he was "not right". A complete physical examination was normal, and mild exostoses were noted in both ears. He was referred for recompression treatment in Auckland, and he required multiple treatments for recalcitrant symptoms. Forty eight hours after leaving RNZNH to return home, he relapsed and complained of numbness in both feet, and a fuzzy sensation in his head. He returned to RNZNH for a further 5 treatments in the chamber. Three days later he felt "100%". He celebrated with 2 cans of beer that night.

Two days later his head felt pressurised, with frequent headaches and fortunately no balance problems or inner ear symptoms other than some tinnitus. A further detailed physical examination was normal. His audiogram remained symmetrical with a minor high frequency loss, probably occupational in nature, with 60-70 db loss above 6000 Hz.

He was reviewed one month later with his wife. Both ears felt blocked 60% of the time, with earache, and subjectively some increase in his hearing loss. Tinnitus was a constant and harrowing problem. Lethargy was a feature, with a desire to go home to bed for a snooze about 2 p.m. A trial of low dose amitryptiline and diazepam was just sedative. Simple analgesics were helpful.

Over the next 2 months, occipital headaches persisted daily. Some features of depression emerged, namely insomnia, low mood, tiredness, and poor energy. He was aware of a staggering gait after drinking minimal alcohol, and his frustration was ongoing as several antidepressants were tried without success. A short term memory deficit now affected his usual job as a mechanic. To stop during a job to answer a phone call or an enquiry would mean he had to restart the entire job again, not knowing what had been done to that point. Insight was quite clear. He estimated his work output as being 40% of his usual. His attention span was poor, with poor libido, problems starting micturition, less frequent bowel motions and insomnia, awake for 4 to 5 hours each night. Minimal motion now made him seasick. His physical examination now demonstrated poor balance, being unable to stand on either leg alone with eyes closed, and a sharpened Romberg test of 3 seconds! He was referred to the local Rehabilitation Unit and an MRI was considered but not done. The orthopaedic consultant in charge replied suggesting from my letter perhaps he had "a reversible organic brain syndrome"!

A reassessment was done at RNZNH on the next obvious referral for assessment, now 6 months after treatment. A little improvement had occurred, but the earache, insomnia, buzzing tinnitus and memory problems were ongoing.

An ENT assessment 2 months later was to reveal nothing new. A formal neurological consultation was arranged a further 2 months later, now 10 months after presentation. The neurologist declared there was no other underlying illness at all, thought he was depressed, and suggested a psychiatric referral . At this time his left sole of his foot was numb, especially the heel which also ached, more so in the cold. His recall for simple names, of good friends and details of vehicles that he had attended recent expensive update courses on maintaining were often blank. A new ache in the groin was worrying him.

Four months later, now 15 months after his illness, he reported feeling moody, irritable, with frequent headaches, worse bladder dysfunction, not incontinence but a poor sense of bladder volume. Still forgetful, but a little less so, he estimated his work output as now 60%. Duodenal ulcer symptoms appeared. A course of Famotidine was helpful, as was a trial of Prozac. He continued to make silly expensive mistakes in his business.

He was then referred for a head injury assessment with a local speech therapist, which confirmed his cognitive deficit, which is ongoing. Auditory short term memory deficit, especially at paragraph and information levels were his main dysfunction. Less so was the inability with written material. Errors in word finding and associative thinking and in name recall were obvious. Some compensation had occurred, but real problems remain in his running a business.

In short, a poor result to his third therapeutic recompression and a further example that the current level of treatment is but rudimentary. As is often the case, opinions from specialist colleagues are blinkered by conventional thought, are frustrating and depressing for the individual with decompression illness.

Key Words

DCI, sequelae, treatment.

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DIVE ACCIDENT MANAGEMENT IN CENTRAL NEW ZEALAND

Tony Hochberg

Preamble

This paper deals with the management of compressed air diving accidents in sport and commercial divers.[1,2]

Decompression illness (DCI) must be considered in the differential diagnosis of anyone becoming ill after a dive. The presentation of DCI may be obvious, delayed or unusual and present a diagnostic dilemma.

Advice and referral to a hyperbaric facility should be considered, even if there is a low probability of DCI, as frequently the severity is underestimated and the prevention of long term sequelae is of prime importance.

The key components of treatment are stabilisation, time to recompression and consideration of adjuvant therapy.

Assessment

The successful management of DCI involves effective communication between the referring agency (diver, dive shop, doctor, hospital etc.) and the physician trained in hyperbaric medicine.

Key questions in the assessment of any diver include the following:

1 Name and age of diver.
2 Status includes the level of consciousness, bleeding and visible injuries.
3 Dive profile, including any dives in the last 7 days, whether in fresh water or salt water, what dive tables or dive computer was used and the stage of the dive symptoms developed etc.
4 Symptoms.
5 Air travel and/or ascent to altitude over 300 m following the dive.
6 Relevant medical conditions, e.g. migraine, high blood pressure, asthma, epilepsy.
7 Medications.
8 Emergency treatment instituted.

Transportation

For referrals from a peripheral location, an intimate knowledge of the available emergency services and the level of skill of the operators is crucial to the successful evacuation of an acutely injured diver.

The modes of transportation for an injured diver include motor vehicle, train and air transport, either helicopter or fixed wing.

The first two modes of transportation are used if the probability of DCI is very low, the presentation is very late (with no serious complications such as neurological DCI that would necessitate the high cost of aeromedical evacuation) or there are adverse weather conditions.

By road the journey from Wellington to Devonport Naval Base, in Auckland. can be accomplished comfortably in 8 hours (a distance of 700 km).

Emergency air evacuation is not without its risks. Air ambulance standards are defined by the Civil Aviation Authority in an information circular Gen A dated July 1983.[3] Unfortunately deregulation of authorised air ambulances over the last few years has resulted in fatal aircraft accidents during aeromedical evacuation.

In New South Wales, Australia, Donaldson[4] provided some figures for comparison on helicopter aeromedical evacuation fatalities: a crude ambulance mortality rate of 3 deaths per 20,000 hours flown, which compares to a NSW road ambulance mortality of 0.015 deaths per 100,000 vehicle hours logged. In the USA the figure is under 6/ 100,000 hours for helicopter flights.

Contraindications to air travel in a diver include, shock, pneumothorax, myocardial infarction, vomiting and aerocele.

Equipment

Aeromedical equipment fits into two categories. The first is equipment integral to the aircraft e.g. stretcher, mountings, oxygen cylinders. The second is equipment carried onto the aircraft to accompany the diver.