Decompression Sickness with Subsequent Lymphatic Manifestation Following Recompression Treatment: 
A Case Report in a Heavy Drinker

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A case of decompression sickness is described in an uniformed diver who developed lymphatic manifestations after recompression treatment. No definite contributing factors to this rare disorder were established. A brief review of cutaneous lesions in decompression sickness is also presented.

(Key Words: decompression sickness, skin lesions, diving)

This paper presents an unusual case of decompression sickness (DCS) manifesting as lymphatic edema following recompression treatment and provides a brief review of the cutaneous forms of DCS which still lack a clear description despite their frequent occurrence.

CASE REPORT

A 40 year old Japan Maritime Self-Defense Force (JMSDF) male diver had been engaged in an aircraft salvage operation commencing February 27, 1984, which was previously described elsewhere concerning the incidence of DCS (11). On March 13 he made his 7th hard hat dive during this operation to a depth of 181 feet sea water (fsw) or 55 meters sea water (msw) with a bottom time of 32 minutes and 16 seconds. Approximately 10 minutes after finishing surface decompression, he noted wide-spread skin redness over his chest and upper abdomen with mild itching in the same area. These lesions were not reported to the medical officer at that time. He claimed that they disappeared on taking a hot shower about 30 minutes after finishing decompression.

His 8th dive on March 20 to a depth of 174 fsw (53 msw) for 20 minutes and 27 seconds bottom time was uneventful.

On March 30, he made a dive to 187 fsw (57 msw) for 34 minutes 14 seconds and was decompressed according to the U.S. Navy surface decompression table using air on 190 fsw (58 msw) for 40 minutes schedule(13). When he was at the 10 fsw (3 msw) stop in the chamber, he noticed slight itching and felt something heavy over his upper body. Just after finishing surface decompression, severe itching developed. One to two minutes later patches of an erythematous rash appeared on his upper body. The erythematous lesions quickly coalesced and covered his chest, back, neck, shoulders, and both upper arms (Fig. 1). In the next 15 to 20 minutes, pale cyanotic areas appeared in the erythematous lesions with an irregular venous network pattern manifesting so-called central cyanosis (6,9). Mellinghof's sign (accentuation of venous markings by coughing or the Valsalva maneuver) was not tested (5). The erythematous lesions blanched on pressure while the cyanotic areas were unchanged. The skin was not tender but slightly edematous. Twenty four minutes after surfacing, the patient was treat-
ed using recompression treatment table 5 (TT-5) (13). On reaching 60 fsw (2.8 atmosphere absolute, ata, or 18 msw), itching decreased markedly. After 5 minutes at 60 fsw cyanotic changes disappeared. The itching disappeared completely after 40 minutes at 60 fsw. The erythematous skin lesions had almost disappeared at the end of recompression treatment, with only small red spots remaining on his arms and back. There was no pain throughout this course. Crepitus was not observed.

The next morning he reported that he felt some stiffness in his neck and shoulders. Marked pitting edema with tenderness was noted on his chest, back, neck, shoulders, and both upper arms (Fig. 2). Recompression treatment using TT-6 was applied with intravenous administration of 250 mg of hydrocortisone and 24,000 units of urokinase as adjunctive therapy. Since considerable edema remained after this treatment, two more recompression treatments (2 sets of TT-5 with adjunctive therapy in the following two days) were needed for complete recovery. Fig. 3 shows complete disappearance of the edema on April 2. Lymph nodes were not palpable throughout this period.

His personal record shows that he had been an active uniformed diver for the last 17 years without significant disorders except for suffering probable DCS 11 years ago during surface supplied mixed gas hard hat diving. It is worth mentioning that he had been a heavy drinker for more than 20 years, i.e., he drank sake, beer or whisky equivalent to at least 200 ml of pure alcohol a day off duty. Despite this history, his liver function tests after four days of abstinence were essentially normal. Platelet count was 250,000/mm$^3$. Prothrombin time was normal. Glutamic oxaloacetic transaminase (SGOT/AST) and glutamic pyruvic transaminase (SGPT/ALT) were both 28 Karmen units. Gammaglutamyltranspeptidase ($\gamma$-GT) was 49 IU/l. Indocyanine green (ICG) retention rate after 15 minutes was 8.5% (normal < 10%).

Over the past three years, he has remained healthy.
Fig. 2  Lymphatic edema developing after recompression treatment

Fig. 3  Disappearance of lymphatic edema.
DISCUSSION

Although skin lesions are common in the DCS syndrome, few studies have been undertaken concerning cutaneous manifestations, probably due to their transient, benign and non-life-threatening nature.

The terminology is obscure. Aldao divided skin lesions into six categories, i.e., pruritus, cutis marmorata, the erysipelas form, scarlatinaform rash, the serious form and emphysema (1). This grouping, however, is a mixture of morphology, severity, and clinical manifestation. Morphological descriptions are also not clear. Rashbash studied pruritus, and clarified that itching on dry diving is caused by exogenous gas in the skin (14). This is generally accepted. Ferris and Engel described in detail the skin changes seen on high altitude exposure, where pale cyanotic areas preceeded erythematous areas (7). Dennison checked the literature and speculated on possible mechanisms of the various forms of cutaneous DCS cited by Aldao, proposing that pruritus and scarlatinaform rash are caused by exogenous gas, while erysipelas and more serious forms are caused by endogenous gas (4). Edmond et al then reported that cutis marmorata commences as a small pale area with cyanotic mottling and then becomes erythematous peripherally (5).

Unlike the above, Elliott and Kindwall (6) and Flynn et al. (8) did not subdivide the rash morphologically. They reported that the mottled lesions are preceeded by itching, beginning as erythematous lesions, and when left untreated, develop into pale cyanotic mottling. In the present case observed by the author throughout, the course followed the latter sequence.

Lymphatic manifestations (edematous lesions due to lymphatic obstruction) were reported to be present in 10% of type I DCS cases (6), most of which shows prompt regression of lymphedema on recompression treatment. Davis and Elliott stated that there have been no reports of recurrence of lymphatic lesions after recompression treatment (5). This case appears to be the first one showing recurrence of this type of lesion after treatment. It is also unusual because a long period — three days — and three sets of recompression treatment were required for complete resolution of the lesions.

Several contributing factors to this rare case can be listed. 1. Although they were not quite at the exceptional exposure limits of the U.S. Navy dive tables (13) the depth and bottom time of this dive were, in retrospect, too deep and long for the decompression schedule used. Recently there have been several reports on reevaluation of the decompression process, where deep and especially long dives have been recognized to show some risk of decompression sickness (15). In this diving operation DCS developed in 19% of dives decompressed using the 190 fsw (58 msw) and 40 minutes schedule (11). 2. Omission of recompression treatment for the possible DCS on March 15 may have left some sequelae which predisposed the diver to more serious problems (2). 3. The treatment table chosen (TT-5) may have been inappropriate or inadequate to treat the effects of the initial DCS. 4. Although it is recognized that heavy drinking may increase susceptibility to aseptic bone necrosis due to the formation of fat emboli or some other mechanisms (10), no clear relationship has been shown between acute DCS and alcohol abuse except for some anecdotal reports (8, 12). Despite this, heavy drinking may have some effect on the development of this rare form of DCS. Liver or coagulation disorders are possible mechanisms, although no apparent malfunction was demonstrated. 5. An underlying disease process such as lymphoma which could cause lymphatic obstruction can be excluded because of the diver’s good health over the past 3 years.

REFERENCES