Delayed treatment for decompression illness: factors associated with long treatment delays and treatment outcome

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Abstract
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Introduction: Effectiveness of delayed hyperbaric oxygen treatment (HBOT) for decompression illness (DCI) and factors affecting treatment delays have not been studied in large groups of patients.
Methods: This retrospective study included 546 DCI patients treated in Finland in the years 1999–2018 and investigated factors associated with recompression delay and outcome. Treatment outcome was defined as fully recovered or presence of residual symptoms on completion of HBOT. The symptoms, use of first aid oxygen, number of recompression treatments needed and characteristics of the study cohort were also addressed.
Results: Delayed HBOT (> 48 h) remained effective with final outcomes similar to those treated within 48 h. Cardiopulmonary symptoms were associated with a shorter treatment delay (median 15 h vs 28 h without cardiopulmonary symptoms, $P < 0.001$), whereas mild sensory symptoms were associated with a longer delay (48 vs 24 h, $P < 0.001$). A shorter delay was also associated with only one required HBOT treatment (median 24 h vs 34 h for those requiring multiple recompressions) ($P = 0.002$). Tinnitus and hearing impairment were associated with a higher proportion of incomplete recoveries (78 and 73% respectively, $P < 0.001$), whereas a smaller proportion of cases with tingling/itching (15%, $P = 0.03$), nausea (27%, $P = 0.03$), motor weakness (33%, $P = 0.05$) and visual disturbances (36%, $P = 0.04$) exhibited residual symptoms. Patients with severe symptoms had a significantly shorter delay than those with mild symptoms (median 24 h vs 36 h respectively, $P < 0.001$), and a lower incidence of complete recovery.
Conclusions: Delayed HBOT remains an effective and useful intervention. A shorter delay to recompression is associated with fewer recompressions required to achieve recovery or recovery plateau.

Introduction

Scuba diving is popular all around the world and at times practised in locations remote from hyperbaric treatment facilities. Therefore, in cases of diving-related injuries the time taken to reach medical facilities can be long. Furthermore, the symptoms of decompression illness are often mild, further increasing the delay. The causes of treatment delay and how they influence the treatment outcome remains a matter of interest to the diving medicine community.

Decompression illness (DCI) is a collective term which includes two pathophysiologically different syndromes: arterial gas embolism (AGE) following pulmonary barotrauma and decompression sickness (DCS) caused by bubble formation from dissolved gas.[1] In this study the term DCI is used as it can be difficult to differentiate between AGE and DCS in a clinical setting,[2] although it is likely that the vast majority of the cases were DCS. The gold standard intervention for DCI is hyperbaric oxygen treatment (HBOT), which can also be used as a treatment for non-diving related injuries, such as carbon monoxide poisoning, gas gangrene, delayed radiation injuries, necrotizing soft tissue infection and severe burns.[3]

The manifestations of DCI can vary greatly in severity. The agreed mild symptoms include constitutional symptoms such as fatigue, limb pain, some sensory changes such as tingling, skin rash and subcutaneous swelling as long as the

manifestations are static and neurological dysfunction is excluded by a diving medicine physician.[4] Therefore, other symptoms are classified as severe. These include dizziness/vertigo, motor weakness, mental, pulmonary, or coordinative disorders, decrease in the level of consciousness, auditory, bladder and cardiovascular symptoms.[1,4]

Whether or not the treatment outcome is influenced by a long delay from symptom onset to HBOT, is still a debated subject, as it is also profoundly affected by the severity of manifestations. There is evidence that a short treatment delay is beneficial in severe cases of DCI.[4] Some older research has also shown that a shorter time to recompression is associated with better treatment outcomes.[5–7] However, these studies did not stratify the presentations according to severity. Other recent studies have shown that although there is some evidence that treatment outcome is better with shorter delays, divers with a longer delay can still benefit from HBOT.[8–9] In addition, worse outcomes may be linked to specific symptoms, such as severe neurological symptoms, not so much to the delay.[10] There is a broad consensus that mild DCI can be adequately treated without HBOT[4,11] particularly, if recompression is logistically difficult or hazardous to access, as the symptoms tend to disappear with time.[12,16]

The aim of this study was to investigate the effect of delayed HBOT (> 48 h) and other factors on treatment outcome for DCI. Moreover, factors affecting the time to the chamber treatment were also evaluated.

Methods

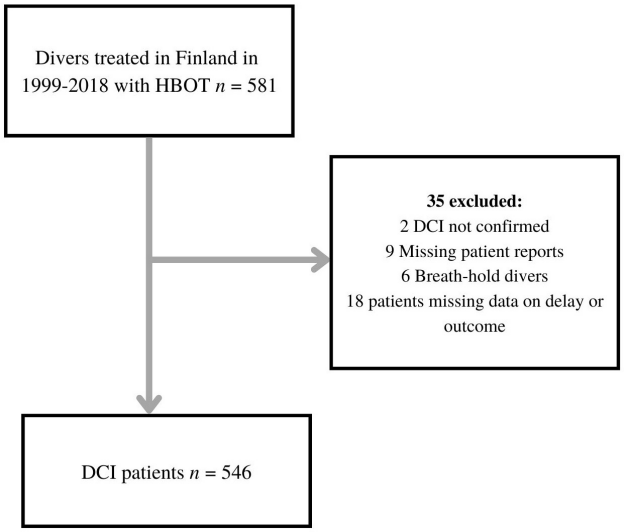
The study received ethics approval from the National Institute for Health and Welfare, Helsinki, Finland (THL/285/5.05.00/2016). The study adhered to the Declaration of Helsinki.

PATIENT POPULATION AND DATA COLLECTION

The data for this retrospective study includes approximately 95% of all treated DCI cases in Finland from the years 1999–2018. The patients were treated in the Hyperbaric Medical Clinic Medioxygen in Helsinki or in the National Hyperbaric Unit of Turku University Hospital in Turku, Finland. Unfortunately, Medioxygen was closed in 2015 and Turku is the only treatment centre currently operational. Both of these were located in the South of Finland resulting in a long journey (up to 1,000 km) from other parts of the country.

Data were collected retrospectively from medical records of 546 patients treated at the two facilities. The flow chart for patient selection is shown in Figure 1. For the majority of cases the initial treatment was United States Navy (USN) TT6 with or without extensions (79%), however, a few milder cases received USNTT5. In the era 1999–2015 the follow-up treatments were usually USNTT9, later mainly USNTT6 or 5.[13,14] The HBOT treatments were continued as long as there was diminishing of the symptoms, until complete recovery or until there was no sustained improvement between two consecutive treatments. Patients were clinically evaluated directly after HBOT and at discharge. If patients left the treatment facility the day they were treated, the physician called the patient the next day to ensure that symptoms had not re-evolved.

Figure 1
Flow chart of patient selection and exclusion criteria for patients treated in Hyperbaric Medical Clinic Medioxygen in Helsinki or in the National Hyperbaric Unit of Turku University Hospital in years 1999–2018



Finland has challenging diving conditions leading divers to travel abroad looking for warmer and clearer waters. However, roughly 78% of the patient population was diving in cold water (4–10°C), whereas 22% were diving in warm water abroad.[13] This dataset includes divers from beginners to professional divers. The training level of the divers was defined as beginner, advanced, or expert. Beginner divers were open water divers (OWD) of any training organisation, advanced divers were advanced open water divers (AOWD) or nitrox divers, and expert divers were those who completed a higher course than AOWD including technical diving. This group also included professional divers. Additionally, the use of first aid oxygen (FAO_2) and any previous DCI treatment were recorded.

TREATMENT DELAY, SYMPTOMS AND OUTCOME

Treatment delay was the number of hours from onset of DCI symptoms to recompression. Delayed treatment was defined as treatment delay greater than 48 h. This time point was arbitrary but has been used before,[9] and is long enough for the secondary symptoms to appear. The treatment outcome was defined as either fully recovered (no residual symptoms after HBOT) or not (presence of residual symptoms). Presenting symptoms were categorised as either mild (as defined by the 2018 consensus guideline[4]) or severe as explained in the introduction.

Table 1
Demographics of the patient population ($n = 546$); data are median (IQR) or n (%); depth and dive time data missing for four and 57 patients respectively; DCI – decompression illness; FAO_2 – first aid oxygen; m – metres (distinction between seawater and freshwater depths not made); min – minutes

Parameter	Data
Age (years)	36 (30–42)
Sex, Male	423 (78%)
Previous DCI	119 (22%)
Depth (m)	30 (21–42)
Dive time (min)	45 (30–64)
FAO_2 provided	145 (27%)
Dive training level	
Beginner	92 (17%)
Advanced	209 (38%)
Expert	136 (25%)
Not recorded	95 (17%)

Treatment delay and outcome were evaluated among groups of symptoms including the following categories: subjective findings including musculoskeletal pain and neurosensorial symptoms (tingling, itching, subjective numbness), and objective findings including skin rash, neuromotor symptoms (motor weakness), vestibulocochlear symptoms (dizziness, vertigo, nausea, hearing impairment, tinnitus), central nervous system (CNS) symptoms (visual, coordination or verbal disturbances, rigidity, tremor, abnormal reflexes, numbness, bladder dysfunction) and cardiopulmonary symptoms. Some symptoms (bowel pain, subcutaneous swelling) were difficult to categorise and were left out of the analysis, as the number of these cases were small (bowel pain $n = 8$, swelling $n = 17$). However, they were taken into consideration in the mild vs severe classification as mild symptoms. If multiple symptoms were present, the patient was categorised based on the most severe symptom. It is important to note that the 'neurosensorial' category consists of only mild symptoms. Vestibulocochlear symptoms were considered severe. In addition to groups of symptoms, the outcome of treatment was evaluated for various individual symptoms and whether the patient recovered fully or had residual symptoms.

STATISTICAL ANALYSIS

We describe the data using counts and percentages for categorical variables and median and interquartile ranges (IQRs) for continuous variables. Categorical variables were compared using Chi-squared tests or Fisher's exact test and continuous variables using Mann-Whitney U tests or Kruskall-Wallis tests depending on the number of categories compared. P-values < 0.05 were considered significant. The analyses were done using R version 4.1.0[15] and the plots were done with the ggplot2-package (open source url GitHub - tidyverse/ggplot2: An implementation of the Grammar of Graphics in R).

Results

PATIENT POPULATION

The demographics of the diver population are shown in Table 1.

TREATMENT DELAY AND THE RELATIONSHIP BETWEEN OUTCOME AND DELAY

Patients with no residual symptoms had a median delay from symptom onset to recompression of 24 h (IQR 12–72) and the patients with residual symptoms had a median delay of 28 h (12–96); a statistically insignificant difference. Of the patients who fully recovered, 59% were treated within 48 h. Similarly, 53% of the patients with residual symptoms were treated within 48 h (also a non-significant difference).

FACTORS ASSOCIATED WITH DELAY TO RECOMPRESSION

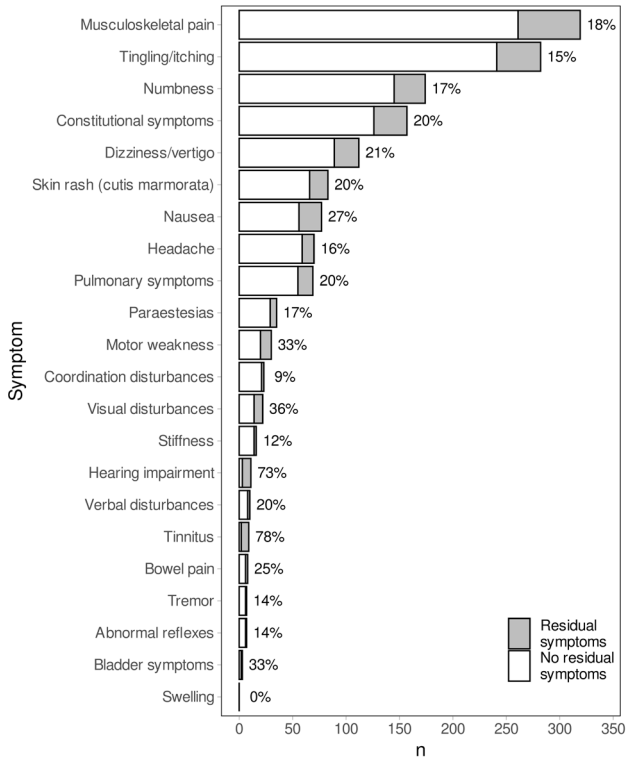
When DCI symptoms were categorised into symptom groups, mild neurosensorial and cardio-pulmonary symptoms had a significant association with treatment delay. Neurosensorial symptoms had a significantly longer delay than patients with no such symptoms (median 48 h vs 24 h, respectively). On the other hand, patients with cardio-pulmonary symptoms had a significantly shorter delay than patients with no such symptoms (15 h vs 28 h, respectively) (Table 2). Other symptom categories (pain only, skin, neuromotor, vestibulocochlear, CNS) did not show a statistically significant difference in terms of treatment delay or the groups were too small for statistical analysis (e.g., AGE, $n = 2$). Patients with severe symptoms ($n = 259$) had a significantly shorter delay than those with mild symptoms ($n = 287$) (24 h vs 36 h respectively, $P < 0.001$).

Patients who used FAO_2 had a significantly shorter delay to recompression; 14 h (5–27) vs 48 h (21–96) where FAO_2 was not used, ($P < 0.001$). There was also a significant difference in the delay between divers of different training levels. Beginners had the longest delays (72 h [21–144]) and the delay decreased as the training level improved as advanced divers had a median delay of 48 h (24–96) and expert divers had a delay of 24 h (7–48) ($P < 0.001$). Additionally, a pattern between the number of HBOT treatments needed and delay was observed. Patients who only needed one treatment had a median delay of 24 h (9–54) whereas those who needed two, three and four or more had delays of 37 h (24–96), 24 h (8–72), and 36 h (20–96) respectively. Therefore, patients who only needed one treatment had a shorter median delay than those who needed multiple treatments (34 h [20–96], $P = 0.002$).

Table 2
Treatment delay in different categories of symptoms; data are median (IQR) or n (%)

Symptom	*n* (%)	Delay without symptom (h)	Delay with symptom (h)	*P*-value
Pain only	76 (14%)	26 (12–96)	24 (20–48)	0.09
Neurosensorial	214 (39%)	24 (8–72)	48 (24–108)	< 0.001
Skin	41 (8%)	25 (14–90)	24 (8–48)	0.09
Neuromotor	21 (4%)	25 (12–72)	24 (11–72)	0.43
Vestibulocochlear	90 (17%)	24 (16–72)	24 (6–96)	0.36
Central nervous system	58 (11%)	26 (12–72)	24 (13–72)	0.32
Cardiopulmonary	43 (8%)	28 (17–96)	15 (6–24)	< 0.001

Figure 2
The relationship between individual symptoms and treatment outcome. Percentages show the number of patients with residual symptoms after HBOT in different symptom categories



ASSOCIATIONS WITH TREATMENT OUTCOME

There were both mild and severe symptoms that were associated with a better treatment outcome, i.e., no residual symptoms after treatment. These were tingling/itching (15% with residuals, $P = 0.03$), nausea (27%, $P = 0.03$), motor weakness (33%, $P < 0.05$) and visual disturbances (36%, $P = 0.04$). Among severe symptoms only tinnitus (78% residuals) and hearing impairment (73%) (both $P < 0.001$) were significantly associated with a worse outcome (residual symptoms). Other symptoms analysed (Figure 2) were not associated with a treatment outcome. However, 85% of patients with mild symptoms had no residual symptoms after treatment, whereas the corresponding number for patients with severe symptoms was 78%, $P = 0.03$.

There was no difference in the treatment outcome between sexes, nor did the use of FAO_2 influence the treatment outcome. A better treatment outcome was associated with younger patients. The median age for patients with no residual symptoms was 35 years (30–41) vs 39 years (32–44) for patients with residual symptoms, $P = 0.01$.

Discussion

DELAYED TREATMENT

In this large study, patients who underwent delayed recompression (> 48 h), had similar treatment outcomes compared to those who were treated with HBOT within 48 h. There are several possible explanations for this finding. Firstly, the effectiveness of HBOT remains good even with long delays until the recompression. This conclusion is also supported by a Chinese study, which emphasised that HBOT treatment should not be abandoned even after long delays, since its effectiveness decreased only minimally.[8] In another retrospective study, the time frame of 48 h for HBOT was used, and the findings were similar to our study.[9] Another possibility relates to the fact that in this cohort truly severe cases were rare, and patients exhibiting only mild symptoms (the majority in this cohort) can be expected to fully recover even without recompression. It follows that delay to recompression would be expected to make little or no difference to final outcome in mild cases. This underpins the expert consensus on the possibility for treating mild DCI without recompression, particularly when the treatment facility is far away.[11] Our data support this idea as Finnish diving is mostly done in relatively remote locations where the transportation to HBOT facility takes many hours and even though patients with mild symptoms had a median 12 h longer delay, they still recovered well.

Although spontaneous recovery in mild cases complicates interpretation, the fact remains that patients in our study who had symptoms and were recompressed even after a long delay mainly became asymptomatic when treated

with HBOT. The placebo effect must, of course, be taken into account, but it is possible that HBOT actually had an effect on DCI secondary changes, such as endothelial damage, impaired endothelial function, platelet activation and deposition, leukocyte-endothelial adhesion and possible consequences of vascular occlusion (ischaemia-reperfusion injury and apoptosis), and therefore contributed to healing the injury.[1]

SYMPTOMS AND DELAY

Patients with certain severe symptoms, such as cardio-pulmonary symptoms, had a shorter delay to recompression. This is expected as someone who is very ill is more likely to seek medical attention. In contrast, mild neurosensorial symptoms were associated with a longer delay to recompression. With such mild symptoms, divers are less motivated to seek treatment or might not even realise they are experiencing symptoms of DCI. There is recent evidence of divers self-treating mild DCI with rest, fluids and normobaric oxygen.[16]

OTHER FACTORS AND DELAY

In this study, the use of FAO_2 was related to a shorter delay, but not with a better treatment outcome. A shorter delay was also associated with a higher diver training level. Therefore, there is a possibility that these factors are linked, as better trained divers may have greater awareness of symptoms and more often have FAO_2 on the diving site. However, they also dive deeper, thus they risk developing more severe symptoms and a worse outcome. In other studies, the use of FAO_2 on the diving site has been associated with faster early recovery in DCI.[17] There is a possibility that the use of FAO_2 prevented more serious symptoms from developing, however any such conclusion would require comparison with a control group of patients with similar symptoms and dive history not receiving FAO_2. A shorter delay was associated with fewer required treatments, which is not only more comfortable for the patient, but also important in terms of cost-effectiveness and hospital resources.

SYMPTOMS AND OUTCOME

Tinnitus and hearing impairment were associated with the lowest proportion of patients fully recovered after completion of all HBOT. Both are considered severe symptoms. Motor weakness and visual disturbances were associated with a higher proportion of patients fully recovered, even though they are also considered to be severe symptoms. Nevertheless, incomplete recovery from motor weakness remains a serious problem for the affected divers (33% in this study). In general, other studies report that severe symptoms are linked to a worse treatment outcome.[8,10,18] Mild symptoms such as tingling/itching were associated with a better treatment outcome, which supports the previous studies suggesting good prognosis for mild DCI symptoms.[11]

OTHER FACTORS AND OUTCOME

There was a relationship between the patient's age and full recovery after HBOT, as patients with no residual symptoms were significantly younger although the median age difference was only four years (35 vs 39). It is often suggested that ageing increases the risks of diving.[19,20] Additionally, age has been associated with a worse outcome in multiple studies, even though the additional risk is not considered of great importance.[18,21,22]

LIMITATIONS

As with many retrospective studies, the data collection in the two HBOT centres was not systematic, especially in the early years, which resulted in missing data in some cases. The majority of our patients had mild symptoms. There is a broad consensus that mild cases tend to get better even without recompression. Therefore, such a cohort is poorly suited to show a correlation between recompression delay and treatment outcome. In addition, very short delays to recompression were rare due to long distances to the remoteness of diving sites in Finland. Therefore, conclusions about the effect of very short delays to recompression cannot be drawn. Severe cases, such as dizziness and vertigo, occurred in only small numbers so the proportions of divers recovered (or not) from severe symptoms must be interpreted cautiously. Additionally, there was no long term follow up, thus no way of knowing if residual symptoms resolved later. However, this dataset was quite large and was gathered from only two HBOT centres, which adds to its strength.

APPLICATIONS AND IMPROVEMENTS

Delay in recompression for DCI is still somewhat of a controversial topic in the diving medical community. Prospective data collection with structured methods would give a more robust database and results allowing stronger conclusions. In order to obtain enough data in a relatively short time period, the collection should be done from multiple HBOT centres with the same treatment protocols. When considering treatment delays and the treatment outcome, an inevitable question arises as to whether the efficacy of HBOT could be evaluated more precisely. This could provide guidance when patients can really benefit from HBOT, which in turn may provide a more cost-effective evacuation and treatment plan. An example of such work appeared in a recent study which found that a simple scoring system for spinal cord DCS helped define the urgency of evacuation of the injured diver.[23]

Conclusions

Recompression and HBOT for DCI remains effective, even after a 48 h delay. Therefore, treatment should not automatically be discounted in the case of longer delays. A short delay to HBOT improves the efficacy of the

treatment in general, indicated by fewer required numbers of treatments. The overall efficiency of HBOT should be evaluated more systematically especially in cases of milder symptoms and delayed treatment.

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