Effect of hyperbaric oxygen treatment on patients with reduced left ventricular ejection fraction

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Abstract
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Introduction: Hyperbaric oxygen treatment (HBOT) is available to a wide spectrum of patients, many with significant co-morbidities. Considering its effects on cardiac physiology and reports of pulmonary oedema following exposure, concerns exist about the safety of patients with compromised cardiac function. Few studies have described adverse events occurring during HBOT and even fewer reports address events arising in the hours following HBOT. A relation between adverse events and cardiac function has not been established. As medical guidance is limited, we aimed to evaluate the risk for patients with reduced left ventricular ejection fraction (LVEF) receiving HBOT.

Methods: This retrospective chart review of patients receiving HBOT from April 2003 through December 2019 at our hospital was designed to describe clinical characteristics of patients and to identify adverse events during HBOT and within 24 hours after HBOT. Patients ≥ 40 years of age with a documented LVEF of ≤ 40% were included. Data are presented as mean (SD) [range] or counts, as appropriate.

Results: A total of 23 patients were included in the final analysis, 2 (1) [0–4] patients per year. Patients received 25 (19) [1–60] treatments. Two patients had an episode of acute decompensated heart failure possibly linked to HBOT.

Conclusions: This study described the clinical characteristics of patients with reduced LVEF receiving HBOT and showed reassuring results, with a majority of patients with reduced LVEF tolerating HBOT well. Prospective research is required to more fully assess the risk.

Introduction

Hyperbaric oxygen treatment (HBOT) is an adjunctive modality that has shown benefits for a wide variety of pathologies. The Undersea and Hyperbaric Medical Society (UHMS) has identified 14 approved indications.[1] The European Committee on Hyperbaric Medicine (ECHM) agrees with a majority of the UHMS indications (except for severe anaemia), and provides a broader list of indications.[2] HBOT is an option for a large spectrum of patients, some with significant co-morbidities, including a high prevalence of cardiovascular problems.

HBOT uses 100% oxygen delivered at a pressure of 202.6–303.9 kPa (2–3 atmospheres absolute [atm abs]). Hyperoxia acts in numerous ways, many affecting haemodynamics and cardiac physiology. It is potentially responsible for an increased oxidation of nitric oxide (NO) radicals, which results in arteriolar vasoconstriction that increases systemic vascular resistance. Hyperoxia also stimulates vagal activity, causing bradycardia. An uneven effect on right and left ventricular contractility, a decrease in left ventricular compliance, and an increased oxidative myocardial stress[3] that possibly persists up to one hour after HBO exposure[4] have also been described. A measure of this myocardial stress has been evaluated indirectly with N-terminal pro-B-type natriuretic peptide (NT-proBNP) in diabetic patients without cardiovascular disease, before and after exposure to HBOT. An increase in NT-proBNP was interpreted to mean that a considerable ventricular wall stress may be induced by HBOT.[5] However, these findings must be considered preliminary since brain natriuretic peptide (BNP) and NT-proBNP levels tend to be higher in persons with diabetes, making it difficult to extrapolate these observations to a non-diabetic population.

Bradycardia, decreased ventricular compliance and myocardial stress are all responsible for a decreased cardiac

output that has to overcome an increased afterload caused by a rise in systemic resistance. These effects could put patients in a hyperbaric chamber at risk of relative volume overload during HBOT. When hyperoxia ceases at the end of treatment, a reversal in the haemodynamic changes develops; peripheral resistances drop and the vagal stimulation causing bradycardia ceases. The fall in peripheral resistance and the increase in heart rate will increase cardiac output in order to maintain an adequate blood pressure, causing another strain on the heart and another risk of relative volume overload after exposure.

When these changes are applied to an already compromised left ventricle, they can potentially exceed the capacity of the ventricle to further manage pressure, putting a patient with reduced left ventricular ejection fraction (LVEF) at higher risk of pulmonary oedema. This rare but potentially life-threatening complication has been reported in HBOT with an estimated incidence of 0.1%.[3] There are at least four case reports of pulmonary oedema associated with HBOT in patients with cardiac disease with reduced LVEF or significant valvulopathy.[6,7] The moment when the first symptoms appear may be important. Except for one case, symptoms all occurred during HBOT. One case described symptoms that developed immediately after decompression, as the patient was exiting the chamber. The timing in these cases could indicate a risk for pulmonary oedema during or immediately after the conclusion of the treatment.

These observations raise important concerns about the safety of patients with compromised cardiac function receiving HBOT. Medical guidance for at-risk patients with reduced LVEF is limited. The objective of this study was to gain a better understanding of the risk, in term of cardiovascular impact, of HBOT for patients with reduced LVEF.

The specific aims of this study were to:
1) Describe the pre-HBOT clinical characteristics of patients with reduced LVEF being treated in the hyperbaric chamber of our facility; and
2) Identify cardiovascular adverse events, including acute decompensated heart failure, during HBOT and within 24 hours after HBOT that could have been triggered by HBOT or the cessation of HBOT.

Methods

The Comité d'éthique de la recherche (CER) du CISSS de Chaudière-Appalaches approved the retrospective study. A waiver of consent was provided for the review of charts of patients receiving HBOT in the hyperbaric chamber of Hôtel-Dieu de Lévis in Chaudière-Appalaches, Québec, between April 2003 and December 2019. The treatments were received in a monoplace chamber from April 2003 to June 2012, then in a multiplace chamber. Oxygen was delivered via a hood for every patient.

Inclusion criteria were: age ≥ 40 years at the time of the treatments and a documented reduced LVEF, with an imaging modality (echocardiogram, nuclear stress test, or cardiac MRI) reporting a LVEF of ≤ 40%; or with a LVEF of ≤ 40% written in the patient's medical history if no imaging report was available. Patients were treated as new cases if there was more than 12 months between treatment cycles, considering that their basic characteristics, indication for HBOT, and LVEF could have changed over time. Patients under the age of 40 years of age were excluded due to the low prevalence of heart failure in the younger population.

Patient selection was performed by an internal medicine resident, using the hyperbaric chamber database and the Hôtel-Dieu de Lévis' electronic charts. Charts from other facilities were not accessible. The hyperbaric chamber database was first used to screen patients.

When a patient met the age inclusion criterion, a list of his or her medical conditions was evaluated. The medical summary of the hyperbaric chamber database was first used when available. The hospital's charts were then screened for more details on cardiac function based on imaging and consultations, mainly in cardiology and internal medicine. Every LVEF report in the patient's chart was recorded. The closest report available either before, during, or after HBOT was considered as the patient's LVEF during the treatments, with a maximum time period of 60 months before the first treatment and two months after the last treatment. If LVEF results were available from two different imaging modalities within one month, the lowest valid value was registered as the patient's LVEF. If there was a difference of more than five percent between the two imaging modalities, the cases were reviewed by the research team. A conservative position was taken to exclude cases that could have been falsely low. Thus, if the closest report to HBOT stated a LVEF > 40%, the patient was excluded from the study.

The hospital's charts were used to find basic clinical characteristics (age, sex, region of origin, co-morbidities, aetiology of heart failure) and any reported adverse events. Every treatment received by a patient was recorded as a single entry and adverse events were associated with specific treatments.

Adverse events were first classified according to their temporal proximity to HBOT. Adverse events 'during HBOT' occurred when the patient was in the hyperbaric chamber, from the beginning of the treatment through to exiting. Adverse events of particular interest were signs and symptoms of a cardiovascular complication, such as acute pulmonary oedema, progressive dyspnoea, chest pain, symptoms of peripheral oedema or neurologic symptoms such as confusion. The hospital's chart provided access to a treatment sheet filled by a hyperbaric centre nurse following each treatment, with descriptions of vital signs (heart rate, blood pressure, oxygen saturation) and any symptoms.

Adverse events 'within 24 hours after HBOT' occurred from the moment the patient was out of the hyperbaric chamber up to 24 hours later, or until re-entering the chamber if the next treatment began within 24 hours.

Adverse events were described with all available details. These included charted signs and symptoms, patient reports, and medical reports with description of symptoms and final diagnosis of any visit to the emergency room. Objective elements that suggested an investigation done for a possible cardiovascular event were noted. Imaging modalities (chest X-ray, electrocardiogram, telemetry reports, and echocardiograms) and laboratory values (troponins and BNP) were assessed.

Once data collection was complete, the research team reviewed every adverse event to evaluate the cardiovascular relevance. Adverse events were classified as '*inconsequential from a cardiovascular perspective*', '*probably not linked to HBOT*', and '*possibly linked to HBOT*'. Adverse events considered inconsequential were symptoms and complications not specific to a cardiovascular event, including common symptoms associated with HBOT or symptoms that the medical team did not considered as needing further investigation. They included otalgia, anxiety, diaphoresis, discomfort, and complications such as hypoglycaemia and convulsions. Adverse events of principal interest were signs, symptoms, and objective elements suggesting an acute cardiovascular event (dyspnoea, chest pain, peripheral oedema, electrocardiogram (ECG) changes, chest X-ray, troponins, BNP, emergency room and cardiology consults and hospitalisations). These adverse events were discussed within the research team to classify them as probably not linked to HBOT or possibly linked to HBOT. They were classified as probably not linked to HBOT when an alternative diagnosis was more probable or when the results of subsequent investigations were normal. Adverse events that could not be satisfactorily explained by another condition or with abnormal test results were classified as possibly linked to HBOT. Cardiovascular adverse events possibly linked to HBOT were further discussed to assess their specificity regarding acute decompensated heart failure.

Data are presented as mean (SD) [range] or counts and percentages, as appropriate. Fisher's exact tests were used to compare adverse event rates in the first five serial HBOT treatments (considered to reflect inexperienced patients) versus the sixth and more serial HBOT treatments (considered to reflect experienced patients), to determine if experience with the hyperbaric environment and procedures played a role in adverse incident rates. Significance was accepted at $P < 0.05$.

Figure 1
Patient selection paradigm

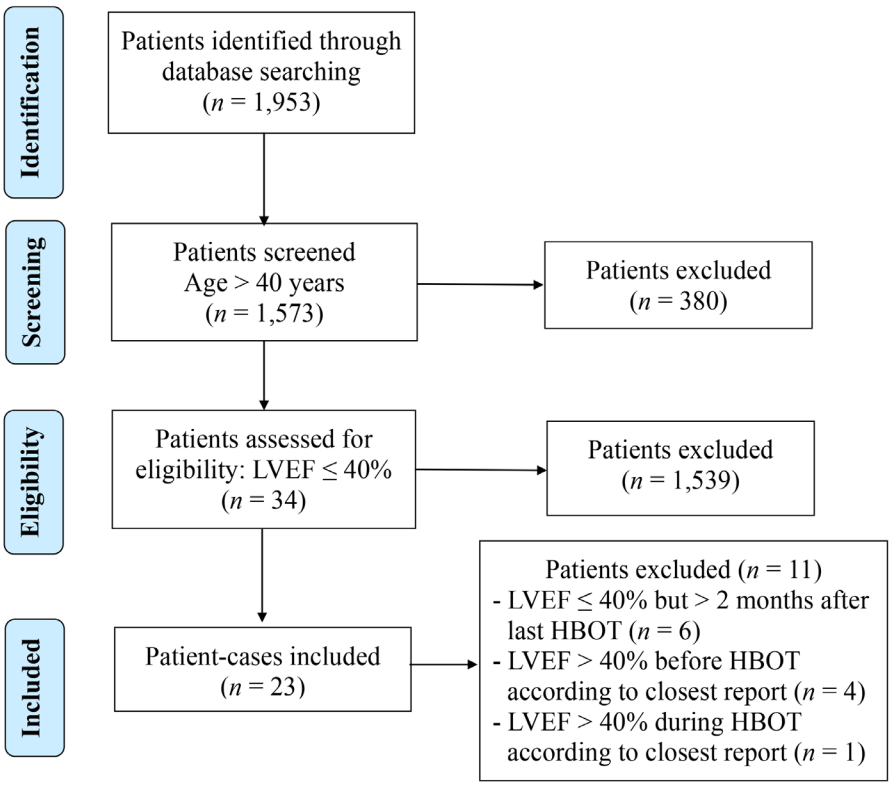

Table 1

Patient and HBOT characteristics; COPD – chronic obstructive pulmonary disease; HBOT – hyperbaric oxygen treatment. Note: percentages are calculated on small sample sizes

Parameter	All	Male	Female
Cases, *n* (%)	23	20 (87)	3 (13)
Age in years, mean (SD) [range]	69 (8) [51–83]	70 (8) [51–83]	63 (5) [58–68]
Treatments, total	564	477	87
Treatments, mean (SD) [range]	25 (19) [1–60]	24 (19) [1–60]	29 (26) [1–53]
Indication for HBOT, *n* (%)			
Improve wound healing	13 (57)	10 (50)	3 (100)
Osteoradionecrosis	6 (26)	6 (30)	0 (0)
CO intoxication	2 (9)	2 (10)	0 (0)
Osteomyelitis	1 (4)	1 (5)	0 (0)
HBOT challenge	1 (4)	1 (5)	0 (0)
Aetiology of heart failure, *n* (%)			
Ischaemic	21 (91)	18 (90)	3 (100)
Ischaemic vs. Takotsubo	1 (4)	1 (5)	0 (0)
Ischaemic + hypertensive	1 (4)	1 (5)	0 (0)
Comorbidities, *n* (%)			
Cardiovascular disease	23 (100)	20 (100)	3 (100)
Peripheral artery disease	17 (74)	15 (75)	2 (67)
Hypertension	14 (61)	11 (55)	3 (100)
Dyslipidaemia	14 (61)	11 (55)	3 (100)
Diabetes mellitus	13 (57)	10 (50)	3 (100)
History of cancer	9 (39)	8 (40)	1 (33)
Pacemaker	8 (35)	7 (35)	1 (33)
Chronic kidney disease	6 (26)	4 (20)	2 (67)
Atrial fibrillation	5 (22)	4 (20)	1 (33)
Obesity	4 (17)	2 (10)	2 (67)
COPD	3 (13)	3 (15)	0 (0)
Hypothyroidism	3 (13)	2 (10)	1 (33)
Anaemia	3 (13)	2 (10)	1 (33)
Cirrhosis	1 (4)	1 (5)	0 (0)
Sleep apnoea	1 (4)	1 (5)	0 (0)
Epilepsy	1 (4)	1 (5)	0 (0)

Results

A total of 1,953 patients received at least one HBOT treatment between April 2003 and December 2019 (Figure 1). Of these, 380 were excluded because they did not meet the age criteria, 1,539 because they did not have a documented reduced LVEF ≤ 40%, and 11 because they had no reports within the inclusion range for HBOT timing. Two patients were entered as separate cases for two different treatment cycles, with time between cycles of 43 and 71 months. The final study group consisted of 23 patients (20 male, three female; 69 (8, [51–83]) years of age), for an accrual rate of 2 (1, [0–4]) per year.

The clinical characteristics of the study group are presented in Table 1. Patients received 25 (19, [1–60]) treatments, with the most frequent indications for HBOT being to improve wound healing (*n* = 13, 57%). Every patient had ischaemia as the aetiology of heart failure. One patient had an alternative diagnosis of Takotsubo, a usually transient stress cardiomyopathy. The most frequent co-morbidities were cardiovascular disease (all 23 patients) and peripheral artery disease (74%). Direct access to charts was available for 57% of the patients.

Two patients had imaging modalities done within a month of each other with marginal LVEF differences (28% vs. 30–35% and 30% vs. 35%). Two patients had greater

Table 2
Reporting and quantification of left ventricular ejection fraction; HBOT – hyperbaric oxygen treatment. Note: percentages are calculated on small sample sizes

Parameter	All *n* = 23	Male *n* = 20	Female *n* = 3
Available report	16 (70)	14 (70)	2 (67)
Unavailable report	7 (30)	6 (30)	1 (33)
Imaging modality, *n* (%)			
Transthoracic echo	14 (61)	12 (60)	2 (67)
Nuclear stress test	5 (22)	5 (25)	0 (0)
Transoesophageal echo	1 (4)	1 (5)	0 (0)
Unknown	3 (13)	2 (10)	1 (33)
Left ventricular ejection fraction, *n* (%)			
40	4 (17)	3 (15)	1 (33)
35–39	6 (26)	5 (25)	1 (33)
30–34	8 (35)	7 (35)	1 (33)
25–29	3 (13)	3 (15)	0 (0)
20–24	2 (9)	2 (10)	0 (0)
Time from HBOT, *n* (%)			
1–2 month after HBOT	1 (4)	1 (5)	0 (0)
During HBOT	5 (22)	5 (25)	0 (0)
Before HBOT	17 (74)	14 (70)	3 (100)
< 12 months before	12 (52)	10 (71)	2 (67)
13–24 months before	2 (9)	2 (14)	0 (0)
25–60 months before	1 (4)	0 (0)	1 (33)
Unknown	2 (9)	2 (14)	0 (0)

differences in their imaging reports; one remained in the study group with the highest LVEF value kept, and one was excluded because his highest LVEF value was above the cut-off. Imaging reports were available for 16 patients (70%) (Table 2). In three patients (13%), the LVEF value was based on data found in the chart before HBOT but the type of imaging modality and the reports could not be found. The time between the report and HBOT was 9 (8) months for reports available before HBOT (*n* = 15, 65%). The LVEF value was found in a time period of 12 months before HBOT until two months after the last treatment in 18 patients (78%).

Sixteen distinct patients (70%) experienced at least one adverse event of any type in the study period (Table 3), with 3 (6, [0–25]) reported adverse events per patient.

Adverse events considered as cardiovascular in nature but classified as probably not linked to HBOT (*n* = 31, 32%) were found in five patients (22%). These included dyspnoea, confusion, chest pain, and hospitalisation potentially explained by another condition as stated in the chart by the medical team or with normal investigations. For example, one patient had multiple episodes of dyspnoea and mild pulmonary oedema on a chest X-ray with no temporal association with HBOT that was explained by his altered renal function necessitating chronic dialysis.

Adverse events considered as cardiovascular in nature and possibly linked to HBOT (*n* = 17, 18%) were reported in four distinct patients (17%), (60–74 years of age), three of whom reported an adverse event within 24 hours after HBOT, and one who report an adverse event during HBOT and another within 24 hours after HBOT. Two of them received HBOT to improve wound healing, one for osteomyelitis, and one for carbon monoxide (CO) intoxication. They received 1–38 treatments. The reduced LVEF was due to coronary artery disease, with one patient having a possible diagnosis of Takotsubo cardiomyopathy. One patient (male, LVEF 36%) had a diagnosis of non-ST elevation myocardial infarction (NSTEMI) following HBOT. Over a period of three weeks, he presented multiple episodes of chest pain and dyspnoea within 24 hours after HBOT, and one episode during HBOT. Symptoms were reproducible with exercise.

Table 3
Occurrence and classification of adverse events, based on data from 23 patients and 564 patient treatments; *A total of 16 distinct patients had an adverse event of any type. Patients may be entered twice in the table if they reported adverse events in different categories

Parameter	Cardiovascular adverse events				Non-cardiovascular adverse events		Total
	Possibly linked to HBOT		Probably not linked to HBOT				
	During HBOT	≤ 24 h after HBOT	During HBOT	≤ 24 h after HBOT	During HBOT	≤ 24 h after HBOT	
Events (*n*)	1	16	4	27	49	0	97
Patients* *n* (%)	1 (4)	4 (17)	2 (9)	3 (13)	11 (48)	0 (0)	
Treatments *n* (%)	9 (2)		23 (4)		38 (7)		70 (12)
Adverse events per patient overall, mean (SD) [range]							3 (6) [0–25]

Investigations done by the hyperbaric team were always negative. Twelve hours after his 38th treatment, he presented to the ER describing chest pain that began two hours before. Troponins were positive, but no signs of pulmonary oedema were seen on the chest X-ray. He was treated for a NSTEMI, evaluated with coronary angiography, and benefited from revascularisation.

A second patient (male inpatient, LVEF 30%), became confused during his third HBOT treatment and was hypoxaemic and febrile when sent back to his room two hours post-HBOT. He expressed no complaints, and the chart had no mention of decompensated heart failure. Eighteen hours later, a chest X-ray showed mild pulmonary oedema and the oxygen requirements were the same as before HBOT. HBOT was discontinued. His LVEF improved one month later to 66%, making the diagnosis of Takotsubo cardiomyopathy possible.

Finally, two patients were sent to the intensive care unit (ICU) with possible signs of decompensated heart failure. The first (male, LVEF 39%) was treated for CO intoxication and then sent to the ICU immediately after treatment because of neurologic symptoms (somnolence and agitation). Considering the elevated troponins, the cardiology team concluded that myocardial necrosis secondary to CO intoxication was more probable than acute coronary syndrome. The echocardiogram done on the day after HBOT showed reduced LVEF, possibly chronic, since regional wall motion abnormality was mentioned, but no older imaging report was available to confirm this. Chest X-rays before and after HBOT were similar, with no signs of acute decompensated heart failure. A diagnosis of cardiomyopathy secondary to CO intoxication was written in his chart. The second patient (male, LVEF 20–25%) was transferred to the ICU 18 hours after HBOT. His first treatment in the morning was well tolerated and the evening was unremarkable according to the charts. Twelve hours after HBOT, he developed tachypnoea, hyperthermia, hypotension (80/50 mm Hg) with desaturation, and an altered level of consciousness. He was transferred to the ICU and volume repletion started. A cardiology consultation completed on the following day noted pulmonary oedema on the chest X-ray and diffuse ischaemia on the ECG, both done 18 hours after HBOT. Diuretics were administered. The final diagnosis of the ICU team was mixed shock; septic and cardiogenic.

The majority of adverse events ($n = 49$, 51%) were classified as inconsequential from a cardiovascular perspective. They included non-specific symptoms such as otalgia (the most common), headache, discomfort, diaphoresis, nausea, vomiting, and anxiety. They led to premature cessation of a single treatment in one patient and to the cessation of HBOT in two patients.

Fisher's exact testing showed a greater rate of adverse events in patients classified as inexperienced compared to those classified as experienced (26/89 [29%] vs. 44/475 [9%]; $P < 0.0001$).

Discussion

These results show that a majority of patients identified with LVEF between 20 and 40% appeared to tolerate HBOT without serious cardiovascular events.

Higher rates of adverse events were reported in inexperienced patients, mostly inconsequential adverse events from a cardiovascular perspective. Three patients reported adverse events possibly linked to HBOT in the first five serial treatments.

HBOT can impart potentially important stressors on heart physiology during or following exposure. BNP levels are a useful marker of cardiac failure as it increases rapidly in response to myocardial wall stress due to pressure overload, but these values were not documented in any charts. This is not surprising since BNP levels are not routinely assayed in stable patients without signs of acute decompensation in heart failure. It is possible that such assays could be helpful to better understand potential repercussions of HBOT in patients with reduced LVEF.

Patients with compromised cardiac function demonstrate fragility and are at risk of decompensation when confronted with any number of stressors, not limited to anaemia, arrhythmia, infections, ischaemia, intoxications, volume overload, and medication changes. The importance of individual and/or combined stressors cannot be determined in the present work. With the data available, we believe that three of the four distinct patients with cardiovascular events possibly linked to HBOT had other factors that could explain the event, such as significant coronary artery disease, infection or CO intoxication. Three of the four distinct patients with reported adverse events possibly linked to HBOT had signs or symptoms of ischaemia that manifested at distance from the pressurisation. These symptoms were attributed to an acute coronary syndrome, rather than to HBOT. However, we cannot exclude that some of these symptoms can also be attributed to HBOT. It is not surprising that the treatment itself was well tolerated; by delivering 100% oxygen at high pressure, HBOT dramatically increases dissolved blood oxygen content, improving tissue oxygenation. HBOT has been described as beneficial for myocardial infarction following CO intoxication.[8]

Acute decompensated heart failure was not reported during HBOT, nor immediately upon cessation of HBOT. This result contrasts with reports by other authors,[6,7] in which all patients reported symptoms during their treatments or immediately after HBOT. Only one patient in our study had an adverse event possibly linked to HBOT during HBOT. The patient had dyspnoea during one treatment, but the final diagnosis of NSTEMI was made many treatments later, 12 hours after HBOT. One patient was immediately transferred to the ICU after HBOT, but for neurologic symptoms without signs or symptoms of acute decompensated heart failure. All other adverse events reported happened within 24 hours after HBOT, occurring between two and 12 hours, and included no mention of symptoms developing in the minutes when the patients came out of the chamber.

Acute heart failure was reported for two patients within 24 hours following HBOT. Analysis of the data could not isolate HBOT as a causal agent as concomitant factors were present in every reported case of adverse events possibly linked to HBOT. The patient with Takotsubo cardiomyopathy possibly showed signs of decompensation related to HBOT, but missing data prevents us from making this conclusion and we cannot exclude that HBOT could be a precipitating factor.

LIMITATIONS

Our study had several limitations, primarily related to data completeness. Because of the absence of a documented LVEF in many charts, patients with reduced LVEF may have been excluded. Accepting imaging reports that were somewhat removed from HBOT may also have introduced error. The majority of medical records held outside of Hôtel-Dieu de Lévis charts were unavailable for assessment. Patients transferred from another medical centre often had only a brief description of their co-morbidities, without any report of their cardiac function. Even with a majority of patients living in the Chaudière-Appalaches' region, consults done in another hospital or clinic were not available. Any hospitalisation, consult to the emergency room, imaging modality or laboratory value done outside Hôtel-Dieu de Lévis hospital was likely missed. Internal records were also incomplete in some cases. For adverse events occurring during HBOT, signs and symptoms were often found in the chart, but information about more specific characteristics of symptoms, vitals signs and/or diagnosis was often lacking. Some patient files also had imaging reports and/or laboratory values without description of symptoms or reason for these investigations. It is also possible that some adverse events, most likely minor ones that were not considered concerning, were not reported to or documented by the medical team.

Conclusions

HBOT is used to treat many conditions, often in patients with severe co-morbidities. It is not uncommon for the medical team of the hyperbaric chamber to evaluate the eligibility to HBOT of patients who have reduced LVEF.

Concerns have been expressed over a possible risk of precipitating heart failure in patients with reduced LVEF, but medical guidance is not firmly established. We retrospectively evaluated a group of patients with a LVEF ≤ 40% receiving HBOT with reassuring results; the majority of these patients tolerated HBOT well and concomitant stressors and co-morbidities unrelated to the hyperbaric treatment could, at least partially, explain the small number of cases of decompensated heart failure that we considered possibly related to HBOT.

It is possible that HBOT may play a role in increasing the risk of acute decompensated heart failure for patients with a reduced LVEF, but we did not see strong evidence of this. We believe that a low LVEF should not be considered an absolute contra-indication to HBOT, but the risk-benefit relationship must still be considered on an individual patient basis. Prospective studies employing systematic cardiological evaluation would provide additional useful information.

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