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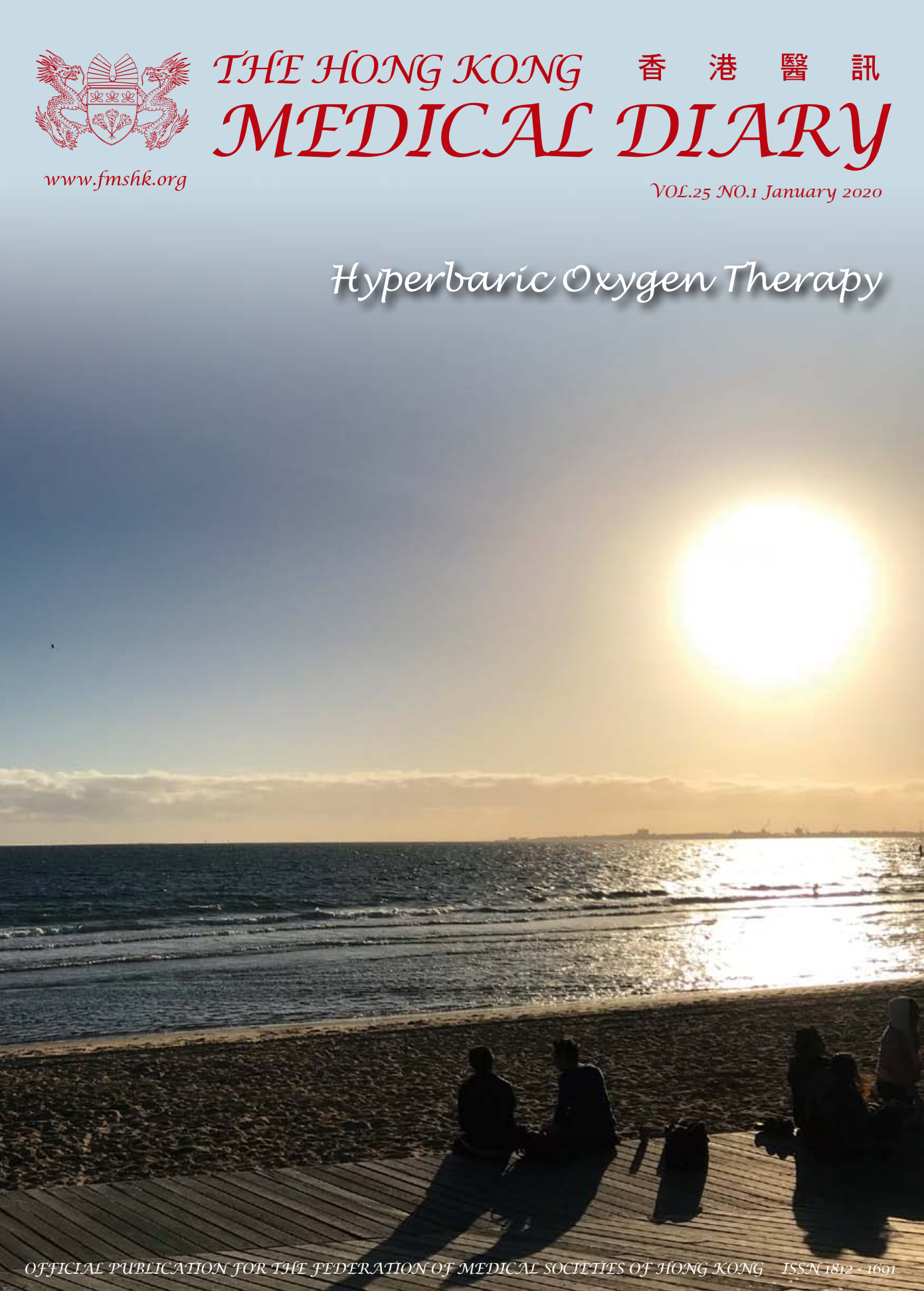
Hyperbaric Oxygen Therapy



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The Cover Shot

This photo of St. Kilda was shot during the Hyperbaric Medicine training in the Hyperbaric Unit of Alfred Hospital, Melbourne. St Kilda Beach is located south of the Melbourne city centre. It is Melbourne's most famous beach. It is located at the north-east corner of Port Phillip and is protected from ocean swell, though still affected by strong westerly winds. The beach harbours one of the colonies of the little penguins in Melbourne. There is a good collection of nice sea-view restaurants at the beachside.

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Welcome to The New Hyperbaric Medicine Era in Hong Kong

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Co-Editors

Dr Joe KS LEUNG Dr Jeffrey CW CHAU

Hyperbaric Oxygen Therapy (HBOT) is a medical therapy through which the patient is being treated inside a big metal chamber with oxygen/compressed air pressure higher than the sea-level atmosphere. HBOT has been well-studied for many years and has been used in clinical practice in many developed and developing countries such as United States, Australia, European countries, Singapore, Japan, Taiwan and China. It has been shown to be especially useful in chronic unhealed vascular wounds, in delaying injury of radiated tissue and in many emergency conditions such as carbon monoxide poisoning and gas embolism.

However, Hong Kong, as an international city with world-class standard of medical service, witnessed the establishment of the first HBOT service in a public hospital as recently as in September 2018. This first hospital-based HBOT centre in Hong Kong is situated in the Pamela Youde Nethersole Eastern Hospital on Hong Kong Island east. As the first and only hospital-based centre in the territory, it receives consultations from all public hospitals in Hong Kong.

HBOT is useful in many different conditions, including life-threatening, emergency and routine indications. Hence, various specialties such as Intensive Care, Orthopaedics, Oncology, Ear Nose & Throat, Toxicology as well as Ophthalmology have been involved. Liaison among all these specialties across the city is an important step towards effective consultation on and transfer and treatment of relevant patients.

Being members of the pioneer group in kickstarting this service, hyperbaric doctors; nurses and technicians need to have adequate training in order to provide safe service. Many overseas standards have been incorporated in order to formulate the best standard which is suitable for the Hong Kong setting. Moreover, many oversea experts have been helping us in building this centre, providing expertise on infrastructure, facilities and equipment as well as guidelines and staff training.

The centre has been in operation for more than a year, during which time basic operation and logistics of patient consultation, transfer and treatment have been smoothened. In the coming years, we will focus on enhancing indications for intervention, clinical service and patient monitoring, as well as staff training and recruitment.

Role of Hyperbaric Oxygen Therapy in Carbon Monoxide Poisoning

Dr Joe KS LEUNG

Director, HBOT Centre, PYNEH

Dr Joe KS LEUNG

This article has been selected by the Editorial Board of the Hong Kong Medical Diary for participants in the CME programme of the Medical Council of Hong Kong (MCHK) to complete the following self-assessment questions in order to be awarded 1 CME credit under the programme upon returning the completed answer sheet to the Federation Secretariat on or before 31 January 2020.

INTRODUCTION

Hyperbaric oxygen therapy (HBOT) is widely accepted as the life-saving treatment of decompression illness. Yet, its use in acute carbon monoxide poisoning has remained controversial because of inconsistent findings in clinical trials.

HBOT has been used in a variety of acute poisoning settings including those caused by carbon monoxide (CO), methylene chloride, hydrogen sulphide and carbon tetrachloride as well as gas embolism resulting from hydrogen peroxide ingestion, and methaemoglobinaemia.[1] This paper focuses on CO poisoning as it remains a major cause of death in non-medicinal poisoning[2] and often results in persistent neurological sequelae (PNS) or delayed neurological sequelae (DNS)[3].

CARBON MONOXIDE POISONING

The pathophysiology of CO poisoning is complex; in brief, CO causes tissue hypoxia by forming carboxyhaemoglobin (COHb) and shifting the oxyhaemoglobin dissociation curve to the left. It also binds to various heme proteins, impairs mitochondrial function, causes the release of nitric oxide (NO) and free radicals, and triggers inflammation through a myriad of mechanisms independent of hypoxia.[3-6]

Oxygen is the standard treatment. It works by reversing hypoxia, competing with CO for haemoglobin binding, and shortening the half-life of COHb (from 320 minutes on room air to about 70 minutes on 100% O2 at 1 atmosphere absolute (ATA)). HBOT further reduces the half-life of COHb to 20 minutes (on 100% O2 at 2.5 ATA), and increases the amount of dissolved oxygen in the plasma.[7] Recent studies have shown that HBOT also restores mitochondrial function[8], reduces brain lipid peroxidation,[9] and inhibits CO-induced inflammatory response by inhibiting β2 integrin-mediated neutrophil adhesion to brain microvasculature and lymphocyte sensitisation to myelin basic protein.[10-12]

HBOT AND CARBON MONOXIDE POISONING

HBOT was first used for CO poisoning in 1960[13] but has remained controversial owing to the conflicting results of randomised control trial (RCTs) on its effect on DNS, which are summarised in Table 1.[14-19] A 2011 Cochrane review of 6 RCTs and 1361 participants showed that HBOT does not have a significant benefit in pooled random-effect meta-analysis (Odds ratio [OR] for neurological deficits 0.78, 95% confidence interval CI 0.54 to 1.12). The reviewers noted that the "significant methodologic and statistical heterogeneity", and "design or analysis flaws" of the included trials warrant cautious interpretation of the results.[20] The American College of Emergency Physicians (ACEP), based on systematic literature review, stated that "It remains unclear whether HBO2 therapy is superior to normobaric oxygen therapy for improving long-term neurocognitive outcomes" in CO-poisoned patients.[21] However, a recent large population-based retrospective cohort study in Taiwan involving 7,278 patients showed that HBOT was associated with reduced mortality in patients with CO poisoning after adjusting covariates, especially in those who were younger than 20 years of age and those with acute respiratory failure.[22] These findings have added weight to support HBOT use in CO poisoning.

Nevertheless, the threshold for HBOT for CO poisoning varies across different centres[23], and uncertainties exist regarding the optimal chamber pressure, number and frequency of sessions, and time window after CO poisoning. In particular, pregnant women pose special challenges as they are at high risks of adverse effects of both CO and HBOT, and yet research data in this group are lacking since they have been excluded from most prospective trials. In view of the devastating fetal outcomes of maternal CO poisoning, including stillbirth, fetal Central Nervous System (CNS) damage and anatomic malformation, COHb thresholds for HBOT are often set lower for pregnant patients (COHb 15-20%) than for non-pregnant patients (COHb ≥ 25%), and HBOT is often considered indicated when there is evidence of fetal distress. Clinical experience in Russia supports the assertion that HBOT is safe during pregnancy.[24, 25] Yet its effect in averting CO-related adverse fetal outcome remains unclear. In CO-poisoned children, indications for HBOT are similar to that for adults, though they have not been evaluated systematically.[26] HBOT has been used safely in paediatric patients, but special paediatric considerations are needed. Readers are referred to the review by Liebert for further information.

Before more convincing evidence is available, clinicians are advised to weigh the benefits, risks, and costs of HBOT carefully on a case-by-case basis when making decision on HBOT for CO-poisoned patients. It is commonly suggested that HBOT should be reserved for situations where there are indicators of higher severity CO poisoning, such as loss of consciousness, abnormal neurologic signs, cardiovascular dysfunction, or severe acidosis or if patients are over 35 years of age or have suffered a prolonged exposure (e.g. >24 hours) or have a high COHb level (e.g. ≥ 25%.). The most recent UHMS Guidelines, however, recommend that HBOT be considered for all cases of acute symptomatic CO poisoning, given the lack of predictive factors for poor long-term outcomes or for which patients might receive the greatest benefit from HBOT.

Table 1. Summary of randomised controlled trials comparing hyperbaric oxygen and normobaric oxygen for carbon monoxide poisoning (Summarised from reference 14 – 19)

Author/Year of publication	Sample size	Study design	Intervention	Key Results	Issues
Raphael et al. 1989[27]	629	RCT Unblinded	If no LOC, 2 h HBOT (2.0 ATA) + 4 h NBO vs 6 h NBO; if LOC, 1 HBOT session vs 2 HBOT sessions	No difference in recovery between groups at 1 month	Lack of objective assessment of neurological sequelae; many patients received HBOT > 6 h after poisoning; suboptimal pressure (2 ATA)
Thom et al. 1995[28]	65	RCT Unblinded	HBOT (2.8 ATA for 30 min, then 2 ATA O_2 for 90 min) vs NBO till symptom resolution (mean 4.2 h)	No DNS in HBOT group vs 23% in NBO group ($p<0.05$); NNT = 4.3	Mild to moderate CO poisoning presented within 6 h; excluded LOC or cardiac compromise; small sample size; lack of sample size calculation
Mathieu et al. 1996[29]	575	RCT Unblinded	HBOT (2.5 ATA) for 90 min vs 12 h NBO	Significant difference in neurologic symptoms at 3 months (HBOT: 9% vs NBO: 15%) but not at 1 month, 6 months and 1 year.	Abstract only; unclear bias in randomisation, allocation concealment and selective reporting; no report of drop-outs
Scheinkestel et al. 1999[30]	191	RCT Double-blind Sham therapy	3 daily 1 h HBOT sessions (2.8 ATA) vs 3 d of NBO (100% O2 at 1 ATA sham dives). Both groups received continuous high-flow O2 for 3 d. 3 additional courses of original treatment for patients with "poor outcome"	HBOT group has a significant worse outcome in the learning test; DNS restricted to HBO group	Large number of suicidal attempt; delayed HBO for > 6 h; high lost to follow-up rate; continuous high-flow O2 not accepted as standard practice
Weaver et al. 2002[31]	152	RCT Double-blind Sham therapy	3 HBOT sessions (3 ATA for 1 h, then 2 ATA for 1 h in session 1, 2 ATA for 2 h in session 2 and 3) in 24 h vs 3 sham chamber sessions (100% O2 at 1 ATA in session 1 and normal air at 1 ATA in session 2 and 3)	Less frequent cognitive sequelae (25 vs 46%) at 6 weeks, 6 months and 12 months	Excluded if > 24h after CO exposure; NBO group had a higher prevalence of cerebellar signs at baseline; trial stopped after the third interim analysis; apparent change in the primary outcome
Annane et al. 2011[32]	385	RCT Unblinded	Trial A (n=179; patients with transient LOC): 1 HBOT session (2.0 ATA) + 4 h NBO vs 6 h NBO Trial B (n=170; patients with initial coma): 2 HBOT sessions + 4 h NBO vs 1 HBOT session + 4 h NBO	Trial A – no difference in "complete recovery" at 1 month (58% vs 61%) Trial B – "complete recovery" rate 47% with 2 HBO sessions vs 68% with 1 HBO sessions at 1 month	HBOT at 2 ATA only; excluded suicidal attempt and nondomestic CO poisoning; lack of objective assessment of neurological sequelae; outcome assessment at 1 month only; premature trial termination because of harm and futility in the interim analysis

Abbreviations: RCT = randomised controlled trial; LOC = loss of consciousness; HBOT = hyperbaric oxygen therapy; ATA = atmosphere absolute; PNS = persistent neurolgical sequelae; DNS = delayed neurological sequelae

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MCHK CME Programme Self-assessment Questions

Please read the article entitled "Role of Hyperbaric Oxygen Therapy in Carbon Monoxide Poisoning" by Dr Joe KS LEUNG and complete the following self-assessment questions. Participants in the MCHK CME Programme will be awarded CME credit under the Programme for returning completed answer sheets via fax (2865 0345) or by mail to the Federation Secretariat on or before 31 January 2020 Answers to questions will be provided in the next issue of The Hong Kong Medical Diary.

Questions 1-10: Please answer T (true) or F (false)

1. **CO Poisoning should be treated with Hyperbaric Oxygen Therapy only.**
2. **The half-life of COHb is 70 minutes when breathing on 100% oxygen at 1 ATA.**
3. **Carbon Monoxide causes tissue hypoxia.**
4. **Hyperbaric Oxygen Therapy can reduce brain lipid peroxidation.**
5. **The evidence of CO Poisoning treating with Hyperbaric Oxygen Therapy to reduce delayed neurological sequelae remains controversial.**
6. **CO Poisoning is treated with hyperbaric oxygen therapy in all hyperbaric centres in the world.**
7. **COHb level threshold for the pregnant woman to be treated with Hyperbaric Oxygen Therapy is higher.**
8. **Loss of consciousness in CO Poisoning patient is an indicator of Hyperbaric Oxygen Therapy.**
9. **Hyperbaric Oxygen Therapy may be useful in acute poisoning including methylene chloride and hydrogen sulphide.**
10. **Hyperbaric Oxygen Therapy is defined as treatment with air in atmospheric pressure above 1 ATA.**

ANSWER SHEET FOR JANUARY 2020

Please return the completed answer sheet to the Federation Secretariat on or before 31 January 2020 for documentation. 1 CME point will be awarded for answering the MCHK CME programme (for non-specialists) self-assessment questions.

Role of Hyperbaric Oxygen Therapy in Carbon Monoxide Poisoning

Dr Joe KS LEUNG
Director, HBOT Centre, PYNEH

1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐ 8 ☐ 9 ☐ 10 ☐

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Answers to December 2019 Issue

Evidence-based Medicine for Lipid-modifying Medications

1. F 2. T 3. F 4. F 5. T 6. T 7. T 8. T 9. F 10. F

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Abbreviations: GBM=glioblastoma multiforme; TMZ=temozolomide

References: 1. Data on file. Novocure. Last updated January 2019. **2.** Stupp, R. *et al.* JAMA 318, 2306–2316 (2017).

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Decompression Illness and Hyperbaric Oxygen Therapy

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INTRODUCTION

Decompression illness (DCI) is a term which describes the clinical manifestations of bubble formation in the blood or tissues after a reduction in ambient pressure.[1] It encompasses two conditions known as decompression sickness (DCS) and arterial gas embolism (AGE). AGE could be due to pulmonary barotrauma or iatrogenic causes, both of which introduce bubbles into the arterial circulation, leading to vascular obstruction, ischemia, and stimulation of inflammatory processes that follow damage to the endothelium. DCS is related to the evolution of bubbles from dissolved inert gas. These bubbles appear in the veins and vulnerable tissues and may cause harm through mechanical distortion of tissues, pulmonary vascular obstruction, or stimulation of inflammatory processes that lead to tissue oedema, haemoconcentration, and hypoxia. Venous bubbles may also enter the arterial circulation via right to left shunts such as a patent foramen ovale.[2] In this article, we present a patient with type 2 DCS treated with hyperbaric oxygen therapy (HBOT) at our centre.

A patient with TYPE 2 DCS

Our patient was a 44 year-old healthy Chinese male who went for recreational diving in Egypt (Red Sea) for six days in July 2019. He had 3 to 4 dives every day, of depth down to 30 metres and duration around 1 hour, with surface intervals of around 1 hour between dives. On the 5th day, he performed rapid ascent and descent; the reason given was that he was chasing after marine animals. He developed bilateral lower limb numbness at the end of that day. He continued diving on the 6th day despite his symptoms. Subsequently, he had worsened lower limb numbness and developed lower limb weakness, as well as bilateral knee and low back pain after ascent from his last dive on the 6th day. His divemaster escorted him to a local hyperbaric facility on the 7th day, and hyperbaric oxygen therapy using US Navy Treatment Table 6 (TT6) was given to him. He experienced partial symptom resolution. However, symptoms recurred the next day, and he was given HBOT using US Navy Treatment Table 5 (TT5) on the 8th day, after which he again had partial symptom improvement. He decided to leave Egypt and took indirect flights back to Hong Kong on the 10th day. During his flight, he experienced worsening of the pain when at altitude and got better once the plane landed.

The patient presented to our Accident & Emergency Department in the evening of the 13th day, shortly after landing. He complained of persistent numbness particularly over his right face and both lower limbs, weakness of his right limbs, numbness and pain in the back, as well as subjective difficulty in swallowing. Examination revealed right limb power of grade 4 out of 5, and diminished light touch sensation over his right face and all four limbs. A diagnosis of type 2 DCS was made. (Please refer to Discussion below for a classification of DCS.) TT6 was initiated the next morning. The patient was pressurised to 2.8 atmosphere absolute (ATA), with subsequent 100% oxygen breathing via a hood, with intervals of air-breaks, for a treatment duration of about 290 minutes. His right limb weakness and swallowing difficulty subsided totally towards the end of the treatment. His back pain and facial numbness also improved significantly. The patient was discharged from the hospital after an overnight observation in our emergency medicine ward.

Our patient later received two further TT5 treatments on 16th and 22nd days, as a result of the recurrence of symptoms including mild right elbow pain, backache, bilateral ankle and knee pain and scalp numbness. Most of the symptoms showed resolution after treatments, apart from some residual backache and occipital scalp numbness. No more limb weakness was reported, and limb power remained at 5/5. In view of the clinical plateau, further HBOT was deemed non-beneficial. At subsequent follow-ups up to day 52, our patient reported resolution of his backache and further reduction in the scalp numbness. He did not report any functional or cognitive impairment. Subsequent magnetic resonance imaging (MRI) of his brain showed no significant abnormality. An echocardiogram was arranged later.

DISCUSSION

The incidence of DCI varies from about 1 in 10,000 dives among trained recreational divers[3] to 1 in 245 dives in indigenous underwater harvesters.[4] In most cases of SCUBA diving, the breathing gas used is air. With increasing diving depth, the tissues of the diver's body are loaded with increased partial pressures of oxygen and nitrogen, as described by Henry's law. Oxygen is utilised by the body tissue and hence does not pose a problem. Yet, nitrogen is physiologically inert. Thus nitrogen content of a tissue increases in proportion to the ambient pressure. This nitrogen content is also related to the time under pressure, tissue perfusion, and the tissue fat content, as nitrogen has a relatively high solubility in fat.[5]

When the diver returns to the surface, the ambient pressure decreases and so do the partial pressures of the gases of the breathing gas (Dalton's law says the total pressure exerted by a mixture of gases is the sum of the pressures that would be exerted by each gas if it were to occupy the total volume)[6], and the additional nitrogen is eliminated from the tissues, down a diffusion gradient. When decompression is performed too fast (e.g. voluntary rapid uncontrolled ascent due to breathing gas depletion/leakage, equipment failure, psychological factors, etc.), the pressure decrease can exceed the pulmonary elimination rate of the gas. This results in a critical supersaturation of nitrogen in the tissue. As a result, liberation of free gas from the tissue can occur, forming bubbles that subsequently block arteries, veins, and lymphatic vessels.[7] In addition to mechanically obstructing blood flow through the vasculature, bubbles may directly contact and damage the vascular endothelium[8] and activate the inflammatory cascade[9], and hence the detrimental consequences, particularly in the cardio-pulmonary system.

TYPES OF DCS

Generally speaking, there are two types of DCS, though many times the clinical features overlap in one another. 42% of patients report symptoms within 1 hour of exposure to decompression stress. About 80% report symptoms within 8 hours, and 98% within 24 hours. Symptom onset beyond 36 hours should prompt the physician to an alternative diagnosis. The earlier the symptoms arise, the more severe DCS patient is likely suffering from.[10]

Type 1 DCS includes musculoskeletal and skin manifestations, often referred to as "bends", and "skin bends". Musculoskeletal manifestations are the commonest symptom, reported by 90% of type 1 DCS patients. The pain is often described as a dull, deep, throbbing, toothache-type pain, usually in a large joint or tendon area. The shoulder is the most commonly affected joint in divers after a shallower than 40-metre dive, whereas the knees are affected more in deep divers. The pain is initially mild and slowly becomes more intense. Because of this, many divers attribute early DCS symptoms to overexertion or a pulled muscle. Upper limbs are affected three times more than lower limbs.[11] "Skin bends" are often described as an itchy or burning sensation, with a rash that varies from deep red or purple to mottling (Fig. 1). One important skin feature is cutis marmorata, described as a marbling discolouration of the skin (Fig. 2), which signifies systemic DCS pathology.[12] Marbling indicates nitrogen bubbles present within tissues and blood vessels,[13] and is due to vascular congestion and an inflammatory response towards the bubbles.[14] The presence of cutis marmorata warrants a high index of suspicion of type 2 DCS. Lymphatic oedema has also been reported. It is uncommon and usually is signalled by painless pitting oedema. The rash usually starts on the chest and will tend to progress down the trunk over the next few days, finishing in the lower legs. Cutaneous and lymphatic symptoms, if found alone, are considered mild and do not usually require treatment.

Type 2 DCS generally involves the neurological, inner ear, and cardio-pulmonary systems. Symptom onset is usually early to immediate, but may still be delayed up to 36 hours. Neurological manifestations could involve the central nervous system (CNS) and/or peripheral nervous system (PNS). CNS features may include headache, dizziness, nystagmus, visual changes (e.g. tunnel vision), cranial nerve palsies, gait disturbances, cognitive and personality changes etc. PNS features may mimic spinal cord trauma. Symptoms such as lower back pain may start within a few minutes to several hours after the dive and may progress to paresis, paralysis, paraesthesiae and loss of sphincter control.[11] Inner-ear symptoms, often referred to as "staggers", may include tinnitus, hearing loss, vertigo, nausea, vomiting, and impaired balance. Cardiopulmonary manifestations, often referred to as the "chokes", comprise a triad of dyspnoea, dry persistent cough, and pleuritic burning substernal chest pain and may progress quickly to pulmonary oedema, respiratory failure, right ventricular dysfunction, and cardiovascular collapse[15], through the pathophysiological cascade described earlier in this article.

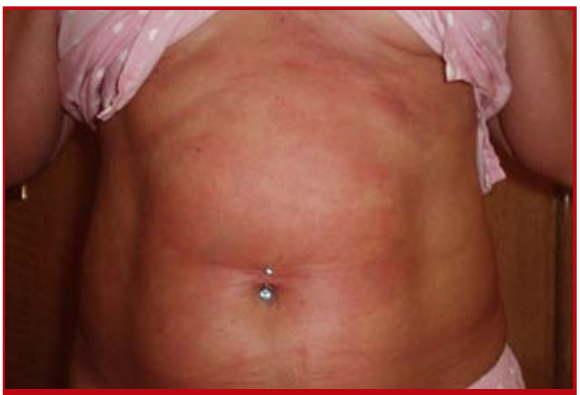

Fig. 1 Skin bends
(Source: Internet info from Midlands Diving Chamber [http://midlandsdivingchamber.co.uk/index.php?id=dci&page=3] retrievial dated 5/12/2019)

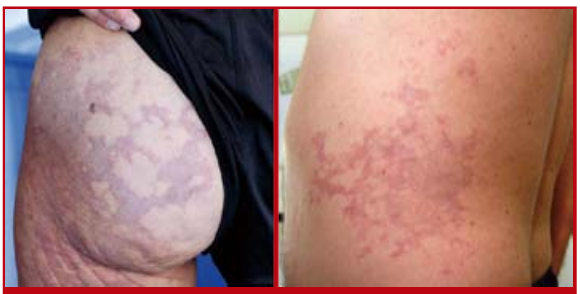

Fig. 2 Cutis Marmorata
(Source: Internet info from Decompression sickness, Diver Alert Network [https://www.diversalertnetwork.org/health/decompression/Signs-and-symptoms-of-DCS] retrevial dated 5/12/2019)

TREATMENT OF DCS

Recompression therapy with HBOT remains the standard treatment for DCS. The primary objectives are 1) to recompress gas bubbles to a small volume and hence relieve local pressure and restore blood circulation, 2) allow sufficient time for bubble

resorption, and 3) hyperoxygenation and hence relieve tissue ischaemia. The type of treatment table used depends on the type of DCS concerned. For Type 1 DCS indicated for HBOT, the US Navy Treatment Table 5 (USN TT5) is used. Under this table, the chamber is pressurised to 2.8 atmosphere absolute (ATA), about the pressure found at 60 feet of seawater (FSW). The patient then breathes pure oxygen, interspersed with scheduled periods of breathing air (air breaks) to reduce the risk of oxygen toxicity. The usual duration of the USN TT5 treatment is $2^{1/4}$ hours. If complete symptom relief is not achieved by 10 minutes at 2.8 ATA, or for Type 2 DCS, the US Navy Treatment Table 6 (USN TT6) is used, which is essentially a longer version of the USN TT5, with extensions possible depending on patient's response. The hyperbaric physician may even resolve to deeper tables if symptoms worsen during treatment (depending on capability of the hyperbaric chamber and staff involved). The number of treatments to be given will vary according to patient's response, and there is no strict consensus. In general, for patients with residual symptoms after the first treatment, up to two further sessions may be provided before deeming the condition as a clinical plateau. The US Navy Diving Manual gives a comprehensive treatment flowchart for DCS and AGE.[10]

Adjunctive therapy may provide additional benefit to DCS patients. Modalities include the judicious fluid (oral or intravenous) replacement, oral medications (e.g. Aspirin, some NSAIDs), low-molecular-weight heparin, etc. However, further studies regarding their efficacies and safety profiles are still on-going.[16]

HBOT is not the endpoint to the treatment of DCS. The patient will need appropriate follow-ups which include not only symptom evaluation, but also essential aspects such as fitness to return to dive, assessment for patent foramen ovale in recurrent DCS cases, and psychosocial assessment if indicated.

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Dermatology Quiz

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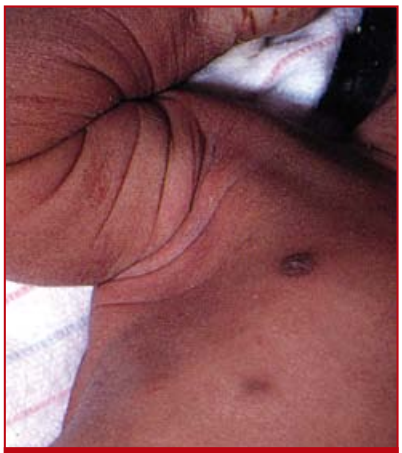

Fig.1: A solitary nodular lesion at the right side of anterior chest

This newborn baby was noticed to have a nodular growth on his chest during a routine examination (Fig.1). His mother had an amniocentesis done during pregnancy. Apart from this, there were no other noticeable physical abnormalities. Her mother was told that it was probably due to an insect bite. However the lesion persisted in time.

Questions

1. What is your diagnosis and differential diagnoses?
2. What other abnormalities have been reported as possible associations?
3. How do you treat this condition?

(See P.33 for answers)

Necrotising Fasciitis: the Role of Hyperbaric Oxygen Therapy in Management

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INTRODUCTION

Necrotising soft tissue infections (NSTI) refer to a spectrum of disease entities in which necrosis of deeper tissue, including fascia and muscle, is found and is precipitated by microorganism infection. These infections are characterised by widespread fascial necrosis, fulminant tissue destruction and severe systemic sepsis and as a result, by high mortality[14]. Early and accurate diagnosis of disease and timely and appropriate treatment are the most crucial factors in increasing the limb salvaging rate and reducing the mortality rate of the patients.

ETIOLOGY AND EPIDEMIOLOGY

Necrotising fasciitis (NF) is a severe form of soft tissue infection that primarily involves the superficial fascia and overlying subcutaneous fat. It tends to progress rapidly through the fascia plane due to its relatively poor blood supply, and in contrast, muscle tissue is frequently spared due to its good blood supply[14].

NF can be divided into 2 types: polymicrobial (type l) and monomicrobial (type ll) [1, 2, 12, 14].

Type I Infection:
This is the most common type of infection, caused by a mixture of bacterial types, typically various species of Gram-positive cocci, *(Staphylococcus aureus, Streptococcus pyogenes, and Enterococci), Gram-negative rods, (Escherichia coli, Pseudomonas aeruginosa),* and at least one anaerobe *(Bacteroides, Clostridium or Peptostreptococcus species).* It usually occurs in older people or adults with underlying comorbidities, in which diabetes mellitus is the most important risk factor.

Fournier gangrene is a form of necrotising fasciitis that is localised to the scrotum and perineal area. It is caused by facultative organisms *(E coli, Klebsiella, enterococci)* along with anaerobes *(Bacteroides, Fusobacterium, Clostridium, anaerobic or microaerophilic streptococci)* [14].

Type II Infection:
This infection accounts for 20 to 30% of cases, mainly involving the extremities. This mainly involves Streptococcus pyogenes bacteria, alone or in combination with staphylococcal infections. Both types of bacteria can progress rapidly and manifest as toxic shock syndrome[12, 13, 14]. Streptococcus species produce M protein, which acts as a superantigen, stimulating a massive systemic immune response which is not effective against the bacterial antigen, precipitating shock. It may occur in any age group and in healthy individuals.

In coastal cities, including Hong Kong, another common bacteria that can lead to necrotising fasciitis was the Vibrio species. The pathogens are halophilic, and high concentration can be found in shellfish. The highest densities of Vibrio species in Hong Kong were found in water samples from the Aberdeen shelter and the Jordon ferry pier[7].

The prevalence of necrotising fasciitis globally has been reported to be 0.4 cases per 100,000 population with a male-to-female ratio of 3:1, mainly correlated with the increased incidence of Fournier's gangrene in men[10]. The mortality rate of NF is controversial, ranging from 8.7% to 76%. A systemic review in 2014 concluded that the median mortality ratio was 21.5%[15].

Apart from diabetes mellitus, the most important precipitating factor for necrotising soft tissue infection, there are other risk factors of NSTI including major penetrating trauma, minor laceration or blunt trauma, skin breach, recent surgery, mucosal breach, immunosuppression, malignancy, obesity and alcoholism[14].

CLINICAL MANIFESTATIONS OF NSTI

The clinical presentation of necrotising fasciitis depends on the stages of the disease. At early stage, only local tenderness, erythema, swelling and raised skin temperature were presented[11]. Severe pain that is out of proportion to clinical signs may also be observed. The typical signs including crepitus, skin bullae, necrosis and ecchymosis are rare[7].

The patients may also present with signs of sepsis including fever, tachycardia, hypotension and disorientation[10,11].

The disease often progresses rapidly over days, with skin colour turns from red-purple to blue-grey patches. When there was progressive thrombosis, caused by the invading pathogens, of vessels that penetrate the fascia to supply the skin, blisters or bullae are formed due to critical skin ischaemia. It would also lead to diminished sensation to pain in the involved area as the superficial nerves in the subcutaneous tissue are also destroyed.

In the very late stage, the lesions turn black and form the necrotic crust, with fascial tissue, and brown, greyish

secretions underneath the crust and the tissues become very fragile and easily removable[7,11].

Table 1. Clinical staging of necrotising fasciitis (Reproduced from JYP Cheung, B Fung, WM Tang, WY Ip. A Review of necrotizing fasciitis in the extremities. Hong Kong Med J 2009; 15:44-52)

Stage 1 (early stage)	- Tenderness to palpation (beyond the apparent area of skin involvement - Swelling - Erythema - Raised skin temperature
Stage 2 (intermediate stage)	- Skin fluctuance/induration - Blisters/bullae formation
Stage 3 (late-stage)	- Skin anaesthesia - Crepitus - Haemorrhagic bullae - Skin necrosis

DIAGNOSIS

Early recognition of the condition of necrotising fasciitis is crucial for saving limbs or even life, due to the natural rapid progression of the disease.

Laboratory findings are non-specific. Patients would present with leukocytosis, metabolic acidosis, coagulopathy and raised inflammatory markers including c-reactive protein and erythrocyte sedimentation rate. Elevation of serum creatine kinase (CK) and aspartate aminotransferaase (AST) concentrations signify deep tissue infection involving fascia or muscle[14].

Imaging investigation may also assist in the diagnosis of necrotising fasciitis. Though not very sensitive and specific, sometimes plain radiography is able to show gas formation in the soft tissue, which is strongly related to *Clostridium* species infection[10]. Computed tomography (CT) or magnetic resonance imaging (MRI) is more sensitive and specific. They can show the extent of tissue infection, fascial swelling, inflammation and gas formation. However, it should not delay surgical intervention when there is rapid clinical progression of disease or when there is already crepitus on clinical examination, which signify late stage of the disease[14].

Blood culture offers a yield of 20% in patient with type I necrotising fasciitis. The yield is higher in type II necrotising fasciitis, reaching 60%, and the blood culture is routinely positive in cases of necrotising myositis[14].

The gold standard for diagnosing necrotising fasciitis remains surgical exploration and debridement with tissue biopsy[7]. The tissue integrity and depth of invasion can be evaluated during exploration. Presence of fascial and muscle necrosis and loss of fascial integrity along tissue planes are diagnostic. Swollen and dull-grey appearance of the fascia, thin exudate without clear purulence, easy separation of tissue planes are the common surgical findings[7].

Characteristics pathological findings of the tissue biopsy of NF include extensive tissue destruction, thrombosis of blood vessels, abundant bacteria spreading along fascial planes and infiltration of acute inflammatory cells[7,10,14].

MANAGEMENT OPTIONS

Prompt diagnosis, adequate supportive treatment, timely and frequent surgical exploration and debridement, together with broad-spectrum empirical antibiotics treatment, are the key elements for treating necrotising fasciitis.

Surgical Debridement

The goal of surgical debridement is to debride all necrotic tissue until healthy and viable tissue is reached, and usually repeated debridement is required in order to achieve this goal. For severe cases involving extremities, amputation is not uncommon in order to control the infection[7,12,14].

Antibiotics Treatment

Appropriate antibiotics are as important as surgical debridement. The initial regimen should be able to cover aerobic Gram-positive cocci, Gram-negative rods and anaerobes. A combination of penicillin and clindamycin is usually used as an empiric antibiotics regimen. The regimen can then be tailored to Gram-stain, culture and sensitivity results when available[7,12,14].

HYPERBARIC OXYGEN THERAPY

Definition

The Undersea and Hyperbaric Medical Society (UHMS) defines hyperbaric oxygen (HBO2) as an intervention in which an individual breathes near 100% oxygen intermittently while inside a hyperbaric chamber that is pressurised to greater than sea level pressure (1 atmosphere absolute, or ATA)[2].

Rationale for HBOT in Treating NF

By putting the patient under HBOT as an adjunct for the treatment of necrotising fasciitis, it can reduce the amount of hypoxic leukocyte dysfunction occurring within an area of hypoxia and infection. HBOT can also provide oxygenation to otherwise ischemic areas, thus limiting the spread and progression of infection. The diffusion of oxygen dissolved in plasma in the circulation, where it is initially carried in large vessels, proceeds to areas of poorly perfused tissue, from regions of very high oxygen saturation down a gradient to lower oxygen levels in tissue. The effect of HBOT on integrin inhibition also decreases leukocyte adherence thus reducing systemic toxicity. Moreover, HBOT can enhance antibiotic penetration into target bacteria[2].

Evidence

A retrospective cohort study in Australia involving a major tertiary hospital has assessed all patients admitted with a diagnosis of necrotising soft tissue infection across 5 years from 1994 to 1999. It shows that there was increased survival with odds ratio of 8.9 and number of 3 need to treat to benefit and reduced

incidence of limb amputation for the patient group with use of HBOT as an adjunctive treatment[5].

In an Italian study, 26 patients with Fournier's gangrene from a single centre were assessed. The Fournier's gangrene patients with HBOT as adjunctive treatment demonstrated a lower mortality rate of 7% vs 42% in patients without HBOT[4].

In another single-centre, retrospective, case-controlled study of 342 patients with the diagnosis of necrotising fasciitis attending a hospital in Australia over 13 years from 2002 to 2014, the overall mortality was 14.4%; 12% in those treated with HBOT, and 24.3% in those not treated with HBOT. Mortality was linked to illness severity at presentation; however when adjusted for severity score and need for intensive care management, HBOT was associated with significant reduction in mortality[3].

Nevertheless, well-controlled, randomised, clinical trials demonstrating a statistically significant benefit of HBOT are lacking, and therefore its use as adjunctive therapy for necrotising fasciitis remains controversial.

Apart from the acute infection, once the infection is under control, HBOT can also induce fibroblast proliferation and angiogenesis, which should be helpful in wound healing and closure[8,9].

In a US study, patients with HBOT achieved wound closure 28 days after the first debridement compared to 48 days for those without HBOT[6].

Regimen of HBOT in Treating NF

The recommended HBOT would be 100% oxygen at 2-2.5 absolute atmospheres for 90 minutes 2 times a day for first few days until there appears to be no further extension of necrosis in previously debrided areas and infection is controlled[2].

Use of HBOT in Hong Kong

Previous use of HBOT in Hong Kong has been limited. The Recompression treatment centre on Stonecutters Island has been operated by the Hong Kong Fire Services Department under the medical supervision of the Occupational Medicine Division of the Department of Health since April 1994.

It was mostly used for conditions related to diving including decompression illness and cerebral acute gas embolism. Medical condition like carbon monoxide poisoning has also been treated in the centre. However, due to its remote location and limited space of the small cylindrical hyperbaric chamber, the utility for other medical condition, especially for those with critical clinical condition, is very limited as a result of high risk of transportation and limited space for handling emergency situation inside chamber.

The first hospital-based, multi-lock, multi-place rectangular hyperbaric chamber under the Hospital Authority started operation in September 2018. It is situated in Pamela Youde Nethersole Eastern Hospital (PYNEH) under the Department of Accident and Emergency (Fig. 1).

It operates 24 hours a day, 365 days a year for emergency cases and is able to handle critically ill patients with the space in-chamber and support from the intensive care unit.

With reducing risk of transportation, convenience in referring to cases for treatment and support from intensive care unit, using of hyperbaric oxygen therapy should be considered an adjunctive treatment for necrotising fasciitis patients.

Fig. 1. Hyperbaric oxygen therapy centre in PYNEH (Photo from personal collection)

Recent Patient with Necrotising Fasciitis given HBOT in PYNEH

An 82 year-old gentleman, with background history of hypertension and hyperlipidaemia, presented to PYNEH A&E for left lower limb pain and redness for two days. On examination, the patient presented with septic shock with blood pressure 80/46mmHg. There were tenderness, redness and swelling of the medial side of the left lower limb. Fluid resuscitation was given, and he was started on intravenous ceftriaxone and linezolid after blood culture was taken. He was then admitted to Orthopedics ward and later transferred to the Intensive Care Unit (ICU) for his critical conditions.

Initial blood tests showed that there were acute renal failure and mild metabolic acidosis.

Multiple emergency operations for left lower limb debridement were performed, with findings of dishwater fluid at the medial ankle and fascial plane easily dissected with finger (Fig. 2 and 3). Urgent gram stain showed gram-positive cocci and later wound swab, and tissue culture results came back to be Streptococcus pyogenes.

In view of his critical condition, HBOT centre of PYNEH was consulted for adjunctive treatment for the patient with NF. Emergency HBOT was arranged and a total of 5 sessions of HBOT, each with pressure up to 2.8 ATA for 125 minutes were performed over consecutive days during the critical phase. The patient's clinical condition gradually improved. He was then extubated

and successfully weaned off his ionotropic support. He was successfully discharged to Orthopaedics ward after 6 days of ICU stay.

After his discharge to general ward and stabilisation of his clinical condition, daily session of HBOT of pressure up to 2.4 ATA for around 120 minutes each, for a total of 25 days were performed for the patient again, for the promotion of wound closure.

Together with adequate wound debridement, appropriate antibiotics treatment and good wound care in the Orthopaedics ward, the patient was able to be discharged home with good wound closure (Fig. 4 & 5) after 2-month stay in hospital. Both the life and limb of the patient was saved.

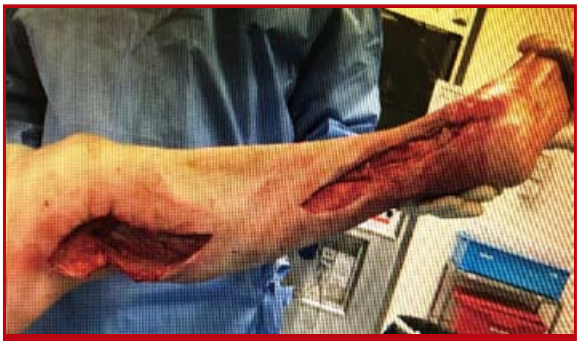

Fig. 2 Clinical photo of patient's left leg following surgery in operation theatre (with patient's consent)

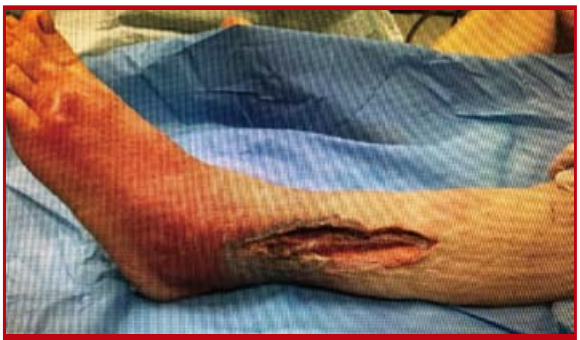

Fig. 3 Clinical photo of patient's left leg following surgery in operation theatre (with patient's consent)

Fig. 4 Clinical photo of patient's left leg wound after hyperbaric treatment (with patient's consent)

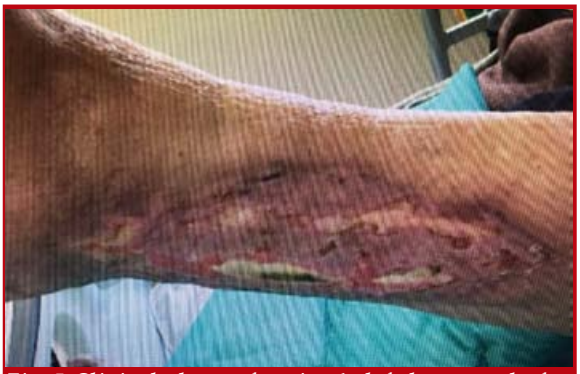

Fig. 5 Clinical photo of patient's left leg wound after hyperbaric treatment (with patient's consent)

CONCLUSION

Treatment of necrotising fasciitis remains a challenge to all healthcare providers, due to the difficulty in diagnosis and the high mortality rate. Timely diagnosis with prompt surgical exploration and debridement and appropriate antibiotics treatment remain the mainstay for achieving a successful outcome.

With the availability and good support of hyperbaric oxygen therapy now in Hong Kong, it should be strongly considered an adjunctive treatment for necrotising fasciitis for the potential of reducing mortality rate and limb amputation rate as well as promoting wound closure even when the infection is under control.

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Hyperbaric Oxygen Therapy for Central Retinal Artery Occlusion

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BACKGROUND

Central retinal artery occlusion (CRAO) is an ophthalmological emergency which can result in severe, sudden, painless visual loss. Internationally, the incidence has been quoted to be 1 in 100,000 people.[1] Occlusion in the central retinal artery (CRA) results in central retinal artery occlusion (CRAO), whereas occlusion in its branches leads to branch retinal artery occlusion (BRAO).

Classical non-arteritic CRAO is the most common type of CRAO.[2] In such cases, an occlusion of the central retinal artery (CRA) is usually caused by an impacted embolus at the narrowest part of the CRA where it enters the sheath of the optic nerve.[2] The natural history of non-arteritic CRAO is difficult to study, but a spontaneous resolution rate of less than 1 to 8% for acute non-arteritic CRAO has been cited.[3, 4]

Various modalities have been tried for treating CRAO, including ocular massage, anterior chamber paracentesis, intraocular pressure-lowering medications, vasodilators and oral diuretics, all of which have been shown to be unsuccessful in the great majority of cases. Intraarterial thrombolytic agents have been investigated but were associated with increased haemorrhagic risk with variable results.[5] The time of symptom onset is also critical for intraarterial thrombolysis to be safe and effective.[6] Finally, it is likely that the significant proportion of CRAOs from calcific or cholesterol emboli cannot respond well to thrombolysis.[7]

RELEVANT EYE ANATOMY

The internal carotid artery gives rise to the ophthalmic artery, which in turn gives rise to the central retinal artery supplying the retina, as well as the posterior ciliary arteries (2 long posterior ciliary arteries and approximately 20 short posterior ciliary arteries) supplying the choroid, iris and ciliary body. The central retinal artery enters the globe within the substance of the optic nerve and directly supplies the inner layers of the retina (the ganglion cell layer and inner nuclear layer). Anterior to the inner nuclear layer is the outer plexiform layer, which is also a watershed region. Anterior to this layer lies the photoreceptors and the retinal pigment epithelium to which oxygen is supplied by the choroidal circulation by diffusion.[8]

The central retinal artery branches into four branch retinal arteries, one for each quadrant, running in the nerve fibre layer beneath the ganglion cell layer. Under normal circumstances, 60% of the retina is supplied by the choroidal circulation.[8]

Internationally, in approximately 15-30% of individuals, a cilioretinal artery is present, originating from the short posterior ciliary artery.[8] Although it is part of the ciliary arterial supply, this artery supplies the area of the retina around the macula. The presence of a temporal cilioretinal artery supplying the fovea of clinical significance as it may spare the fovea in case of CRAO, leading to a better visual outcome due to the dual blood supply to the inner retina. One study in north China has quoted the incidence of cilioretinal arteries in Chinese Han population to be 35.0% (one, two or more), with equal distribution between men and women.[9]

ETIOLOGY OF CRAO

The causes of CRAO include thrombosis, embolus, arteritis, vasospasm and foreign material (such as fillers from aesthetic procedures).[10] In CRAO, the inner retinal layers normally served by the retinal circulation will typically lose viability, resulting in painless visual loss.

It is known that risk factors predisposing to other cardiovascular and cerebrovascular events are often present in CRAO. One prospective RCT found that vascular risk factors were present in 78% of CRAO patients.[11] These include hypertension, diabetes, hyperlipidaemia, carotid artery stenosis, cerebral ischaemic stroke and various heart conditions such as ischaemic heart disease, valvular heart disease and atrial fibrillation (which may predispose to distal embolism).

SIGNS AND SYMPTOMS OF CRAO

Patients typically present with painless visual loss to the point of only light perception (LP) or counting fingers (CF). Amaurosis fugax may precede loss of vision in up to 10% of patients.[2]

For those with visual acuity (VA) of no light perception, occlusion may have happened at the ophthalmic artery resulting in no flow to both the central retinal artery as well as the choroidal vessels.[8] In such cases, hyperbaric oxygen therapy (HBOT) may not be helpful as there will be no oxygen supply from the choroidal circulation to the retina.

Fundoscopy typically reveals a pale yellow to white retina due to ischaemia or necrosis. A cherry-red spot may be found in the macula, but may not always be present, and is usually a late sign with poorer

prognosis.[12] Other findings in CRAO may include: relative afferent pupillary defect (RAPD), segmentation of retinal blood columns (boxcarring or "cattle-trucking"), stagnant flow or absent flow of segmented blood columns, diffusely narrowed retinal arteries (also seen in ocular ischaemic syndrome), spontaneous pulsation of the CRA (systolic filling and diastolic collapse of CRA), embolus in the CRA and emboli in the retinal arterial branches (taken to be diagnostic of embolic CRAO when associated with other signs of CRAO).[7]

Features not suggestive of CRAO include the presence of flashes or floaters preceding visual loss, eye pain, history of recent eye trauma and patient age below 40. In such cases, an alternative diagnosis for sudden visual loss should be sought.[8]

DIAGNOSIS OF CRAO

The diagnosis of CRAO is typically made with fundoscopy. Confirmatory fluorescein angiography can be done if the diagnosis is not confirmed. In the acute phase, fluorescein angiography shows slowed or absent filling of the central retinal artery with a normal filling of the choroid. If the choroid is also affected, giant cell arteritis (GCA) should be considered, especially in an older adult patient.[8] It should be stressed that confirmation of diagnosis should not delay the initial treatment of CRAO.

Other tests may be done to look for possible modifiable risk factors, such as diabetes and carotid artery stenosis. In particular, erythrocyte sedimentation rate (ESR) and serum C-reactive protein (CRP) should be done for CRAO patients aged above 50 when no visible retinal emboli are seen, to exclude the diagnosis of giant cell arteritis.[13] Magnetic resonance imaging (MRI) of CRAO patients has found 32% had had acute or subacute incidental brain infarcts.[8, 26]

There have also been case series reports on the use of optical coherence tomography (OCT) in CRAO. OCT shows initial thickening of ischaemic areas of the inner retina soon after central or branch retinal artery occlusion, with subsequent thinning that is apparent in a few weeks. The outer layers of the retina are spared.[13, 27]

HYPERBARIC OXYGEN THERAPY FOR CRAO

Hyperbaric oxygen therapy is a medical treatment method that aims at increasing the level of dissolved oxygen in the tissues. Patients inhale 100% oxygen at pressures exceeding 1 atmosphere absolute (1 ATA) in a closed hyperbaric chamber. The goal of using HBOT for CRAO is to decrease hypoxia and prevent irreparable anoxic damage to the retina while awaiting the retinal artery to undergo spontaneous or assisted recanalisation.[14, 15] Spontaneous retinal recanalisation has been reported to occur ranging from 4.5 hours to up to 72 hours.[16,17]

TREATMENT PROTOCOL

A detailed management protocol was proposed by the Undersea and Hyperbaric Medical Society (UHMS).[8] UHMS recommends that HBOT for CRAO should be done within 24 hours. The strongest evidence for symptomatic improvement was reported from cases with fewer than 12 hours of delay[18], but literature in ophthalmology had published cases in which humans with CRAO had regained significant vision even when treatment was delayed for up to two weeks.[19]

In the most recent protocol by UHMS, it was suggested that patients should receive surface O2 at the highest possible fracture of inspired oxygen (FiO2) upon diagnosis.[8] If vision returns to normal, the patient should be admitted to hospital and given intermittent normobaric oxygen for 15 minutes followed by 45 minutes of breathing room air. This cycle is continued up to a maximum of 96 hours. If vision does not respond to normobaric oxygen in the first 15 minutes, patient should be sent for HBOT. HBOT from 2 ATA up to 2.8 ATA will be given using various treatment tables depending on the clinical response.

In 2018, Butler proposed a protocol further detailing the monitoring and management of patients who have received HBOT.[20] It was suggested that if vision worsens on room air 15 minutes after HBOT therapy, supplemental oxygen or even repeated HBOT should be immediately given. Supplementary oxygen or repeated HBOT is continued for up to a maximum of 96 hours until improved vision is sustained or central retinal artery recanalisation by fluorescein angiography is demonstrated.[20] The author, however, admits that this is an intensive regimen to undertake both for staff and patient. Whether the regimen will improve outcome remains unknown.

EVIDENCE FOR HBOT IN CRAO

HBOT has been investigated for the treatment of CRAO as early as the early 1960s.[8,21,22] In 2001, Beiran published a retrospective controlled trial of 35 retinal artery occlusion patients treated with HBOT compared with 37 matched controls. Patients in the HBOT group received 2.8 ATA for 90 minutes BD for three days then once daily. 82% of the HBOT patients improved compared to 29.7% in the control group. Improvement was defined as reading at least three lines better on the Snellen chart.[23]

In 2012, a case series by Menzel-Severing compared HBOT and haemodilution (51 patients) with haemodilution only (22 patients). An aggressive protocol was used, aiming at five treatments within 48 hours, with a pressure of 2.4 ATA for 90 minutes. In the HBOT group, mean improvement in the initial VA of 3 lines compare with baseline ($p<0.0001$) was shown. In the control group, mean improvement was 1 line ($p=0.23$). A recovery of 58.8% (30 patients) was demonstrated. At three months follow up, VA remained significantly different from the baseline in the HBOT group, but not in the control group. VA improvement of at least three lines was still present in 10 patients (35.7%) in the HBOT group.[18]

In 2017, Elder and Rawstron conducted another study using 2.0-2.8 ATA with 31 patients. 21 Patients had a temporary return of vision during or immediately following their first HBOT. Nine patients (29.0%) had permanent visual recovery, of which seven patients had good visual recovery (VA: 6/18 or better by Snellen Chart), and 2 with modest recovery (VA: 6/60). All nine patients who had permanent recovery received their HBOT treatment within 10 hours of symptom onset, suggesting that the sooner patients are treated with HBOT the better.[24]

In 2017, Hadanny et al conducted a retrospective analysis including 128 patients of non-arteritic CRAO with no cilioretinal artery treated with HBOT (2.0-2.4 ATA). For patients with no cherry-red spot on fundoscopy (n=43), the improvement in BCVA was prominent, with 86% gaining clinical improvement (logMAR change ≥0.3) and over 60% gaining BCVA ≤1 logMAR. For patients with cherry-red spot (n=85), 57.6% had significant clinical improvement (logMAR change ≥0.3), yet only 7.1% regained BCVA to ≤1 logMAR.(Fig. 1) Overall, 67.2% (86 out of 128) of non-arteritic CRAO patients had clinical significant improvement after HBOT. The time delay from symptoms to treatment was correlated with better outcome. For each hour delay, the VA by logMAR was decreased by 0.03.[12]

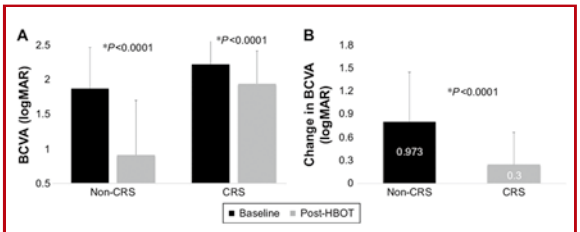

Fig. 1: BCVA comparing baseline and post-HBOT in non-arteritic CRAO patients. Abbreviations: BCVA = Best corrected visual acuity, CRS = Cherry red spot. Source: Handanny[12] Reproduced from ResearchGate. Link: https://www.researchgate.net/figure/hBOT-effect-on-BCVa-in-patients-with-and-without-Crs-Notes-A-Shows-significant_fig2_311977840

Currently, HBOT for treating CRAO is considered level IIb evidence using the American Heart Association Classification.[8] Fair to good evidence exist to support its use with retrospective controlled case series, but no prospective randomised controlled trials are yet available. The difficulty in creating a well-designed control arm for HBOT makes conducting randomised controlled trials for HBOT quite difficult in general.

PROGNOSIS

Hayreh and Zimmerman in 2005 described that the most important prognostic factor for CRAO is the type of disease, including the type (non-arteritic vs arteritic) and progression (transient or permanent) of occlusion. However, no reliable methods exist to determine which individuals with CRAO will gain or lose vision over time.[10] Overall, 66% of cases have shown improvement when treated with HBOT.[8] As previously mentioned, patients with cherry-red spot on fundoscopy have poorer prognosis compared with those without.[12]

Another study investigated other prognostic factors affecting the final best-corrected VA (BCVA) in CRAO.[25] The correlation between disorganisation of retinal inner layers (DRIL) score, using spectral-domain optic coherence tomography (SD-OCT) at five points, with the final BCVA in logMAR for CRAO patients was measured. DRIL score was negatively correlated with the final BCVA, negatively correlated with the change in BCVA, positively correlated with referral time to clinic, and negatively correlated with HBOT sessions count. However, no control for HBOT treatment was available in that study to independently investigate the role of HBOT for CRAO.[25]

Overall, further studies are needed to further determine and confirm the prognostic factors for CRAO.

USE OF HBOT IN CRAO IN HONG KONG

Since Nov 2018, Pamela Youde Netheresole Eastern Hospital (PYNEH) has started providing hyperbaric oxygen treatment for patients diagnosed with CRAO in Hong Kong. From Nov 2018 to Mid Oct 2019, PYNEH has treated thirty patients with suspected CRAO. Five out of thirty patients were finally confirmed not to have CRAO upon subsequent investigations, some of which were finally diagnosed with branch retinal artery occlusion instead. The majority of patients in Hong Kong initially presented directly to public hospital via the Accident and Emergency Departments (A&E). Patients also presented to their private ophthalmologist (around 7% in our locality), who would confirm the diagnosis of CRAO and refer the patient to A&Es. If the time from onset of symptom to attendance at A&E or an ophthalmologist is less than 6 hours, PYNEH HBOT Centre will be consulted. HBOT will then be given at 2.4-2.8 ATA for 90 minutes, for up to 6 to 10 sessions depending on the clinical response.

CONCLUSION

CRAO remains to be a challenging disease to treat. HBOT has been shown in various studies to produce a clinical response in visual improvement, yet higher level of evidence by further studies are needed. At present, HBOT remains to be one of the most promising treatment options available for the treatment of CRAO.

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INTRODUCTION

Established in September 2018, the Hyperbaric Oxygen Therapy (HBOT) Centre (Fig. 1) is the first and the only hospital-based HBOT Centre in Hong Kong, offering hyperbaric oxygen treatment services to local emergency patients. The centre complies with international standards for hyperbaric facilities, hyperbaric safety and hyperbaric operations, and is equipped with the latest technology in hyperbaric medicine, such as a hyperbaric chamber capable of treating patients ranging from ambulatory stable ones to those who are highly unstable in an intensive care unit.

Fig. 1. Hyperbaric Oxygen Therapy Centre, PYNEH (Photo from personal collection)

BACKGROUND

Plans for constructing the HBOT Centre began as early as 2013. In view of the prevalence of CO-poisoned patients in Hong Kong, toxicology experts noticed the effectiveness of hyperbaric oxygen in reducing the risk of delayed neurological sequalae[1] in such patients and started to explore the possibility of building a hyperbaric chamber in Hong Kong public hospitals. HBOT has been used clinically in many countries for more than 30 years. It has been implemented in hospitals worldwide with clinical evidence of treating different types of diseases.

In Hong Kong, there is in fact a government-owned, hospital-detached recompression treatment centre (RTC) with a hyperbaric chamber operated by the Fire Services Department since 1994 on remote Stonecutters Island. The centre is used for medical purposes by the Occupational Safety and Health Centre under Labour department, and the staff is trained as attendants and operators under the Fire Services Department.[2] Offering no critical care support to intensive care patients, the centre requires lengthy transportation from its nearest acute hospital, thereby increasing the risk of patient deterioration, equipment faults and discontinuity of care. Since it is an old-style cylindrical hyperbaric chamber, the risk of patient complications increases. The lack of a sophisticated patient monitoring system increases the difficulty in clinical care and this prohibits junior clinical doctors from helping out in this regard.

Owing to these potential hazards in patient care in the RTC, having a hospital-based hyperbaric centre in Hong Kong is essential to providing safe and effective hyperbaric oxygen therapy for patients in need. Additionally, in a cosmopolitan metropolis like Hong Kong, medical care in terms of hyperbaric oxygen therapy has been lagging behind for more than 20 years as compared with developed countries such as the United States, Australia, United Kingdom, Singapore, Japan and even Taiwan. As a Hong Kong physician, we aim for long-term local development of this technology and hope more patients will be able to benefit from this treatment.

LOCATION OF HBOT CENTRE

The HBOT Centre is situated in the Pamela Youde Nethersole Eastern Hospital (PYNEH) in Chai Wan, the eastern part of Hong Kong Island. It is located on the ground floor of the PYNEH next to the Accident and Emergency Department and the X-Ray and CT suite. Intensive care patients can reach the centre's entrance via elevator service. The PYNEH is also one of the two hospitals in Hong Kong equipped with a helicopter pad, which enables unstable patients to be directly sent to the hospital for treatment.

CAPABILITIES OF HBOT CENTRE

The PYNEH's HBOT Centre is equipped with a triple-lock rectangular hyperbaric chamber (Fig. 2) capable of treating both ambulatory and intensive care patients. A rectangular chamber provides a comfortable environment for ambulatory patients to receive treatment; a comfortable environment is important as claustrophobia is one of the significant side effects and contraindications in HBOT. We are also equipped with head-tents as breathing apparatuses for patients instead of breathing masks. Head-tents provide patients with a comfortable breathing environment in view of the long treatment duration of two hours or longer. These

tents also reduce the chance of breathing mask-induced compression injury to the face.

Fig. 2 Rectangular Hyperbaric chamber (Photo from personal collection)

As hypoglycemia is a frequent complication in diabetic patients undergoing hyperbaric treatment[3], we provide hyperbaric-compatible hemoglucostix to be used inside the chamber, in order to offer immediate treatment to patients suffering from hypoglycemia.

Moreover, to cater to the needs of intensive care patients, the HBOT Centre is equipped with two hyperbaric-compatible mechanical ventilators which are of the same brand as those in Intensive Care Units (ICU), allowing continuity of ventilator support in the hyperbaric chamber and reducing the risk of faulty ventilator setting due to brand and model differences. As the physiological monitor is also essential to ICU patients, we have a hyperbaric-compatible physiological monitor showing all the necessary parameters for use as in the ICU. Seeing that unstable ICU patients usually require multiple medications for life support, we have several safe hyperbaric-compatible syringe pumps. Through all the ICU-specific equipment, patients can receive the same level of ICU treatments inside the chamber.

Patients with chronic unhealed wounds, one of the most commonly treated indications, can be assessed with a transcutaneous oximeter (TCOM), a unique piece of equipment used to measure the partial pressure of oxygen under the skin, in the HBOT Centre. Doctors familiar with this parameter can decide whether it is beneficial to treat such patients with hyperbaric oxygen therapy; the TCOM can also serve to gauge the treatment progress and end-point. The parameter can also provide objective measurement results for the patient to understand the cause and prognosis of his disease.[4]

Equipped with all these sophisticated facilities, the HBOT Centre can maximally serve six to seven ambulatory patients and two intensive care patients per treatment lock. However, in addition to equipment, manpower is one of the most important hurdles in the centre's capability to provide treatment. To ensure the safety of patients, we must have at least one inside attendant accompanying the patients during their treatment. Nevertheless, there are multiple constraints on inside attendants under the Occupational Safety and Health Ordinance, and thus, limited manpower is usually the bottle-neck in providing patients with treatment.

What is Hyperbaric Oxygen Therapy

Hyperbaric Oxygen Therapy involves the treatment of a patient with 100% oxygen under pressure greater one ata. The patient is treated inside a hyperbaric chamber by breathing in 100% oxygen through a breathing apparatus. There are both mechanical and physiological effects exerted upon the body through HBOT.[5]

Mechanically, HBOT provides a pressurised environment which exerts direct effects on bubbles in the body according to various laws of Physics. Boyle's Law describes the inversely proportioned relationship between pressure and volume under a constant temperature. Dalton's Law states that in a mixture of non-reactive gases, the total pressure exerted is equivalent to the sum of the partial pressures of the individual gases. This is important as under high pressure, we could estimate the amount of oxygen breathed in according to the maximum pressure achieved. Henry's Law states that the amount of dissolved gas in a liquid is proportional to its partial pressure above the liquid. This is important for estimating how much nitrogen is dissolved in the body during treatment. Gay-Lussac's Law states that the pressure of a given mass of gas varies directly with the absolute temperature of the gas when the volume is kept constant. It explains why during hyperbaric pressurisation and depressurisation periods, the temperature will change accordingly.[6]

These laws of Physics are particularly important for HBOT in treating patients with decompression sickness because the pathophysiology of decompression sickness is based on the nitrogen gas bubbles obstructing different tissues in the body, causing different symptoms. The mechanical compression of the bubbles can directly relieve the mechanical obstruction and irritation from body tissues. This can relieve the symptom of decompression sickness very quickly, and we can see the clinical effects promptly.[7]

On top of the mechanical effects, HBOT will increase the partial pressure of oxygen (paO2) inside the body. The elevation of oxygen partial pressures to 1,000-1,500 mmHg impacts each of the two processes leading to neovascularisation: the growth of new blood vessels from local endothelial cells (angiogenesis), and the recruitment and differentiation in the bed of the wound of circulating stem or progenitor cells to form new vessels. HBOT can ameliorate the inhibited vascularisation in diabetes as well as the mobilisation of diminished stem and progenitor cells caused by radiation and chemotherapy.[8]

HBOT has been reported to reduce coronary artery stenosis after balloon angioplasty and increase myocardial tissue salvage. Tissue reperfusion is inhibited by the adherence of circulating neutrophils to vascular endothelium.[9] Exposure to HBOT causes an inhibition of such adherence that results in a wide range of biological advantages, including improved reperfusion after injury in the brain, heart, lung, liver,

muscles and intestines; reduced smoke-induced lung injury and encephalopathy due to CO poisoning; and reperfusion after gas embolism.[10]

HBOT also exhibits a sharp and direct bactericidal effect upon obligate anaerobic microorganisms. The lack of scavenger enzymes in anaerobic bacteria makes them sensitive to the high concentration in oxygen-free radicals. HBOT contributes to the increase in antimicrobial activity and cellular apoptosis.[11]

Regulating inflammatory molecules carries the possible advantage of reducing the extension of injury. Both animal and human researches indicated that HBOT lessens leukocyte sequestration, tumour necrosis factor and interleukin-6; HBOT also helps regulate fibroblast growth factor and collagen synthesis. HBOT has been shown to lead to increased nitric oxide production within the proximity of the wound and to promote increased granulation tissue deposition, epidermal migration and wound closure. HBOT impairs neutrophil adhesion and reduces the inflammatory burden on the chronic wound.[12]

Disease Indications for HBOT

The disease indications currently treated in the HBOT Centre are based on evidence from the Undersea and Hyperbaric Medical Society (UHMS) Hyperbaric Oxygen Therapy indications. UHMS was formed in 1967; it is an international non-profit association serving more than 50 countries in the fields of hyperbaric and dive medicine. It is an important source of scientific and medical information pertaining to the hyperbaric medicine involving hyperbaric oxygen therapy and diving through its bimonthly peer-reviewed journals, symposia, workshops, books and other publications. Clinical information, an extensive bibliographic database of thousands of scientific papers which represent the results of over 100 years of research by military and university laboratories around the world are contained in the UHMS Schilling library.[13]

Based on the UHMS indications, and following thorough discussion with the coordination committees on relevant specialities, our HBOT Centre is currently treating both emergency and elective indications. Emergency indications include decompression sickness (DCS), cerebral arterial gas embolism (CAGE), carbon monoxide poisoning (COP), central retinal artery occlusion (CRAO) and necrotising soft tissue infection (NSTI). Elective indications include unhealed chronic wounds, delayed radiation injury and idiopathic sensorineural hearing loss.[5]

Referral Mechanism for HBOT Services

The HBOT Centre provides around-the-clock services for all listed emergency indications. For referral to our centre, the care team needs to consult the on-call hyperbaric physician through the operator of PYNEH. On-call hyperbaric physicians will discuss with the referral source the patient's indications and condition. If the patient is referred, treatment will be provided. It is then necessary to transfer the patient to the corresponding department in PYNEH before HBOT can be commenced.

Other than emergency indications, elective referrals can be faxed to our centre, and they will be screened by an on-duty hyperbaric physician. The patient will be contacted by hyperbaric nurses and arrangements will be made for the first visit for patient assessment and education. During the first assessment, information such as indications, benefits, risks and complications will be discussed with the patient. The patient will given a guided tour of our centre for familiarisation of the environment so as to reduce the risk of anxiety and claustrophobia. If the patient understands and agrees to receive the treatment, a consent form will be signed, and a date for the first treatment will be scheduled.

Patient treatment statistics in HBOT Centre from October 2018 to October 2019

During this year, we mainly served emergency patients with decompression sickness, cerebral arterial gas embolism, carbon monoxide poisoning, necrotising soft tissue infection and central retinal artery occlusion respectively.

Decompression sickness (DCS) is diagnosed clinically. The patient typically suffers from muscle or joint pain or other neurological deficit within 24 hours after diving. Once it is suspected, our centre will be consulted, and the patient is transferred for hyperbaric treatment. DCS is typically treated by US Navy table 5 or 6; the condition will mostly improve or subside after one to three hyperbaric treatments.[14] There are a total of 28 patients consulting us for hyperbaric treatment, four refusing treatment after consultation, mostly for very mild symptoms. A total of 34 treatment sessions were provided and the symptoms of all these patients resolved following treatments, except for two patients suffering from Type II DCS. Nevertheless, these two patients have been gradually recovering after subsequent follow-up treatments.

Cerebral arterial gas embolism (CAGE) is mostly iatrogenic and patients usually suffer from severe symptoms such as cardiopulmonary distress or severe neurological deficit. One CAGE patient was treated last year initially with cardiovascular intervention, followed by intensive care support and four treatment sessions. His general and neurological conditions partially improved; following subsequent transfer back to the parent unit for rehabilitation, the patient can currently do self-care.

Carbon monoxide poisoning, one of the most prevalent modes of suicide in Hong Kong, can be treated by hyperbaric oxygen therapy to reduce the risk of delayed neurological sequelae resulting from the carbon monoxide particles' impact on the the biochemical cascade. Increased lipid peroxidation causes nerve damage and reperfusion injury resulting in delayed neurological damage. The Hong Kong Poisoning Information Centre (HKPIC) is responsible for screening the inclusion criteria for COP patients being considered for hyperbaric oxygen therapy. Under the present evidence-based medicine, one to three sessions of HBOT can be performed to reduce the risk of delayed

neurological sequelae.[1] In the past year, a total of 59 patients who had been advised by HKPIC to undergo hyperbaric oxygen were assessed in our centre, and a total of 127 treatment sessions were provided for these patients. Forty-eight out of the 127 sessions involved ICU support as the treated patients were on mechanical ventilation and sedated/unconscious. All of the patients were offered toxicology and psychiatry Specialist Outpatient follow-up. However, the default rate was very high for these patients.

Central retinal artery occlusion (CRAO) is an ophthalmological emergency, as this condition can lead to permanent blindness. To date, there is still no effective ophthalmological treatment for this disease, and most patients turn blind as time goes by. However, HBOT is proved to be effective especially when treatment of the patient can be commenced within 24 hours.[15] Upon consensus building, the Ophthalmology Coordinating Committee set 6 hours between symptom onset and first consultation with the Hospital Authority's Accident and Emergency Department or any ophthalmologist as the inclusion criterion. The rationale of HBOT used in CRAO is to buy time for first few days in order to make the emboli spontaneously dislodged. Thus, our HBOT treatment protocol for CRAO patients is three to five days' HBOT treatment following the ophthalmologist's assessment and advice. Last year, there were 31 patients assessed for CRAO management and a total of 200 treatment sessions were provided for these patients. Out of these patients, only one has had no improvement at all in visual acuity. All the others have experienced 20-70% improvement in subjective visual acuity.

Necrotising soft tissue infection (NSTI) is one of the most severe forms of infection, which can lead to mortality within hours of presentation. Thus, intensive care support, as well as surgical intervention to remove the source of infection, is the mainstay of management. HBOT is an adjunctive therapy which reduces reperfusion injury and enhances bactericidal effects, white cells' activities, and wound healing. The number of sessions provided for the patient depends on the severity of sepsis and the needs for inotropic support.[16] However, since NSTI patients are usually highly unstable and requiring frequent surgical interventions, transfer of patients for HBOT is a big challenge and becomes a major hurdle for patients to receive this therapy. We have offered consultations to five patients with NSTI in the previous year, and a total of 13 sessions of HBOT were provided. Elevan out of the 13 sessions needed ICU support. One of the patients succumbed despite HBOT treatment.

PROSPECTS

Hong Kong carries a different disease spectrum from that in other countries: the high incidence of intentional CO poisoning outnumbers most of the overseas countries. This discrepancy is multi-factorial, with Hong Kong being a stressful city where psychological illnesses are common, andresidential areas in Hong Kong being minute, rendering suicide by burning charcoal not difficult. This geographic uniqueness may provide us with a significant sample size for studying the clinical efficacy of HBOT on the prevention of delayed neurological sequelae from CO poisoning s. On another front, the number of CRAO patients referred to us also exceeded the originally expected patient load, which offers our Centre an excellent opportunity to optimise the number of HBOT treatment sessions necessary to achieve the best outcome in CRAO.

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Rottnest Island – Meet the Quokkas

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A 25-minute ferry ride from Fremantle, Western Australia, lies the Rottnest Island, a beautiful island with spectacular scenery. Every year, it attracts hundreds of thousands of visitors. The 19.7 km Rottnest Channel Swim from Cottesloe Beach to Rottnest Island, one of Australia's largest open water swimming events, is held yearly with thousands of participants.

The island has six major habitats: coastal, salt lakes, brackish swamps, woodlands, heath and settled areas. The beautiful bays and beaches are popular for water sports such as swimming, diving, snorkelling, and surfing. The limestone coral reef surrounding Rottnest provides a home to much of Rottnest's marine life and is an attraction for divers.

Fig. 1. The Basin, one of the popular swimming and snorkelling areas (Photo from personal collection)

Fig. 2. The Basin and Bathurst Lighthouse (Photo from personal collection)

Fig. 3. A salt lake (Photo from personal collection)

With no cars on the roads, bicycles and Segways are available for hire on the island. There is also hop-on-and-off Bayseeker Bus for one to travel around. People could also choose walking trails to explore the landscape of the island by themselves.

History lovers could join the Oliver Hill Train and Tunnel Tour, featuring military remnants from World War II including a 9.2-inch gun and maze of underground tunnels. The location of the island was seen as being important to the defence of the port of Fremantle, as bombardment of any attacking ships could be made from the island before the ships would come into range of the port. A light railway was built to transport materiel and munitions from the jetty to the guns. A number of lookouts were built around the island also. In the 1990s the gun emplacements and railway were reconstructed and introduced to tourists to admire the beauty of the Island's southside.

Fig. 4. Illustrations on the gun and underground tunnels (Photo from personal collection)

Fig. 5. View from Oliver Hill (Photo from personal collection)


Fig. 6. The train (Photo from personal collection)


Fig. 7. A quokka (Photo from personal collection)

All plants and animals on Rottnest Island are protected by law and well conserved. One of the main highlights on the Island is the Quokkas. The Quokkas were first misidentified as large rats by a Dutch explorer, William de Vlamingh, in 1696, and the island was named Rottnest – literally translating to "rats nest". They are in fact marsupials, mammals that have a special pouch for carrying their babies. The name "quokka" comes from the name given to the animal by the Aboriginal people living in Western Australia. There are 8,000-10,000 of quokkas on the island.

Quokkas are generally nocturnal and spend most of the day sleeping and resting under bushes and dense vegetation. On the island, they can be seen feeding in the shades during the day. The quokka's diet consists of grass, leaves, seeds and roots. Guided walks are available for tourists.

"All things bright and beautiful...", one could spend the whole day enjoying the beauty of Rottnest Island, in the sea or on the land, appreciating the greatness of nature.


Fig. 8. View from the jetty (Photo from personal collection)

The 10-year journey of developing a Dental Clinic Management System (DCMS) towards Electronic Health Record Sharing

Dr Johnny WONG
Chairman of Information Technology Committee, Hong Kong Dental Association

Dr Johnny WONG

74% of the registered dentists are working in the private sector (according to the Health Manpower survey 2015), and this sector supports the majority of the dental care service in Hong Kong. Most of the dentists manage their practices independently, though there are some group practices.

For a long time, most dental clinics have no computer system to support their clinic operations. Those with computer systems under-utilize them mostly for inventory and billing management. Users do not computerize their patients' medical records or surgical procedures records, reflecting a slow take-up in the use or sharing of electronic health records (eHR) amongst the profession. Although there are several existing clinic management systems for dentistry, none of them is good enough to address the needs of clinic management as well as to facilitate the management and sharing of medical and dental records which are designed for the situation in Hong Kong.

The key difficulties for the dental professionals in adopting computerized clinic management systems are:

1. Lack of vendor support: from IT vendors' perspective, Hong Kong's dental care industry is a dispersed and diversified market as most clinics are operated by independent dentists, so the requirements at each clinic may be small in scale yet varied in detail.
2. Lack of an open solution: the existing solutions are controlled by specific vendors who may themselves be small companies. High chance that there is a risk that those vendors will go out of business, leaving the dentists stranded with obsolete systems with no support.
3. Usability: most existing systems in the market have been developed by IT companies that have little understanding of the need for dental care practice and the legal requirements of *the Hong Kong Dental Council* for record keeping. Hence the resulting systems may be technically sound but difficult to operate for the practitioners.

In addition to the above deficiencies, existing commercial solutions suffer from additional limitations and concerns:

1. Extensibility: most of the existing solutions are used by a small number of clinics, so their cost of on-going maintenance support is high, and the pace of enhancement is very slow.
2. Data standard and interoperability: most existing systems are not developed with data sharing or system interoperability in mind, and they create silos of isolated and incompatible data. In case of data retrieving for medical concern or clearance will be very difficult and time consuming for the dentists.

Furthermore, the eHR sharing initiative put forward by the Government to allow sharing of patients' eHR between public and private healthcare providers, has yet to be widely and fully understood by the dental profession. This, added to all the above factors, could have affected the readiness and willingness of the IT industry in proactively developing a solution for Hong Kong's dental professionals.

In order to develop full population-wide healthcare records, standardized, sharable electronic patient dental records are necessary. Given the above, the HKDA, with the support from the Food and Health Bureau, has been working since 2009 to develop a strategic roadmap for developing and implementing a standard and unified dental clinic management system for dentistry and to improve the profession's computerization, encourage adoption of eHR and to pave way for the participation in Hong Kong's eHR Sharing System (eHRSS).

In May 2010, the eHR Office of the Food and Health Bureau provided support to HKDA to pursue the above objectives. In June 2010, DCMS 1.0, the first open-sourced dental clinic management system was launched at the Hong Kong International Dental Expo and Symposium (HKIDEAS), a major international event for the dental care profession. DCMS 1.0 offered a full solution for dental clinics to computerize their in-house operations. By 2012, more than 150 dentists have deployed DCMS 1.0 at their clinics, making DCMS 1.0 a leading de facto solution for dental clinics.

Since then, DCMS 1.0 was extended to support the pilot project on Outreach Primary Dental Care Services for the Elderly in Residential Care Homes and Day Care Centres. Moreover, the project website has also been visited regularly by both local and overseas visitors.

After the success of DCMS 1.0, HKDA continues to gather user feedback, via surveys and field support of the users.

As with all technological innovation, DCMS 1.0 satisfied the demand of the early adopters. However, to attract broader adoption, the system had to be more user-friendly to dentists and staff who wanted a hassle-free, out-of-the-box solution. In 2015, HKDA with the support of eHR Office, embarked on the development of DCMS 2.0, a more versatile, flexible and most importantly more user-friendly version to the typical users. What is more, the system is also capable of connecting with the territory-wide eHRSS.

DCMS 2.0, launched in 2016, in addition to numerous enhancements, supports integration with other dental equipment (such as digital x-ray machine or digital intra-oral camera) to enable a more integrated workflow.

Different from its earlier version, DCMS 2.0 is the first dental clinic management system that connects with eHRSS. Being one of the first healthcare professions that is enabled access to eHRSS, dentists registered with eHRSS can view a patient's medical record on the eHRSS, allowing them to have a more comprehensive view of a patient's profile. Optionally, dentists can also share data including appointment, diagnosis, allergy, and prescription to eHRSS, contributing to a patient full medical record to facilitate continuity of care.

To date, more than 1800 dentists and staff in the profession have undergone training on DCMS 2.0 and eHRSS. More than 100 private dentists have installed DCMS 2.0 and registered as eHRSS healthcare professional users, allowing them to access a patient's health record stored in the territory-wide system.

Today, DCMS 2.0 is a matured and versatile solution for managing 21st-century dental clinics. Users include not only solo clinics but also by major groups with multiple clinics, each with dozens of dentists.

The user feedback from the DCMS 2.0 has been constructive and encouraging. More than one fifth of the participants had shared their comments on the usage of DCMS 2.0, contributing to the improvement of the system and its further development plan. We found that the majority of participants are satisfactory for the new function added in the DCMS 2.0. In response to their valuable comment, we wish the system could be further improved to include more functions to benefit doctors and patients receiving dental service.

If you are interested in installing the DCMS 2.0, please contact us for the instruction for the installation procedure and requirement. Should you have any question regarding the DCMS 2.0, please do not hesitate to contact HKDA secretariat at 2528 5327. If you are interested in learning more about eHRSS, please approach the eHR Registration Office at 3467 6230.

[Dr Johnny Wong's interview at eHealth News: https://www.ehealth.gov.hk/en/publicity_promotion/ehealth_news_10/boarding_eHR_train.html]

FMSHK Annual Scientific Meeting 2019

Dr Chun-kong NG

2nd Vice-President,
The Federation of Medical Societies of Hong Kong

Dr Chung-kong NG

On 22 September 2019, the Federation of Medical Societies of Hong Kong (FMSHK) successfully held the Annual Scientific Meeting (ASM) 2019 at the Sheraton Hotel and Towers, with the theme of "Innovative Medical Technology".

A total of 23 medical talks were delivered by a panel of distinguished speakers. They shared with us the latest knowledge and developments in Health Service Management, Respiratory Health, Urology, Depression, Dermatology, Care for Advanced Diseases, Cardiovascular Diseases, Child Health, Diabetes Mellitus & Renal Health, Neurosurgery and Rheumatology & Immunology.

The opening ceremony of the ASM was star-studded by the following officiating guests: Prof the Hon Sophia Siu-chee CHAN, JP, Secretary for Food & Health Bureau; Prof Chak-sing LAU, President of Hong Kong Academy of Medicine; Dr Tony Pat-sing KO, Chief Executive of the Hospital Authority: Dr the Hon Edward Che-hung LEONG, GBM, GBS, JP; Mr Wen-shen LI (李文慎副部長), Liaison Office of the Central People's Government in Hong Kong SAR; and Dr the Hon Pierre CHAN, Legislative Councillor (Medical). The FMSHK feels much privileged and honoured by their kind presence.

Prof CHAN and the honourable guests identified the eight elements that archive Innovative Medical Technologies, namely, Target 目標, Strategy 策略, Connectivity 聯繫, Innovation 創意, Methodology 方法, Evaluation 評估, Synergy 協同 and Leadership 領導.

We would like to take this opportunity to express our sincere gratitude to our officiating guests, honourable guests, co-chairmen, chairpersons and speakers for their contribution to making the event a great success. Our gratitude also extends to various sponsors for their generous support.


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Medical Diary of January

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
			1	2	3	4
5	6	★HKMA-HKS&H CME Programme 2019-2020 Topic: Advance Decision Making Towards End of Life Care in People with Advanced Illnesses ★Diagnosis and Treatment of Adults with Community Acquired Pneumonia 7	★The Hong Kong Neurosurgical Society Monthly Academic Meeting –Nanotechnology and Neurosurgery 8	★Ultra-Long Acting Basal Insulin: Addressing Barries in Achieving Optimal Glycemic Control in T2DM 9	★Advances in Renal Disease Management 10	11
12	13	14	★Thyroid Nodules? Cancer, What a GP Needs to Know? 15	★Radiology and Intervention Musculoskeletal, Pain and Oncology Management 16	17	18
19	20	21	22	★HKFMS Foundation Meeting ★FMSHK Executive Committee Meeting 23	24	25
26	27	28	29	30	31	

Date / Time	Function	Enquiry / Remarks
7 TUE 1:00 PM	**HKMA-HKS&H CME Programme 2019-2020** **Topic: Advance Decision Making Towards End of Life Care in People with Advanced Illnesses** Organiser: Hong Kong Medical Association, Hong Kong Sanatorium & Hospital; Speaker: Dr. LEUNG Man Fuk, Edward; Venue: The HKMA Wanchai Premises, 5/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai	HKMA CME Department Tel: 2527 8452 1 CME Point
1:00 PM	**Diagnosis and Treatment of Adults with Community Acquired Pneumonia** Organiser: HKMA-KLN West Community Network; Speaker: Dr. LAM Sau Yee; Venue: Fulum Palace, Shop C, G/F, 85 Broadway Street, Mei Foo Sun Chuen	Miss Antonia LEE Tel: 2527 8285 1 CME Point
8 WED 7:30 PM	**The Hong Kong Neurosurgical Society Monthly Academic Meeting –Nanotechnology and Neurosurgery** Organiser: Hong Kong Neurosurgical Society; Speaker: Dr LI Ronald; Chairman: Dr CHEUNG Fung Ching; Venue: Seminar Room, G/F, Block A, Queen Elizabeth Hospital	1.5 points College of Surgeons of Hong Kong Name: Dr. WONG Sui To Tel: 2595 6456 Fax. No.: 2965 4061
9 THU 1:00 PM	**Ultra-Long Acting Basal Insulin: Addressing Barries in Achieving Optimal Glycemic Control in T2DM** Organiser: HKMA-New Territories West Community Network; Speaker: Dr. AU YEUNG Yick Cheung; Venue: Pak Loh Chiu Chow Restaurant, Shop A316, 3/F, Yoho Mall II, Yuen Long	Miss Antonia LEE Tel: 2527 8285 1 CME Point
10 FRI 1:00 PM	**Advances in Renal Disease Management** Organiser: HKMA-KLN City Community Network; Speaker: Dr. HO Chung Ping, MH, JP; Venue: President's Room, Spotlight Recreation Club, 4/F, Screen World, Site 8, Whampoa Garden, Hunghom, Kowloon	Miss Antonia LEE Tel: 2527 8285 1 CME Point
15 WED 1:00 PM	**Thyroid Nodules? Cancer, What a GP Needs to Know?** Organiser: HKMA-Central, Western & Southern Community Network; Speaker: Dr. WONG Chun Kuen; Venue: The Chinese Banks' Association Ltd, 5/F, South China Building, 1 Wyndham Street, Central	Miss Antonia LEE Tel: 2527 8285 1 CME Point
16 THU 1:00 PM	**Radiology and Intervention Musculoskeletal, Pain and Oncology Management** Organiser: HKMA-KLN East Community Network; Speaker: Dr. WAI Man Wah; Venue: V Cuisine, 6/F, Holiday Inn Express Hong Kong Kowloon East, 3 Tong Tak Street, Tseung Kwan O	Miss Antonia LEE Tel: 2527 8285 1 CME Point
23 THU 7:00 PM	**HKFMS Foundation Meeting** Organiser: The Federation of Medical Societies of Hong Kong; Venue: Council Chamber, 4/F, Duke of Windor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Nancy CHAN Tel: 2527 8898
8:00 PM	**FMSHK Executive Committee Meeting** Organiser: The Federation of Medical Societies of Hong Kong; Venue: Council Chamber, 4/F, Duke of Windor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Nancy CHAN Tel: 2527 8898

Answers to Dermatology Quiz

Answers:

1. Accessory nipple (supernumerary nipple)

   An accessory nipple is a minor birth defect that consists of an extra nipple. Usually, it is located along the embryonic milk lines which extend on both sides of the body from slightly above the armpit, down the chest and abdomen, to the inner thighs near the groin. Sometimes more than one of them can occur in same person. They are not inherited. The condition is more prevalent in men than in women. They can be present with no other tissue (polythelia) or with breast tissue and ducts (polymastia). The differential diagnoses include melanocytic naevus, birthmark, amniocentesis scar, acrochordon, etc.

2. Most isolated accessory nipples are not associated with any disease. A thorough workup for other malformations is usually unnecessary in a person who is otherwise healthy, although it has been reported that it might be linked to certain renal and urinary tract malformations and solid organ malignancies or some congenital syndromes, these have not been validated and may just be due to chance findings.

3. Most accessory nipples do not have any symptoms or complications; therefore treatment is usually not necessary. Occasionally they can be removed by surgery for aesthetic purposes or if there is discomfort, such as lactation or tenderness.

Dr Lai-yin CHONG
MBBS(HK), FRCP(Lond, Edin, Glasg), FHKCP, FHKAM(Med)
Specialist in Dermatology & Venereology

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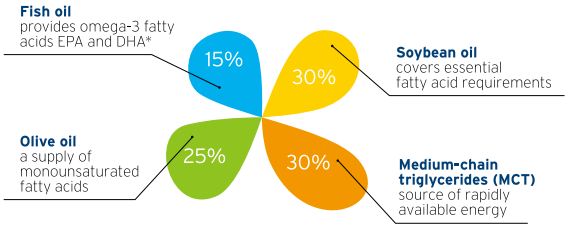

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