UNIVERSITY of CALIFORNIA, SAN DIEGO
MEDICAL CENTER HYPERBARIC DEPARTMENT

PORTAL VENOUS GAS ASSOCIATED WITH DECOMPRESSION SICKNESS IN SPORT DIVERS: A CASE SERIES

MD Koenig, MD, AJ Medak, MD, KB Van Hoesen, MD

Background: Atmospheric decompression, as seen with ascent while SCUBA diving, mountaineering, or flying in an unpressurized aircraft creates a thermodynamic disequilibrium in body tissues which affects the inert gases dissolved in the tissues. Depending on the degree and rapidity of change in atmospheric conditions, the gas can be eliminated physiologically without stressing organic systems, or it can evolve into gas bubbles which can create pathologic symptoms known as Decompression Sickness (DCS). This phenomenon is especially evident during diving when a person is breathing compressed air while at increased ambient pressures and creating a condition of supersaturation in the bodily tissues. During a controlled ascent a diver gives their body sufficient time to physiologically remove excess nitrogen, via the circulatory and respiratory systems, and achieve equilibrium while ascending to the surface. Factors such as depth of dive, length of dive, and selection of inhaled gas mixtures affect the amount of nitrogen distributed into bodily tissues. Rate of ascent determines whether this gas while be harmlessly off-gassed through normal physiologic mechanisms or form gas bubbles that can create pathological changes within the body, namely DCS.

Venous gas embolism (VGE), describes the presence of intravascular gas bubbles. Although VGE is associated with DCS, it is not pathognomonic for the condition. VGE was once thought to be exclusively related to cases of DCS, but this notion has been clearly refuted. Spencer, et al describe intravascular bubbles in SCUBA divers after no-decompression dives (Spencer, MP; J of App Phys, 1976). The majority of these divers with Doppler-detectable VGE do not develop DCS. These gas emboli are physiologically filtered in the pulmonary circulation through gas exchange at the alveolar level. In the scenario where the rate of ascent, or the extent of tissue nitrogen levels, exceeds the pulmonary capacity to off-gas the nitrogen, DCS may occur. This phenomenon is due to VGE traversing the pulmonary circulation and passing into the systemic circulation via the left heart and arterial circulation. The physiologic effects of these gas bubbles are vast, and many mechanisms have been described. Proposed mechanisms include damage to the vessel lumen and endothelial cells, activation of platelet and leukocyte aggregation, enhanced complement and coagulation activity, and proinflammatory cytokine release (Brubakk, AO and Neuman, TS; Phys and Med of Diving; 2003).

Case I

4/02/06

A 55-year-old male was referred to the Emergency Department (ED) by Diver's Alert Network (DAN) for evaluation of abdominal pain and a rash that began after diving. The patient was a certified rescue diver who dove that morning in good conditions with a dive-buddy and a dive computer. Patient's previous dive was over 1 week prior. Patient dove to 115 feet of seawater (fsw) maximum depth for total bottom time (TBT) of 30 minutes. According to the computer, this dive profile was a no decompression dive (no–D dive). The patient made a safety stop, had a controlled ascent, felt well, and swam back to shore with his buddy. After a 2.5 hour surface interval (SI) the patient and his buddy made their second dive; 85 fsw for 44 minutes. The computer again stated this was a no-D dive. Patient made a safety stop, had a controlled ascent, felt well at the surface, and swam to the beach. Patient denied gear malfunctions or problems with the dive.

While driving home approximately 45 minutes after surfacing the patient began to feel "crampy" epigastric abdominal pain without nausea or vomiting. The patient also noted gradual onset of "blurry" vision at this time. After he arrived home the patient noted a non-pruritic "bluish painful rash" in the epigastric area. Patient called DAN, and, after he described his symptoms, he was referred to the ED for evaluation.
When he arrived in the ED the patient noted the rash was nearly gone, his vision had returned to normal within minutes of onset of symptoms, and his abdominal pain was improving. He denied chest pain, shortness of breath, headache, and current vision changes. The patient noted he ate shrimp at a restaurant the previous day and experienced some diarrhea that morning prior to diving.

A complete review of systems was otherwise negative. The patient's past medical history was significant for dyslipidemia, benign prostatic hypertrophy, hypertension, and prostate surgery. The patient was taking zocor, niaspan, and norvasc. The patient's past dive history was significant for certification as a rescue diver with more the 300 dives. The patient denied allergies to any medications and also denied tobacco, heavy alcohol, or recreational drug use.

The patient was afebrile, with vital signs within normal limits. Physical exam revealed mild epigastric tenderness to palpation without rebound or guarding. The rest of the exam was unremarkable. There were no demonstrable rashes or cutaneous findings in the ED.
Diagnostic studies revealed normal liver transaminases, a complete blood count with elevated white blood cell count of 16.7 with 86% segmented neutrophils and 3% bands. The patient also had an elevated creatine kinase of 533 and his electrolytes were all within normal limits. Computed tomography (CT) of the abdomen revealed a small amount of gas in the inferior vena cava (IVC) as well as gas in the portal venous system.

Although the patient's dive profile was considered non-provocative for DCS, the timeline of symptoms and finding of hepatic portal venous gas prompted treatment with United States Navy Treatment Table 6 (USN TT6) for presumed DCS.

The patient had no complaints during his treatment, and after approximately 10-15minutes at 2.8ATA patient stated that "all symptoms were gone". The patient was subsequently discharged from the Hyperbaric Medicine Department, and he was advised to refrain from diving activities for 6 weeks.

Case II

7/22/06

A 34-year-old male diver was brought in by paramedics after a syncopal episode with complaints of left arm numbness as well as abdominal, thigh, and buttock pain. The patient reported diving at the Yukon (a submerged ship off the coast of San Diego) on Trimix 25% to 105 fsw for 65 minutes, SI of approximately 2 hours, and a second dive to 105 fsw for 56 minutes with a 12 minute decompression stop. He had no rapid ascent, no problems during the ascent, and no symptoms immediately on the surface after the dive. Approximately 30 minutes after surfacing the patient experienced "firey" abdominal pain radiating into his buttocks and bilateral thighs anteriorally. The patient related that he often gets these same symptoms when doing deep dives (200 fsw) and attributes this to nitrogen in his fat tissue. About 30 minutes after onset of abdominal pain, the patient went into the bathroom, felt nauseated, and experienced a syncopal episode. The patient was unsure of the length of time he was unconscious. Upon regaining consciousness he felt slightly better and got into his car to drive home. While driving, he began to have blurred vision and left arm numbness without weakness. The patient stated that he pulled over and experienced a second syncopal episode. After regaining consciousness for the second time the patient called 911 and was brought to the ED by paramedics.

A complete review of systems was otherwise negative, and the patient denied any existing medical problems or medications. The patient's past diving history included a career as a commercial diver involving use of mixed gas for deep water and cave dives. The patient denied any previously diagnosed or treated DCS or AGE. The patient also denied allergies to any medications as well as tobacco, heavy alcohol, or recreational drug use.

The patient was febrile to 102.2 F and mildly tachycardic to 102 with normal blood pressure and respiratory rate. The patient's oxygen saturation was 100% on room air. Physical exam revealed diffuse abdominal tenderness to palpation without rebound. The rest of the exam was unremarkable.

Diagnostic studies revealed mildly elevated liver transaminases with AST/ALT of 53/59. A complete blood count had an elevated white blood cell count of 16.8 with 85% segmented neutrophils and 0% bands. The patient also had an elevated creatine kinase of 512, and his electrolytes were all within normal limits. Computed tomography (CT) of the abdomen revealed a small amount of air in liver and the radiologist noted that it was "probably in portal or hepatic veins" but interpretation was limited by the absence of IV contrast.

Based on his presentation, the patient underwent treatment with USNTT6. While he was being treated, during his second oxygen period at 60 fsw, the patient endorsed complete resolution of his abdominal pain. The patient's repeat temperature was 97 F and heart rate was 75, and he was discharged home.

Case III

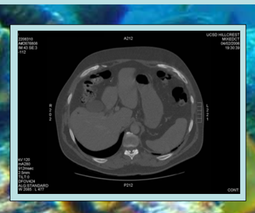
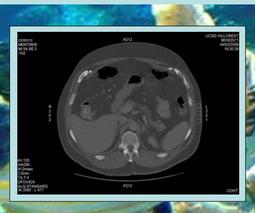
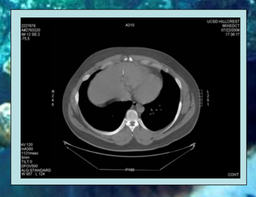
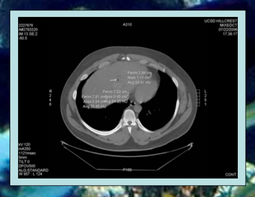
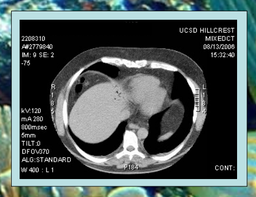
8/13/06

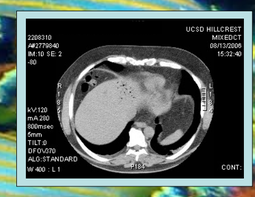
The patient from Case Study One presented once again to the ED with complaints of generalized abdominal pain following a dive earlier that day. The patient stated he dove off La Jolla Shores (a popular beach dive location in San Diego) earlier that day using a Suunto dive computer with the same dive buddy present in April. The patient dove twice; his first dive was to 80 fsw for approximately 50 minutes (with 20 minutes at depth) followed by a 90 minute SI and a second dive to 60 fsw for 62 minutes. During the second dive, approximately 40 minutes were spent at depth. During each of the two dives, the divers always had more than 12 minutes of no-decompression time left on their dive computers. The patient denied any adverse symptoms after either dive. The patient stated that 75 minutes after the second dive he started having symptoms of abdominal pain and nausea associated with transient visual blurring (lasting moments), left arm/elbow pain and left lateral leg numbness. He called his dive buddy (also a rescue diver) who promptly came to the house and started him on supplemental surface oxygen at 15L/min by non-rebreather mask. By the time the patient reached the ED (within an hour of symptom onset), his extremity symptoms had entirely resolved. His only presenting complaint was generalized abdominal discomfort. These dives were the first deeper dives (I would not consider these profiles to be "deep") the patient had done since prior HBOT, and he had completed 40 shallow dives since April without incident.

A complete review of systems was otherwise negative. The patient's past medical history, medications, and dive experience were unchanged since his last presentation to the UCSD Hyperbaric Center (please see Case Study One). The patient was afebrile, with vital signs within normal limits. Physical exam revealed mild diffuse abdominal tenderness to palpation without rebound or guarding.

The rest of the exam was unremarkable.
Diagnostic studies revealed a complete blood count with normal white blood cell count of 11 with 86% segmented neutrophils and 0% bands. The patient also had an elevated creatine kinase of 274. Computed tomography (CT) of the abdomen revealed small foci of gas within the IVC, intrahepatic portal veins, and mesenteric vessels of the transverse colon. Considering the patient's history and presentation he was treated with USN TT6. Upon reaching treatment depth and starting on 100% oxygen the patient stated that his abdominal pain had completely resolved.

He was discharged in good condition.



Discussion: Decompression Sickness (DCS) also commonly known as "caisson disease" or the "bends" was first described in 1841 by Triger, based on his observations using compressed air in coal mines in France (Brubakk, AO and Neuman, TS; D & E Phys and Med of Diving; 2003). It has subsequently been observed in multiple populations of people including sponge and oyster divers, bridge and tunnel builders (caisson workers), and military and recreational divers. Research performed in the commercial, military, and academic venues has provided insight to the pathophysiology and treatment as well as the prevention of this disease process.

Manifestations of DCS are many, and they include neurologic, musculoskeletal, constitutional, audiovestibular, cutaneous, cardiopulmonary, and lymphatic signs and symptoms. Gastrointestinal symptoms are not commonly described.

Abdominal pathology is not commonly associated with DCS. In the authoritative text on diving physiology, Francis and Mitchell do not describe gastrointestinal symptoms as a manifestation of DCS (Brubakk AO, Neuman TS; B & E Phys and Med of Diving; 2003). Further, reports of hepatic venous gas are rare. A case report by Butler, et al describes two volunteers who were compressed to 2.5 ATA in a hyperbaric chamber and then decompressed to 1 ATA at which point they underwent ultrasound examinations (Butler BD, et al; Undersea and Hyperbaric Med; 1995). One volunteer had hyperechoic foci within the liver, not present on baseline exam, that were presumed to be small bubbles within hepatic venules. Neither subject complained of abdominal pain.

Butler has also described using trans-esophageal echocardiography to evaluate decompression-induced bubbles in anesthetized canines (Butler BD, et al; Undersea and Hyperbaric Med; 1995). Portal venous bubbles were incidentally observed in all animals, but were observed to be completely extracted by the liver sinusoids in all but one case (this animal had a portal-to-hepatic venous shunt). Butler observed that bowel compression created showers of venous bubbles to be detected in the portal venous system.

As this portal VGE phenomenon has not been well described to date, its implications are as yet unclear. Other groups have made observations of gastrointestinal, and specifically hepatic, dysfunction in the setting of DCS. Freeman and Philip described elevations of GOT and GPT in rats with severe DCS (Freeman DJ, Philp RB; Aviat Space Environ Med; 1976). They have also encountered a human subject with DCS, who developed symptoms of gastroenteritis with elevations of LDH, GOT, and GPT (Philp RB, Freeman, DJ; Undersea Biomed Res, 1975).

Because reports of portal VGE with DCS are rare, Butler concludes that further studies on liver function in the setting of DCS are indicated (Butler BD, et al; Undersea and Hyperbaric Med; 1995). He suggests that portal VGE resulting from changes in atmospheric pressure (i.e. SCUBA diving, hyperbaric chamber exposures) are not clinically significant. This is in stark contrast to portal venous gas associated with bowel ischemia or infarction, which is a striking radiographic finding which often portends a poor prognosis.

Our case series at UCSD suggests that there may be correlation with portal venous gas after SCUBA diving and abdominal pathology due to DCS. The recurrent symptoms in both subjects could indicate some as yet undefined predisposition for DCS in these individuals, despite their having no known preexisting bowel disease. Further study, including CT evaluation of the hepato-portal circulation in SCUBA divers (with and without evidence of DCS) could provide much needed additional data in this investigation.

Key Points:

Decompression sickness following SCUBA diving is a significant clinical entity.
Portal venous gas has been visualized by multiple modalities following decompression.
Case reports exist documenting elevations of transaminases in individuals with abdominal pain following decompression.
Few studies exist which further investigate this phenomenon of portal VGE after SCUBA diving.
This case series suggests that there may be a relationship between hepatic portal venous gas and atypical abdominal manifestations of DCS after SCUBA diving.

Conclusions:

Further study, including CT evaluation of the hepato-portal circulation in SCUBA divers (with and without evidence of DCS) could provide much needed additional data in this investigation.