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CT finding of VGE in the portal veins and IVC in a diver with abdominal pain: A Case Report
N Bird. **Undersea & Hyperbaric Medicine**. Bethesda: Nov/Dec 2007. Vol. 34, Iss. 6; pg. 393, 5 pgs

Abstract (Summary)
The **finding** of **abdominal** venous gas emboli (**VGE**) on computerized tomography (**CT**) is reported for the first time **in a** recreational **diver**. The patient presented 2-3 hours after surfacing from two deep air dives and subsequently complained of visual blurring and **abdominal pain**. Gas bubbles **in** the inferior vena cava (**IVC**) and **portal veins** were found incidentally by computerized tomography (CT) during his work-up for **abdominal pain**. The patient was treated for decompression sickness (DCS) with **a** US Navy Treatment Table 6 and achieved complete resolution of symptoms. The routine use of **CT** for venous bubble detection **in** symptomatic **divers** is not endorsed, but may provide objective evidence of **VGE** when DCS is **in** the differential diagnosis and corroborative evidence would alter management.

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[Headnote]

Bird, N. CT finding of VGE in the portal veins and IVC in a diver with abdominal pain: A Case Report. Undersea Hyperb Med 2007; 34(6):393-397. The finding of abdominal venous gas emboli (VGE) on computerized tomography (CT) is reported for the first time in a recreational diver. The patient presented 2-3 hours after surfacing from two deep air dives and subsequently complained of visual blurring and abdominal pain. Gas bubbles in the inferior vena cava (IVC) and portal veins were found incidentally by computerized tomography (CT) during his work-up for abdominal pain. The patient was treated for decompression sickness (DCS) with a US Navy Treatment Table 6 and achieved complete

resolution of symptoms. The routine use of CT for venous bubble detection in symptomatic divers is not endorsed, but may provide objective evidence of VGE when DCS is in the differential diagnosis and corroborative evidence would alter management.

INTRODUCTION

The previously unreported finding of gas in the portal veins and IVC is described in a recreational diver. Venous bubbles (likely VGE) were discovered, incidentally, by abdominal CT when the diver presented to the emergency room with abdominal pain of uncertain etiology within 2-3 hours of surfacing from the second of two deep dives. The bubbles were noted prior to recompression treatment but were undetectable after a US Navy Treatment Table 6. The CT findings influenced the therapeutic approach and make this case unique.

The abundance of non-specific physical signs and ambiguous clinical histories has long made the diagnosis of DCS a challenge. Suspicion by non-medical personnel is often the initial trigger that prompts rescue efforts and transportation for definitive medical evaluation and treatment. The lack of reliable objective markers makes a provocative history, physical signs, and clinical acumen necessary to make an accurate diagnosis (1). The absence of symptoms is taken as the indicator of safe dive profiles and the basis for the original US Navy decompression limits (2). Venous gas emboli detected by Doppler, while not diagnostic of DCS, can be used as a measure of dive profile severity (1,2,3,4). Thus, if the likelihood of developing DCS is associated with circulating bubble volume, quantification of bubble load may provide a means of grading both dive severity and relative risk of DCS (1-11). Attributing relative risk values may guide clinical decision-making when the history and physical signs are ambiguous and the patient presents proximate to surfacing. Computerized tomography may provide additional or distinct bubble volume information from that of Doppler ultrasound and may further our understanding of the relationship between VGE and DCS.

CASE REPORT

The patient was a 5 5-year-old male referred to the Hyperbaric Medicine Department at the University of California San Diego by the Divers Alert Network for possible DCS. He was an experienced diver who had performed similar dive profiles without incident. The patient presented 2-3 hours after completing two computer-assisted no-decompression dives on compressed air. His first dive was to 115 fsw for 30 min with a 3-min safety stop at 15 fsw. His ascent was controlled and he had no complaints after the dive. Following a 2.5-hour surface interval, he returned for a second dive to 85 fsw for 44 min that also included a safety stop. Both dives were completed using a Suunto Gekko dive computer that did not indicate the need for a decompression stop. The patient denied having equipment problems or symptoms of pain, shortness of breath, loss of consciousness or discomfort on reaching the surface.

Forty-five minutes after the conclusion of his second dive, he developed blurred vision on the drive home. He stated that the visual symptoms were short-lived and resolved within minutes of onset. Once home (approximately one hour after surfacing from his second dive), he started having abdominal pain and noted a blue/ purple rash on his abdomen that was tender to the touch. He denied a history of recent trauma, suit squeeze, nausea, vomiting, headache, chest or joint pain, loss of consciousness, shortness of breath, incontinence, urinary retention, numbness, weakness, or paresthesias.

He called the Divers Alert Network and was referred to the UC San Diego Hyperbaric Medicine Center for further evaluation. He arrived at the Emergency Department approximately 2 hours after exiting the water from his last dive. On presentation, his visual symptoms and abdominal rash had resolved. His epigastric pain was still present, but mild (2 out of 10 on the pain scale). He had no other complaints of pain. He had no positive neurological findings or neurocognitive deficits based on a complete

neurological examination. In addition, his Folstein mini-mental status examination score was 30/30 and his cortical function assessment tests (Trail Making Test, part A and clock drawing) were normal. A chest radiograph was negative. Examination of his joints was negative for swelling, weakness or limitation ofmovement. He hadno subcutaneous emphysema, rash, or lymphedema.

Because of his abdominal symptoms, he underwent an abdominal CT scan with contrast, which noted air bubbles in the portal veins and inferior vena cava IVC). Additional work up included a CBC (WBC 17,000/uL, Hct 44%, Hb 14g/dL, Pit 276,000/uL, bands 3%, polys 86%) and a liver panel (ALT, AST, alkaline phosphatase, albumin, direct and total bilirubin and total protein) that were all within normal limits. Based on his provocative dive profiles, clinical history, the timing of his symptoms relative to leaving the water, air in his IVC and elevated white count, a diagnosis of possible DCS was made, and he was treated with a US Navy Treatment Table 6 with no extensions.

Prior to recompression therapy he had only mild abdominal discomfort, which resolved within 10-15 minutes of reaching treatment depth and starting on hyperbaric oxygen. The remainder of his treatment proceeded normally and the patient had complete and sustained resolution of his abdominal pain. After treatment, the abdominal CT was repeated and was negative for air in the portal veins and IVC.

DISCUSSION

The use of Doppler as a method of evaluating dive procedures and decompression tables dates to the 1970's (1,2,3,5-11). The amount of venous bubbling has been used to alter diving practices, table configuration and the establishment of decompression limits. High bubble loads are considered a key factor in the development of DCS (1,2,3,4). Venous gas has been detected in both provocative and nonprovocative dive profiles in both symptomatic and asymptomatic cases. However, Doppler detection of venous gas correlates poorly with the occurrence and diagnosis of decompression sickness (1,2,3,4,5,6). Due to low diagnostic specificity and undocumented impact on disease management, bubble-imaging techniques are not commonly employed in cases of suspected DCS, other than to exclude confounding conditions such as cerebral hemorrhage or pulmonary blebs (1,5,14).

Venous gas bubbles were noted on CT 2-3 hours after the completion of his second dive. According to Flook, initial bubble detection in compressed air breathing tunnel workers, was commonly seen up to an hour after surfacing with a peak bubble volume 90-120 minutes after surfacing and persisting unchanged for 2-3 hours (15). In divers, this timeline is accelerated with peak bubble loads seen in the first half hour after surfacing (1-7,11,12).

On review of this patient's prerecompression abdominal CT, gas was present in the peri-colonic veins, the portal system and IVC, but none was visible superior to the liver. The detection of venous gas 2-3 hours after the completion his dives, suggests that he had a "significant" nitrogen load and persistent release of gas from his tissues. However, the exact origin of these gas bubbles remains uncertain. It is conceivable that they originated within the bowel wall due to supersaturation. It is equally plausible that they originated from gas within the bowel lumen. Others have reported portal venous gas in non-diving situations such as blunt abdominal trauma and gastric distention (16,17,18). This explanation would alter the etiology of this case to bowel distention on decompression secondary to barotrauma rather than intravascular bubble formation.

The dive profiles described above, when evaluated by US Navy or recreational dive tables are provocative and require mandatory decompression. The dive computer credits the user for time spent at shallower depths and thus permits longer dive times. In contrast to the computer, the dive table is based on the premise that all time spent underwater (or under pressure in the case of hyperbaric chamber

exposures) occurs at the maximum depth and accounts for stricter mandatory decompression requirements. Despite the no decompression status of this patient's dive profiles by computer, his practices nonetheless pushed the limits and played a significant role in the decision to treat his presenting symptoms as DCS.

In addition to the provocative nature of his dive profiles, a decision to treat was based on the proximal occurrence of visual blurring and abdominal pain and rash following his second dive coupled with the finding of gas on his abdominal CT scan. The presence of venous gas on CT is consistent with established Doppler findings but is not diagnostic of DCS or a "stand-alone" indication for recompression therapy. Conversely, the absence of venous bubbles would also not support withholding HBOT if other corroborative signs or history were present that suggested DCS.

Despite the weak correlation between the presence of VGE and DCS symptoms, bubble resolution constitutes the physiological basis for the use of HBOT The finding of VGE in this patient is consistent with many Doppler studies that show venous bubbles after dives (1-4, 6-12). The unique aspect in this diver is the finding of portal and IVC gas on CT where DCS could not be ruled out. Although this may have been part of the normal off-gassing process after the dive, and its significance remains unclear, the abdominal CT was indicated to evaluate other causes for his abdominal pain. Venous bubbles in this case, incidental or not, did influence the decision to treat because his clinical presentation was not diagnostic of DCS. The finding of VGE by no means recommends the routine use of CT to support a clinical diagnosis of DCS. However, as CT is usually available in the emergency room and is unlikely to significantly delay definitive treatment, its use in the setting of abdominal pain after diving could be considered if the diagnosis is unclear. Research is needed to determine if VGE on CT is a common finding in the post-dive period and if it identifies a distinct population from those with VGE by Doppler.

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