Diver with acute abdominal pain, right leg paresthesias and weakness: A case report

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Wang J, Corson K, Minky K, Mader J, A diver with type II decompression sickness and acute appendicitis: A case report. Undersea Hyperb Med 2002; 29(4): 242-246 - A 29-year-old man was brought to an emergency department by the United States Coast Guard with chief complaints of severe abdominal pain, right leg paresthesia and weakness following four deep air dives. Physical examination before recompression treatment was remarkable for diffuse abdominal tenderness and right leg weakness. The patient was diagnosed in the emergency room with type II decompression sickness (DCS) and underwent standard recompression therapy. He experienced complete resolution of weakness after hyperbaric oxygen (HBO) therapy, but his abdominal pain was persistent. Further investigation led to the diagnosis of acute appendicitis with perforation. The patient underwent appendectomy and intravenous antibiotic therapy and was discharged to his home on hospital day five without complications. This case reinforces the importance of careful clinical assessment of divers and illustrates the potentially wide differential diagnosis of DCS. This is the first reported case of recompression treatment of a diver with acute appendicitis and type II DCS.

DCS, HBO₂, diver, abdominal pain, paresthesias, appendectomy

INTRODUCTION

Recreational scuba diving has become a popular sport in the United States, with almost 9 million certified divers. DCS is an uncommon condition, but poses a serious risk of scuba diving with potential significant morbidity and mortality (1). Diagnosis of DCS can be difficult because the presentation is often nonspecific. We report a case of a 29 year-old white male who presented with severe abdominal pain, right leg paresthesia and weakness following four deep air dives. The patient was initially diagnosed with possible DCS and treated with hyperbaric oxygen (HBO) therapy. After closely following the patient’s clinical course, a perforated appendix was
also suspected. The patient underwent emergency laparotomy where intra-operative findings demonstrated a suppurative gangrenous appendix and grossly purulent fluid in the peritoneum. He was treated with intravenous antibiotics, and recovered fully without any complications.

CASE REPORT

A 29-year-old white male was brought to a university hospital emergency room by helicopter with acute onset of abdominal pain, right leg paresthesia and weakness following four deep air dives. He had been in his usual state of health, and he was in the second day of a diving trip. His dive profiles on the day of admission were: 92ft /34min; 82ft/40min; 74ft/20min; 72ft/25min. Twenty minutes after his last dive, the patient developed sudden right leg numbness, tingling, weakness and epigastric pain. The abdominal pain was associated with nausea and vomiting, and became progressively worse over the subsequent three hours. Investigation indicated that the patient had approximately 67 minutes of omitted decompression on the last four deep air dives. He was started on oxygen immediately and flown by the Coast Guard to the hospital. At the time of arrival, he was alert and oriented, he complained of right leg weakness and abdominal pain, however, the numbness, tingling sensation in his right leg reported earlier had resolved. He denied fever or chills, had no change of vision, and no chest pain or shortness of breath, or muscle or joint pain. The past medical history was only significant for hypospadias, for which he underwent surgical repair as a child. Physical examination revealed a well-nourished, well-developed white male. He was alert and oriented, apparently in acute distress. Blood pressure was 130/78 mmHg, pulse 87 beats per minute, respiratory rate 28 per minute, and temperature 37°C. The mucous membranes appeared dry, and no skin rash, ecchymosis, or lymphadenopathy was found. The chest was clear to auscultation and his heart had a regular sinus rate and rhythm without murmur. The abdomen was soft, tender to palpitation diffusely, but no guard and rebound; his bowel sounds appeared hypoactive. The scrotal area showed mild erythema and was tender upon palpation and rectal tone was normal.

A neurological examination at emergency room showed his cranial nerves were grossly intact, sensation including light touch, vibration and position were intact. Mild weakness was noted in right lower extremity, with a motor strength III-IV/V. Deep tendon reflexes were slightly hyperactive in the right lower extremity. The Romberg test, finger to nose and Babinski sign were negative.

A chest x-ray at emergency room showed his lungs were clear and a plain film of the abdomen showed no free air or dilated loops. An electrocardiogram showed a normal sinus rate and rhythm. Other laboratory findings showed a white blood cell count of 22,000/mm³ with granulocytes 88%, lymphocytes 4.4%, monocytes 7.7%, eosinophils 0.3%, basophils 0.3%. Blood hemoglobin was 16.5 g/dl, hematocrit 46%, platelet 187/µl. Serum sodium was 137mEq/l, potassium 4mEq/l, chloride 99 mEq/l, bicarbonate 24 mEq/l, BUN 15 mg/dl, creatinine 0.87 mg/dl, serum glucose 122 mg/dl, alkaline phosphatase 103 u/l, aspartate aminotransferase (AST) 45 u/l, alanine aminotransferase (ALT) 46 u/l, gamma-glutamyltransferase (γ-GGT) 25 u/l, lactatedehydrogenase (LDH) 844 u/l, amylase 57 u/l, lipase 35 u/l, total protein 8.2 mg/dl, albumin 4.8mg/dl, total bilirubin 0.7 mg/dl, troponin T (TnT) <0.01 µg/l, creatine kinase (CK) 288 u/l, creatine kinase isoenzyme MB (CK-MB) 7.1 u/l, CK-MB index 2.5, partial thromboplastin (PT) 18 seconds, activated partial thromboplastin time (APTT) 38 seconds, and international normalization ratio (INR) 1.8.
A tentative diagnosis of Type II DCS was made on the basis of the patient’s neurological symptoms, the timing of onset of symptoms, and the history of omitted decompression. The patient was started on intravenous fluid and sent to the hyperbaric chamber for oxygen recompression therapy. The patient was initially recompressed on a Comex 30 treatment table using 50/50 N2/O2 at maximal depth and in accordance with the table at 60 fsw. The patient was then switched to United States Navy (USN) Table 6. The patient reported a near complete resolution of his right lower extremity weakness after 30 minutes of treatment, but continued complaining of abdominal pain and vomited twice during the first 30 minutes of treatment. Urinary retention was also noted during treatment and after catheterization 450ml of urine was obtained and a sample sent for analysis. The abdominal pain persisted and gradually localized to the right lower quadrant of his abdomen.

Physical examination performed immediately after completion of the HBO₂ therapy showed: blood pressure 170/100 mmHg, pulse 92 beats per minute, respiratory rate 29 per minute, and temperature 37.8°C. The patient’s abdomen became rigid with severe tenderness to palpation in the lower abdomen. Along with maximal tenderness at McBurney’s point, rebound tenderness was also present. Costovertebral angle tenderness was absent and a careful neurological examination was normal.

General surgery was consulted and the patient taken to the operating room for exploratory laparotomy. Intra-operative findings demonstrated suppurative gangrene of the appendix and grossly purulent fluid in the peritoneum. Appendectomy was performed, the peritoneal cavity was carefully irrigated, and a drain left in place. Culture of the peritoneal fluid yielded *Citrobacter Pfreundii*, and histopathology confirmed acute transmural appendicitis and peri-appendicitis. The patient was treated with intravenous antibiotics postoperatively and discharged to home on hospital day six without complications.

**DISCUSSION**

Serious decompression injuries can be life-threatening incidents. DCS may be classified into Type I (mild or pain only) and Type II (serious or neurological) (1). Neurological symptoms or signs beginning after diving require immediate investigation for DCS and delays in diagnosis are undesirable due to the potentially progressive nature of the condition. The most common presenting symptoms in type II DCS are sensory findings such as paresthesias and limb weakness. Abdominal and lower back pain are encountered in spinal cord DCS (3). Abdominal pain in diving also has been reported in abdominal barotrauma causing rupture of stomach (6,7), intestine (8), and diaphragm (9,10). Mesenteric venous thrombosis following diving (11) can also present as abdominal pain. Pathophysiological events in DCS predispose to vascular obstruction and venous infarction (12,13).

The diagnosis of DCS can be challenging since the presentation of DCS is variable and nonspecific, and there are no diagnostic tests currently available other than clinical response to recompression. The urgent nature of the problem demands rapid clinical assessment and decision-making; misdiagnosis or delay in recompression may lead to poor clinical outcome, especially in cases of type II DCS. Other medical conditions can also mimic or co-exist with DCS. Butler (16) reported a case in which an initial evaluation considered musculoskeletal DCS, but a pneumothorax was identified and successfully treated without sequelae. Siemonowski et al (17) presented a diver with viral infection masking DCS and another diver with DCS misdiagnosed as viral infection. Beckman (18) reported a diver initially diagnosed as having type II DCS who was later found to have viral meningitis. Vasculitis masquerading as...
neurological DCS has also been reported (19). A diver who became unconscious after gastroenteritis was diagnosed as having DCS, but subsequent investigations revealed mental status changes resulting from maple syrup urine disease (partial-chain ketoacid decarboxylase deficiency) (20). Appendicitis co-presenting with DCS has not been reported previously.

In our case, a diagnosis of DCS was initially made based on the patient’s presenting symptoms and signs (right leg paresthesia, weakness pain, along with abdominal pain and dysfunction of the bladder), timing of onset of symptoms (twenty minutes after diving), risk factors (multiple dives and dehydration), and omission of decompression (approximately 67 minutes during four deep air dives). Carefully observing the progress of clinical signs of abdominal pain during and after HBO treatment shifted attention to the second diagnosis of acute abdomen. The patient underwent emergency appendectomy, was treated with intravenous antibiotics, and recovered fully without complications. The case reinforces the importance of differential diagnosis of neurological DCS, especially when primary treatment with oxygen recompression tables is not completely successful. To our knowledge, our case is the first report of recompression treatment of a diver with type II DCS and acute appendicitis.

REFERENCES
