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PNEUMOPERITONEUM AFTER DIVING – TWO CLINICAL CASES AND LITERATURE REVIEW

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ABSTRACT

Pneumoperitoneum after diving is a rare symptom. Diagnosis and treatment strongly depends on the primary source of the air in the abdominal cavity. There are two main sources of air entering the perineum: perforation of the gastrointestinal tract and pulmonary barotrauma. The management is different and additionally, in both cases, the decompression sickness and arterial gas embolism as consequences of inappropriate decompression phase of the diving should be included in the clinical diagnosis and treatment. The multidisciplinary team including hyperbaric physicians and surgeons is necessary for proper management of such cases. In this paper two cases of pneumoperitoneum of different origins are presented and similar cases reported in the literature are discussed.

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INTRODUCTION

During diving all gaseous spaces are subjected to the Boyle's law which relates changes of volume with ambient pressure ($P \times V = const$). When gas in enclosed in the anatomical cavity, its volume changes accordingly to fluctuations of the ambient pressure. Relative changes are greatest nearby the surface, which is against the so-called "common sense". Therefore the beginning of the dive (descent), as well as its ending (surfacing) are critical phases. Pathophysiological consequences of those changes in volume and pressure depend on elasticity of anatomical cavities, where the gas is enclosed. The pressure difference greater that compensatory limits of the compressible or expandable structure, can result in dysbaric injury of that space called barotrauma (1). The injury which occurs most often in recreational diving, is the middle ear barotrauma. This is usually caused by the upper respiratory tract's infections leading to situations when the eustachian tubes do not open freely. On the other side, the most serious of the dysbaric injury is the pulmonary barotrauma associated with ascent. It usually occurs when diver holds his breath or inhales water. In a situation like this, rupture of the thin-stretched walls of the alveoli will occur and air embolism, pneumothorax or subcutaneous emphysema will be the end result. Other forms which are rare include: sinus barotrauma, face squeeze and eye barotrauma (as result from wearing a tight mask and not equalizing pressure during descent), aerodontalgia. Due to great elasticity of the gastrointestinal (GI) tract its barotrauma is extremely rare and usually results in stomach rupture and pneumoperitoneum. As a symptom, free gas in peritoneal cavity can be easily diagnosed, however its presence does not confirm the perforation of the GI tract, as there are cases reported when pneumoperitoneum was recognized with no evidence of perforation of the GI walls. In every case of pneumoperitoneum after diving, as well as in all other diving accidents, the differential diagnosis and related treatment needs also to include the decompression obligations of the diver caused by the inert gas dissolved in tissues which sometimes require the hyperbaric therapy.

THE AIM

This paper describes two clinical cases of patients with pneumoperitoneum due to different origins related to recreational diving, and discusses the pathophysiological aspects as compared to other similar reports from literature.

CASE REPORT

Clinical case 1

A 45-year-old male amateur diver had been submerged for about 30 minutes diving alone to the maximum depth of 13 meters using compressed air. At the end of the dive he practiced the exercise of leaving the scuba equipment on the bottom of the lake and surfacing „on exhalation" from the depth of 10 meters simulating the emergency ascent. According to his report he exhaled air from lungs "as instructed" on the diving course. Just after surfacing he experienced the intense abdomen pain and this feeling stopped him before diving again to recover his diving gear. He was able to swim himself on surface to the beach, where he called for help. The abdomen pain was still intense and this was accompanied by abdominal distension with feeling of fullness in the stomach, and later nausea occurred without vomiting. In his medical history there was arterial hypertension and myocardial infarct in the past (when he was 34-year-old).

As an emergency, he was referred to the nearest general hospital, where neither neurological sign nor symptom was diagnosed. Because pulmonary barotrauma of ascent with possibility of air embolism was suspected due to the high risk underwater activity, he was admitted to the National Centre for Hyperbaric Medicine in Gdynia.

At presentation the patient complained of generalized abdominal pain with diffuse abdominal distension, tenderness with guarding and rebound tenderness. There was no clinical evidence neither of decompression illness nor neurological deficits, however – due to intense abdominal pain – the Romberg test could not be performed. Blood tests showed: WBC 16.2 G/l (normal range: 4.0-10.0); HGB 15.2 g/dl (normal range: 14.0-18.0); HCT 43.2 % (normal range: 38.0-54.0); OB 2 mm/h (normal range: < 10); PLT 251 G/l (normal range: 130-440); ALAT 63 U/l (normal range: 5-41); ASPAT 48 U/l (normal range: 5-37); CK 286 U/l (normal range: < 170); troponin I 0.06 ng/ml (normal range: < 0.5); Na+ 143.3 mmol/l (normal range: 135-148); K+ 4.6 mmol/l (normal range: 3.5-5.3). Chest and abdominal X-ray demonstrated gross pneumoperitoneum (Figure 1), but no evidence of pneumothorax or pneumomediastinum. Because there was no evidence of decompression illness, the recompression treatment was not commenced. The patient was referred to the Department of Surgery of the General Hospital in Gdynia with the diagnosis of suspected perforation of the GI tract. The laparotomy was commenced with no further delay and the gas was evacuated from the peritoneum. However – regardless of careful examination of the GI tract – no evidence of leakage was found. On the following day, the CT scans were performed, and stranding in the perigastric fat adjacent to the fundus and to the lesser curvature was found. Because there was no evidence of leakage of contrast medium, it was interpreted

as perforatio tecta. In chest CT scan there were some subtle evidences of diffuse pulmonary infiltrates and atelectasis. The patient was discharged 4 days later with no further treatment necessary. He was advised to perform medical examination for fit-to-dive certification after several weeks.

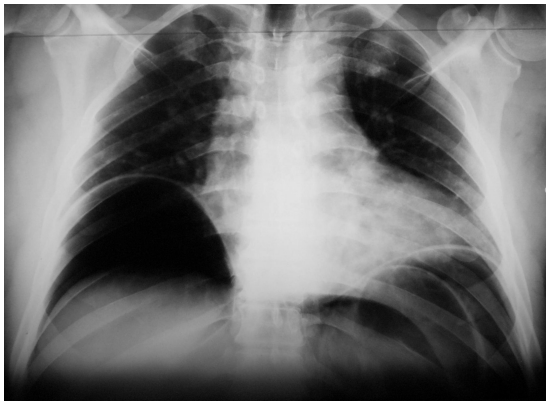

Figure 1. Chest X-ray showing pneumoperitoneum (clinical case 1).

Clinical case 2

During training dive to the depth of 35 meters, a 29-year-old male amateur diver had to conduct procedure of simulated emergency surfacing after failure of breathing apparatus. According to the plan, he should ascent to the depth of 5 meters without any inspiration, only exhaling. During the ascent, he felt the fullness in his abdomen, and after surfacing he claimed on the chest discomfort, however without cough and haemoptysis typical for pulmonary barotrauma. He was referred to the nearest general hospital, where subcutaneous emphysema on his face, neck and upper thorax was noticed. He was haemodynamically stable, with no clinical evidence of neurological deficits. Chest X-ray revealed pneumomediastinum and subdiaphragmatic air. The patient was referred to the National Centre for Hyperbaric Medicine in Gdynia, where repeated chest X-ray confirmed free gas below the diaphragm (Figure 2, Figure 3) and subcutaneous emphysema, however there was no radiographic evidence of pneumomediastinum any more. Additionally, in lower segments of the left lung there were observed inflammatory infiltrates. Laboratory tests showed: WBC 11.6 G/l (normal range: 4.0-10.0); HGB 15.3 g/dl (normal range: 14.0-18.0); HCT 45.5 % (normal range: 38.0-54.0); OB 8 mm/h (normal range: < 10); PLT 137 G/l (normal range: 130-440); ALAT 20 U/l (normal range: 5-41); ASPAT 29 U/l (normal range: 5-37); CK 250 U/l (normal range: < 170); Na+ 143.5 mmol/l (normal range: 135-148); K 5.74 mmol/l (normal range: 3.5-5.3). The recompression treatment was not indicated

because there was no clinical sign of decompression illness. The patient was still haemodynamically stable and he did not complain of abdominal pain, so he was only observed for any complications that could happen. On a third day, the free gas below the diaphragm resolved and in the chest X-ray there were evidences of regression of inflammatory infiltrates in lower segments of the left lung. The patient was then discharged from the hospital with advice not to dive again.

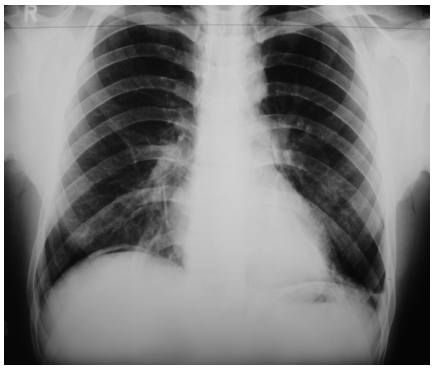

Figure 2. Chest X-ray showing pneumoperitoneum (clinical case 2).

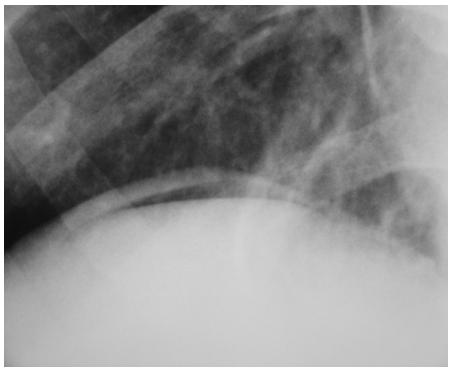

Figure 3. Zooming of sliver of free air below the right hemidiaphragm (clinical case 2).

DISCUSSION

Problems with gastrointestinal tract after diving are not so rare as could be expected based only on its high elasticity preventing from significant changes of pressure inside it. In response to a questionnaire sent to recreational divers (2), 2053 scuba divers gave information about GI discomfort during or after diving. GI complaints (including nausea) were reported in 275 cases (13.4%). One hundred and eleven reports (5.4% of all 2053) were interpreted as results of significant GI distension, excluding other causes which may have been secondary to vertigo, sea sickness, impure breathing mixtures, nitrogen narcosis or food poisoning.

The most frequent reason for GI distention during diving is air ingestion, called aerophagia. This phenomenon can be induced by several independent causes: Toynbee's maneuver (swallowing against occluded nostrils) for equilibration of the middle ear pressure during descent or hypersalivation induced by mouthpiece of breathing

apparatus or by incorrect diving technique (eg. during descent with head down the regulator is oversensitive and sometimes pushes the air into divers' mouth due to pressure difference between mouth and center of lungs). The incorrect diet can also sometimes lead to abdominal discomfort due to GI distension.

On ascent, the volume of the distended GI tract changes according to the Boyle's law. Surfacing from the depth of 10 meters results in reducing the ambient pressure from 2 ATA down to 1 ATA. This results in doubling of volume of any enclosed gas spaces including GI tract. This can occasionally lead to perforation of the GI tract, regardless of its elasticity and possible evacuation of gas by natural openings. Such GI tract perforations are rare; however they heave been reported in the literature. Since 1969 twenty cases of GI tract perforation during diving had been reported (3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18). The dives associated with this clinical event were deep (more than 25 meters) and brief (less than 15 minutes). Almost always there were some problems with diving equipment or with divers leading to gasping and ingesting water accidentally forcing divers to surface in emergency mode. Usually after surfacing, the symptoms typical for GI tract perforation (including pneumoperitoneum) occurred. In most publications there is a lack of detailed information about the past history of the previous pathology of the GI tract (stomach and duodenum). Lamy in 1972 (after (4) reported a case where X-ray examination conducted few days after conservative management of the GI tract perforation, revealed two peptic ulcers of the lesser curvature of the stomach and in the duodenum. In other 17 cases referenced above, the clear identification and localization of the lesions was performed using gastroscopy or explorative laparotomy. Most of those lesions (14 of 17 cases, 82.4%) were located on the lesser curvature of the stomach (3, 4, 5, 9, 10, 11, 12, 13, 14, 15, 16, 18), one lesion was located on the greater curvature of the stomach (4), one lesion was located on cardia (4) and in one case the lesion was localized in the upper cardia just at the gastro-esophageal junction (the Mallory-Weiss syndrome) (19).

Experimental examinations confirmed that most of the lesions during perforation of the GI tract will almost always be on lesser curvature of the stomach (11). In these experiments Margreiter et al. found in cadavers that 1750-2200 ml of water are necessary before rupture of the stomach occurs. Pressures necessary to rupture the stomach ranged from 96 to 156 mmHg. The explanation for the lesser curvature of the stomach as the typical location of the lesion is that on the lesser curvature, the gastric wall is composed of only one muscular layer (lack of mucosal folds) instead of three as elsewhere (4).

In full-thickness lacerations of the gastric wall, the surgical intervention is usually necessary to close the lesion. In other types of injury (non-full-thickness tear), the indications for operation are not so clear. Surgical intervention was commenced in 14 of

20 cases reported in the literature (3, 4, 5, 9, 11, 12, 13, 14, 15, 16). In 6 cases the conservative management was possible (4, 6, 7, 13, 19), including 3 cases, where the paracentesis was commenced in order to alleviate the patient's symptoms (4, 6).

Regardless of surgical treatment, in 6 out of 20 cases reported in the literature the hyperbaric therapy was conducted due to decompression sickness or arterial gas embolism as a consequence of emergency ascent to the surface (4, 5, 9, 14, 15). In all reported cases, the recompression therapy was performed before the surgical intervention. The rationale for such order of procedures is as following: 1) Cerebral arterial gas embolism needs immediate recompression in the hyperbaric chamber; on the other hand, surgical closure of the gastric perforation can be usually postponed for several hours in situations when patient is under strict supervision and hyperbaric session is conducted in multiplace chamber with medical attendant capable to diagnose any worsening of the clinical status inside the chamber; 2) During hyperbaric treatment the volume of free gas in the peritoneum decreases according to Boyle's law proportionally to the ambient pressure and this results in decreasing of symptoms related to the pneumoperitoneum; it must be remembered that during subsequent decompression after treatment, the volume of gas increases again, therefore the paracentesis should be performed before the hyperbaric therapy.

Pneumoperitoneum after scuba diving raises a real diagnostic problem, especially when decompression illness coexists. Proper multidisciplinary approach should lead to good prognosis as there was a full recovery in every case reported in the literature. The only exception is the single case of death after diving, when the perforation of the GI tract was diagnosed post mortem (10). In this case the leakage was localized on the lesser curvature of the stomach. The diver was heavily obese and he dived alone to the depth of 6 meters. He was found unconscious on the surface and he was subjected for resuscitation procedures. In this case, the direct cause-effect relation between diving and GI tract perforation cannot be ensured, as there are cases reported in the literature which confirm that artificial breathing during CPR in non-diving accidents can lead to GI tract perforation (20, 21, 22).

One of the clinical case presented here (case 1) is a typical case of a GI tract perforation during diving. There was no health problem with diver while under water, but simulated procedure of surfacing from a depth of 10 meters without breathing from diving apparatus is hazardous. Regardless of negative past history for GI problems, the GI tract perforation was diagnosed as based on clinical symptoms (abdominal distension and pain, fullness, tenderness and guarding) and pneumoperitoneum was clearly shown on the chest X-ray. After exclusion of arterial gas embolism, the explorative laparotomy was commences, but the surgeon found no evidence of leakage. The leakage was confirmed only during post-operation CT scan (perforation tecta) and it was localized

on the lesser curvature of the stomach. The subtle areas of lung involvement seen on CT scan can be explained by alveolar overdistention. When we now have knowledge, that in this case no perforation has been found during laparotomy, the interesting question is whether the clinical course would be similar if conservative management would have been conducted instead of surgical intervention. But the risk of peritonitis in case of leakage in the GI tract is high. Therefore we believe that when in doubts, the operation should be commenced. In the literature there are several reports of cases with pneumoperitoneum after scuba diving, when no lesion could be found during explorative laparotomy (4, 7, 14).

Even if in most cases of rupture of the GI tract the result is pneumoperitoneum, the free gas in the perineum can be detected also in other cases than leakage from the GI tract (23). In such cases, the main sources of gas are lungs. The most frequent cause is the positive pressure in lungs, eg. during the artificial ventilation or pulmonary barotrauma. A direct correlation between increased airway pressures and barotrauma has been demonstrated in animal models (24). The intratracheal pressure >40 cmH2O routinely resulted in interstitial emphysema; when pressures are >50 cmH2O, pneumomediastinum develops, and at pressures >60 cmH2O, subcutaneous emphysema and pneumoperitoneum are observed. Similar pressure thresholds were suggested for human data. In 28 clinical cases of pneumoperitoneum caused by mechanical ventilation, most peak inspiratory pressures exceeded 40 cmH2O (25). The open question is the pathway of air from thoracic to abdominal cavities. Two alternative mechanisms have been proposed (after (23)): 1) the direct passage of air through pleural and diaphragmatic defects, and 2) the classically described passage from mediastinum along perivascular connective tissue or major diaphragmatic portals to the retroperitoneum and finally to the peritoneum. In the literature there are only 4 cases reported with pneumoperitoneum as a result of pulmonary barotraumas during "abnormal" diving, most often connected with emergency ascent with incomplete exhalation (8, 17, 26, Zannini 1973 after (4)). In 3 of those 4 cases the pneumoperitoneum was the only symptom with no evidence of pneumomediastinum, pneumothorax nor subcutaneous emphysema (8, 17, 26), and in one case there was pneumoperitoneum and pneumomediastinum (Zannini 1973 after (4)). In all cited cases, the patients were recompressed in the hyperbaric chamber because of confirmed or suspected arterial gas embolism as result of pulmonary barotrauma. In one case the paracentesis was commenced before recompression to decompress the distended abdomen (17). Finally, in all 4 cases the full recovery was obtained without the surgical intervention.

The other case described here (clinical case 2) was similar to those 4 cases of pneumoperitoneum without the GI tract perforation reported in the literature. There was

risky activity performed underwater (simulation of emergency ascent from the depth of 35 meters to the depth of 5 meters) and typical symptoms with subcutaneous emphysema, which confirmed pressure injury to the lungs (pulmonary barotrauma). In our case, there was no evidence for arterial gas embolism, so the recompression treatment in the hyperbaric chamber was not indicated. The conservative management of the subcutaneous emphysema, pneumomediastinum and pneumoperitoneum resulted in full recovery in several days.

SUMMARY

Pneumoperitoneum after diving is a rare symptom, which needs an appropriate diagnosis and correct treatment approach. Differential diagnosis should include primary perforation of the GI tract and primary pulmonary barotrauma.

Management of the GI tract perforation and searching for lesion should be focused on its typical location on the lesser curvature of the stomach. This region should be checked carefully. There is no doubt that full-thickness tear of the GI tract needs to be surgically sutured. On the other hand, management of the non-full-thickness tears is not so obvious, especially when symptoms are not severe and clinical status of the patient is stable. It seems that conservative management and strict observation should lead to avoid surgical intervention at least in some cases.

On the other hand, pneumoperitoneum as a symptom of the pulmonary barotrauma can be safely treated conservatively, with paracentesis for decompression of distended abdomen when needed.

In every case of pneumoperitoneum after diving, the possible complications of breathing compressed gases under pressure (decompression sickness, arterial gas embolism) should be evaluated. The need for placing patient in the hyperbaric chamber for recompression therapy should be evaluated and the correct order of procedures should be decided. Therefore, in such cases, the multidisciplinary team including hyperbaric physicians and surgeons is needed.

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