CASE REPORT

Scuba diving, acute left anterior descending artery occlusion and normal ECG

Sébastien Xavier Doll,¹ Fabio Rigamonti,² Marco Roffi,² Stéphane Noble²¹Adult Intensive Care, Geneva Teaching Hospital (HUG), Geneva, Switzerland²Invasive Cardiology Unit, Geneva Teaching Hospital (HUG), Geneva, Switzerland**Correspondence to**
Dr Sébastien Xavier Doll,
sebastien.doll@hcuge.ch**SUMMARY**

We report the case of an acute proximal occlusion of the left anterior descending coronary (LAD) artery following a scuba diving decompression accident and associated with normal ECG. Following uneventful thromboaspiration and coronary stenting, the patient was discharged on day 4 with secondary preventative therapies. A transthoracic echocardiography performed at this point showed a complete recovery compared with an initial localised akinesia involving the anterior and apical portion of the left ventricle upon admission. This case highlights that significant acute coronary lesions involving the LAD can occur without any ECG anomaly. The presence of acute and persistent angina associated with troponin elevation should prompt physicians to consider coronary angiography without delay, independently of the ECG results.

BACKGROUND

Despite clear guidelines regarding the management of acute coronary syndrome (ACS) which state that a normal (ECG) does not preclude such a diagnosis, repetitive exposure to significant acute coronary disease associated with ECG anomalies may lead well-trained medical professionals to ignore this possibility. Furthermore, the advent of a concurrent clinical event (such as a scuba diving accident) may incite medical staff to pursue alternative diagnosis

despite clear signs and symptoms of ACS, with possible dramatic consequences.

CASE PRESENTATION

A male scuba diver in his mid-50s performed emergency surfacing without decompression stops after 15 min spent at -50 metres, as his auxiliary oxygen tank emptied abruptly. The patient was admitted pre-emptively to the emergency department of a tertiary hospital to rule out decompression sickness. On admission, the patient was completely asymptomatic. In particular, the patient was alert, oriented in time and space and cooperative and had no muscle or skeletal pain. Stage I obesity (body mass index at 34 kg/m²), hypertension and smoking were his known cardiovascular risk factors and the patient had no relevant medical history. Two hours after admission, the patient developed chest pain of moderate intensity without irradiation or other associated symptoms. Clinical examination during the chest pain episode revealed a blood pressure of 125/70 mm Hg, a regular heart rate of 86/min, a respiratory rate of 20/min. O₂ saturation was 97% on pulse oxymetry at an FiO₂ of 35%. Cardiac and pulmonary auscultations as well as neurological examination were all normal, as were the chest x-ray and the ECG (figure 1).

Initial laboratory analysis showed a haemoglobin level of 138 g/l (norm: 140–180 g/l), thrombocytes

Figure 1 Initial 12 derivations (ECG).

To cite: Doll SX, Rigamonti F, Roffi M, et al. *BMJ Case Reports* Published online: [please include Day Month Year] doi:10.1136/bcr-2012-008451

Figure 2 (A) Right anterior oblique projection showing the occluded LAD (left anterior descending artery) arrow. (B) Late image in the same projection showing a distal filling of the LAD by collaterals (see arrow).

counts at 192 G/l (norm: 150–350) and leucocyte counts at 9.9 G/l (norm: 4–11). International normalised ratio, blood glucose, electrolytes and creatinemia were all within normal

range. Troponin-I dosage, performed as routine, was elevated at baseline (0.8 µg/l, upper limit of normal 0.090 µg/l) and further increased 6 h later (15 µg/l). Suspecting a myocardial ischaemia in relation to nitrogenous emboli in the context of an acute decompression sickness, no further cardiological investigation was performed and the patient was transferred to our centre for hyperbaric chamber therapy.

A transthoracic echocardiography (TTE) was performed at arrival in our emergency room, showing a localised akinesia involving the anterior and apical portion of the left ventricle (LV), with a slightly decreased LV function. The ECG remained within normal limits for over 6 h. Owing to persisting chest pain and troponin elevation suggesting ongoing myocardial ischaemia, aspirin and unfractionated heparin were administered and emergency coronary angiography was performed. The invasive assessment revealed a thrombotic occlusion of the proximal left anterior descending artery (LAD), with partial collateralisation.

OUTCOME AND FOLLOW-UP

Following uneventful thromboaspiration and coronary stenting (figures 2 and 3), the patient was transferred to the intensive care unit for monitoring. Repeat TTE showed a regression of the anterior and apical akinesia and a complete normalisation of the LV function. The peak troponins were at 25 µg/l. Multiple subsequent ECGs were unchanged; in particular no R loss or no other abnormality was observed in the anterior leads. No hyperbaric treatment was performed and, following an uneventful hospital stay, the patient was discharged on day four.

DISCUSSION

In the care of the patient described, physicians were ‘fooled’ twice: first, they assumed a diving accident as the cause of chest pain; second, while myocardial ischaemia was suspected, a complete occlusion of the LAD without any ECG correlate was unexpected.

To our knowledge, this is an extremely rare case of ACS involving a complete occlusion of the proximal LAD associated with completely ‘silent’ ECGs. While the scuba diving incident

Figure 3 (A) Right anterior oblique projection post-thrombectomy with an export catheter (Medtronic). Culprit atheroma plaque shown by the arrow. (B) Material extracted by thrombectomy. (C) Final image post-stent implantation, arrow shows the stent.

was a distracting element in the initial management of this patient, it has certainly acted as a triggering factor leading to the rupture of a pre-existing atheromatous plaque in several ways.¹ First of all, the stress generated by emergency surfacing, fear of drowning and a cold environment, may have all contributed to the development of an ACS. Second, diving itself induces both an increase in ventilatory efforts and a redistribution of the blood flow towards the thorax associated with increasing venous return, all contributing to a higher cardiac workload. Hyperoxia occurs when scuba diving and has been associated with direct vasoconstriction of both peripheral and coronary arteries.² Finally, alteration of platelets functions and counts has been reported in association with uncontrolled decompression and decompression sickness, possibly leading to thrombus formation.^{3 4}

Silent ECG and coronary occlusions involving the circumflex coronary artery, the distal LAD or minor branches such as diagonals or marginal branches have been described elsewhere.^{5 6} In our case though it was the proximal LAD that was occluded over the first 50 mm of its proximal portion on coronary angiography. Late and incomplete filling of the distal LAD was observed, possibly resulting from partial collateralisation. It is assumed that the proximal LAD occlusion resulted in signs and symptoms of acute coronary occlusion, with the associated modifications observed on the initial TTE and troponin elevation. At the same time, minimal perfusion from those distal collaterals might have contributed to the complete lack of ECG anomaly presented by this patient.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Vann RD, Butler FK, Mitchell SJ, *et al.* Decompression illness. *Lancet* 2011;377:153–64.
- 2 Lafay V. The heart and underwater diving. *Arch Mal Coeur Vaiss* 2006;99:1115–19.
- 3 Tanoue K, Mano Y, Kuroiwa K, *et al.* Consumption of platelets in decompression sickness of rabbits. *J Appl Physiol* 1987;62:1772–9.
- 4 Barak M, Katz Y. Microbubbles: pathophysiology and clinical implications. *Chest* 2005;128:2918–32.
- 5 Turnipseed SD, Trythall WS, Diercks DB, *et al.* Frequency of acute coronary syndrome in patients with normal electrocardiogram performed during presence or absence of chest pain. *Acad Emerg Med* 2009;16:495–9.
- 6 Goodacre S, Pett P, Arnold J, *et al.* Clinical diagnosis of acute coronary syndrome in patients with chest pain and a normal or non-diagnostic electrocardiogram. *Emerg Med J* 2009;26:866–70.

Learning points

- The advent of a concurrent clinical event may distract medical staff to pursue alternative diagnosis despite clear signs and symptoms of acute coronary syndrome.
- Significant acute coronary lesions involving the left anterior descending artery can occur without any ECG anomaly.
- The presence of acute and persistent angina associated with troponin elevation should prompt physicians to consider coronary angiography without delay independently of the ECG results.

Copyright 2013 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- Submit as many cases as you like
- Enjoy fast sympathetic peer review and rapid publication of accepted articles
- Access all the published articles
- Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow