APRIL CASE DISCUSSION

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The subject of this case, passed away during the climb of Aconcagua. The death certificate granted by the local police does not account for the actual cause of the death. The subject was a fit 67 years old, male. He had previous experience of high altitude sojourns, such as Mt. Kilimanjaro (5,900m), Mt. Elbrus (5,642m), and Mt. McKinley (6,194m) without any severe altitude related problems. His health certificate at sea level examined just before his climb to Mt. Aconcagua reported that he had no health problems except in ECG, which showed sinus bradycardia (44/min), left ventricular hypertrophy and ST depression on II, aVf and V6 leads. Further examination such as loaded ECG test was recommended but he did not accept this suggestion because he had been examined by these tests one year before which revealed no abnormal findings.

On January 10th, 1999, following the two nights rest at base camp (4,250m) of Mt. Aconcagua and previous acclimatization climb, he climbed to the 2nd camp which was located at 5,800m. His SpO2 was 88% and HR was 55/min and he seemed to be in very good condition the next morning. The party then proceeded further up at 5:15 of the next morning (Jan 11th). He led the party at the top up to 6,600m, where he vomited. He claimed that the sports drink he took there made him feel sick. He began to get behind the party at around 6,800m, but, finally he managed to get to the summit of 6,962m at 16:30, 2 hours later than the first member's summit. At 17:05 I met the subject climbing down from the top. The subject told me that though a little bit tired he could return safely by himself by going slowly and steady. At 17:30, after reaching the summit, I caught up with the subject. He complained that he had bursting pain on his back bone. I tried to help and evacuate him with a local guide but he could not walk by 18:00, though his consciousness was so clear that he could talk. At 18:15, he became unconscious and while CPR was performed for 30 min he never recovered. I did not hear rales on his lung fields. Due to bad weather conditions, carrying down of his body was postponed until February 5th. The body, they said, had been buried in the snow. He was autopsied 25 days after of his death by the local police. According to the death certification issued by the local police, the cause for the death was "High Altitude Pulmonary Edema". The examiner who did autopsy reported to the family of the subject that the pulmonary edema induced myocardial infarction. He also mentioned that no evidences of dissection of an aortic aneurysm was found. I cannot believe that this autopsy report describes the actual cause of his death because he was so well that he climbed at the head of the party just below the top.

Case supplied by Kiyoshi FURUNO via Shigeru Masuyama, Department of Chest Medicine, Chiba University 1-8-1, Inohana Chuo-ku, Chiba 260-0856 Japan (e-mail:masuyama@med.m.chiba-u.ac.jp)

Gerald Dubowitz, US

This individual had ascended exceptionally fast. For a 67 year old performing so well, he was either an outstanding athlete or pushing himself too hard. The

consequences would seem to imply the latter. Peer pressure and even individual over-drive is a recognised contributor to acute mountain sickness (AMS), high altitude cerebral edema (HACE) and high altitude cerebral edema (HAPE).

This speed of ascent would easily make him prone to HAPE and an exhausting climb may well induce an MI or cardiac ischaemia in any individual with pre existing coronary problems.

I am not at all surprised that the autopsy showed HAPE as this fits the events well. Whether this was the primary cause of death is almost impossible to ascertain. There is no doubt that HAPE would, and almost certainly did, contribute to his death. It is equally possible that the HAPE lead to myocardial infarction (MI) or an MI plus cardiac hypoxia lead to the HAPE. Either way I think the cause was rapid ascent and physical overexertion, but to be sure of what came first is pure conjecture.

Slow ascents do not definitely rule out problems occurring but they certainly improve your odds (and the reverse applies to rapid ascents).

Bernard Marsigny, France
In my opinion, with our experience of patients with such symptoms occuring during the ascent of Mont-Blanc or the descent of the Vallee Blanche, it was certainly a cardiac infarct.

Charlie Houston, US
The first reaction is a dissecting aneurysm with acute pulmonary edema. If the autopsy is correct, this would be ruled out. More probable is acute myocardial infarction with subsequent and/or terminal pulmonary edema. I do not believe this is HAPE.

Zubieta Jr, Bolivia
Although the autopsy report is not specific, it sounds like multiple myocardial infarction, in a previously diseased heart and pushed to the limit. Furthermore, "silent myocardial infarction" at high altitude is well known. The pulmonary edema, is probably secondary to the myocardial infarction, no doubt aggravated by high altitude hypoxia.

The WILL to reach the summit is fundamental, but note that: W (work) + ILL is not equal to WILL, it is equal to IMPRUDENCE, which in this case has a fatal outcome. Whenever there is a medical suspicion that something is just not right (like bradycardia, left ventricular hypertrophy and S-Tdepression in this case), patients should be alerted and advised to postpone climbing until improvement.

Unfortunately, patient stubbornness and/or ignorance leads to life loss.

Jim Milledge, UK
This is a puzzling case. My first thought when I read about the "Bursting pain in the back" was of a dissection of the aorta but the post-mortem examination seems to rule this out. The cause of death was given, apparently, as HAPE which caused myocardial infarction. I have not heard of this as a complication of HAPE and Dr Masuyama's account does not fit with HAPE. His previous ECG suggests some

myocardial ischaemia presumably due to coronary artery disease and if an MI was found at PM I would have thought this was the likely cause of his problem and death. The "bursting pain", though felt in the back, might have been due to angina from an extending infarct.

John Severinghaus, US
If the pathologists really did look for and not find a dissecting anneurysm, which would have been my first bet, I presume he had a posterior wall MI. It does not sound like HAPE.

Ken Zafren, US
This is a very unfortunate case of sudden death at high altitude. The clinical clues include an episode of vomiting at the summit of Aconcagua, which is consistent with a myocardial infarction, but hardly diagnostic and the patient's complaint of "bursting pain in his back bone." I would be quite suspicious of a dissecting aortic aneurysm in spite of the pathology report. It's also possible he had a myocardial infarction. Aortic aneurysm can cause a secondary myocardial infarction. The pulmonary edema may also have been a secondary finding. The absence of crackles on physical examination might indicate that the edema was mild.

Michael Yaron, US
Sudden death secondary to cardiac arrest must be considered as the most likely cause in this case. it is unclear where in his back he experienced this "bursting back pain" but dissecting thoracic aortic aneurysm was excluded on the autopsy.

Atypical chest pain from a myocardial infarction would be my guess as the cause of the pain. This was also likely to be the cause of his nausea. No mention of coronary artery occlusion was made in the autopsy. The lack of rales on exam, the clear sensorium, and the sudden rapid decline in his condition that resulted in his death are all unlikely to be caused by HAPE.

Peter Hackett, US
The clinical description and the observations by an apparently reliable observer make it abundantly clear that this man did not die of HAPE. There may have indeed been some degree of pulmonary edema on autopsy because of the postmortem CPR. For a clear-cut diagnosis of antemortem pulmonary edema sufficient to cause death, however, the lung weights would have to be at least twice normal, more likely in the range of 1000 grams. Lung weights probably were not mentioned. Autopsy reports from agencies such as that mentioned are highly suspect. To local authorities, such deaths are a nuisance requiring time and paperwork, and shortcuts are common. I recently assisted with an autopsy of a man brought back from Kilimanjaro. The autopsy report from the authorirties in Tanzania clearly described swelling of the brain. On our repeat autopsy, we discovered that the cranium had never been opened! The clinical story suggests a diagnosis such as ruptured aortic aneurysm as much more likely; I would ignore the autopsy report and try to do a repeat autopsy if possible.

Brownie Schoene, US
Hard to tell but have to rely on clinical history, ie spine pain, still curious about

dissecting aortic aneurysm, can't rule out MI although in light of great previous health it is not likely. Pulmonary edema finding on xray would be hard to diagnose as HAPE in light of the fact that anyone who dies with cardio-vascular collapse will have fluid-filled lungs.

Simon Gibbs, UK
This subject had electrocardiographic evidence of left ventricular hypertrophy with some non-specific ST changes before the trip. He made a rapid ascent to almost 7000 m, complained of interscapular pain and died. The post mortem report comments on pulmonary oedema and there is informal mention of myocardial infarction. Aortic dissection was apparently excluded at post mortem.

I think the most likely differential diagnosis is myocardial infarction with either acute heart failure and / or preceding high altitude pulmonary oedema; or left ventricular hypertrophy associated with an arrhythmia causing acute heart failure. Pulmonary embolism is a further possibility. HAPE itself would not cause myocardial infarction but could precipitate it in somebody with ischaemic heart disease.

I would like to know what the pre-departure ECG evidence for left ventricular hypertrophy actually was since the ST changes described are not consistent with left ventricular hypertrophy: were these in fact ischaemic changes masquerading as left ventricular hypertrophy? Had he ever had echocardiography to assess the hypertrophy? The cause of the left ventricular hypertrophy may be important: we are not told about hypertension in the past although this is the most likely cause, but hypertrophic cardiomyopathy or aortic stenosis are also possible. He is in an age group at risk from coronary disease and left ventricular hypertrophy if caused by hypertension would increase this risk. The timing of the event coincided with maximum sympathetic activation. In summary this sounds like cardiac death associated with underlying heart disease and precipitated by exertion without full acclimatisation and hypoxia.

Oswald Oelz, Switzerland
This subject on Aconcagua was a fit but 67 years old male. I am surprised about his heart rate at an altitude of 4250 m which was 55/min at a saturation of 88 %. Even considering his excellent physical condition this heart rate is too low suggesting some cardiac pathology. Subsequently after summiting the subject developed back pain and died. Considering the autopsy report I have no doubt that this young old man suffered a myocardial infarction which was complicated by left ventricular failure.

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