JANUARY CASE DISCUSSION

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Case

A 47 year old mountain guide seeks advice. He has been climbing at very high altitudes since 1977 with numerous ascents over 5000m, a number of trips above 7000m on expeditions and 2 ascents over 8000m including an ascent of Everest. On an early trip he came across a research expedition and was informed that his haematocrit was very high but he was asymptomatic. 4 years ago, on a trip on the North ridge of Everest he developed numbness in his right hand at 6500m. By 7000m the numbness was worse and associated with nausea and vomiting. He descended immediately and in Kathmandu was found to have reduced sensation over his abdomen, groin and right arm. Haematocrit was 65%. He returned to his home in Europe but developed a headache over the right side of his head, slight dizziness and was noted to stagger to the right when walking. He was admitted to hospital 5 days after onset of symptoms by which time all symptoms were resolving. He was noted, on detailed neurological examination, to have decreased sensation over the right side of the anterior abdominal wall and tips of his fingers on the right hand. All investigations were normal including a CT brain scan. He made a complete recovery. Since then he has not climbed at very high altitude.

He has now been offered a place as a guide on a trip to Cho Oyu (8200m). What would you advise? What is the aetiology of high altitude neurological syndromes like this. How should someone who develops this syndrome be treated when they develop symptoms at 7000m? What is the risk of recurrence? How common are high altitude stroke-like events?

(permission has been obtained from the guide for the history to be published)

Lorna G Moore, USA
My advice is to reconsider what one's goals are here. This person is clearly at risk; another attempt at very high ascents is not advisable. Unless their goal is self-annihilation, I suggest a reorientation toward ascending equally challenging but lower height mountains elsewhere in the world.

James Milledge, UK
This climber clearly suffered a cerebro-vascular incident on Everest 4 years ago. The only such case which I have had first-hand experience was 37 years ago. At that time this condition was almost unknown but recently, with greatly increased numbers of climbers going to extreme altitudes, it is being seen much more often. There is a need to collect such cases to look for common factors in the aetiology and to study the prognosis. First we need a name for the condition and I would suggest, "Cerebro-Vascular Incident of Extreme Altitude" - CVIEA. I suggest "incident" rather than "accident" since in the lay mind accident might be misleading.

Cases typically occur well into the expedition, often on re-ascent to extreme altitude so seem not to be an aspect of AMS. The neurological manifestations are varied but

typically can be assigned to a cerebro-vascular field. The time course of symptoms and signs are longer than seen in transient ischaemic attacks but shorter than strokes seen in hospital practice, perhaps because the patients are young. The time course is similar to the attacks we used to see in patients with mitral valve disease with left atrial thrombi. I think it likely that the aetiology is thrombotic in many cases. Recovery is usually complete over a period of one to 5 days. This case is typical except that possibly the symptoms lasted longer than usual.

As regards prognosis; I believe we simply do not know what is the risk of recurrence since most victims do not expose themselves to extreme altitude after such a frightening incident. Until we know to the contrary, caution must dictate that we advise against re-ascent to extreme altitude. In this case there is the added suggestion that this subject may tend to an unusually high haematocrit at altitude which may well be a factor in the
aetiology of the condition. So, regretfully, I would advise him pass up this invitation to Cho Oyu and stick to the many wonderful peaks below say 6500 m.

Shigeru Masuyama, Japan
High altitude related stroke-like events especially among middle to old mountain climbers are not rare. My cases;
A 60 year old Japanese mountain climber lost his right eyesight at 8600m of Mt.Everest, while his eyesight recovered after he came back to South-Col. His fundus examination at Base Camp (BC) and brain CT examination in Japan was negative. He confessed that he had experienced syncope twice during trekking to BC.

A 55 year old female developed right hemiparesis at 4500m during trekking in Tibet. I sent her back to Japan 48 hours after the onset, where no abnormal CT findings were found on her brain and she made a full recovery.
These cases tell us that altitude stroke-like events or TIA are possible without positive CT findings. In these cases as well as the case above, CT examination alone might not be appropriated to investigate lacunar and small infarctions. MRI studies with adequate contrast medium would be appreciated in case of lacunar infarctions and a PET study would be recommended in case of ischemic brain damage.

How to treat at altitude? Rest, descent and evacuation. It is difficult to distinguish non-organic stroke-like events from an early phase of infarction or hemorrhage in the field. A 55 year old expedition doctor was found to be dead in his tent at Mt. Everest BC. Pathological autopsy in Kathmandu showed that he died of intracerebral hemorrhage with pathological evidences of HACE and HAPE.

Recurrency? No idea. The above 60year old male has continued high altitude mountain climbing without problems after the accident. Prophylaxis may be preventing dehydration and reducing blood viscosity by drinking sufficient fluid.

Tom Hornbein, USA
I know of no data on stroke-like events (they seem often more akin to that than TIA's in duration often), but anecdotally there's an accumulation, including some fairly notorious/famous mountaineers, and not so old. I've known one who heeded no

advice and returned to altitude, with no new damage to report (maybe the first hit took out his judgement center). It's an interesting phenomenon.

As a paying client, this individual would not make me happy as a companion at high altitude, at least to the extent that my own destiny might be influenced by his well being. Cannot imagine a commercial airline crew composed of such a questionable derelict. Now, what such an individual does in his own time and pleasure is another matter.

Of course some of the responsibility must rest with those who would employ the guide.

John Severinghaus, USA
This man may have either polycythemia vera or some serious oxygen desaturation at sea level. The first job is a hematology workup to rule out vera. If he spends enough time at high altitude to have chronic mountain sickness he may also have lost most of his carotid chemosensitivity. That much polycythemia would be unusual in someone who presumably spends some of his time at low altitude. It would be useful to test whether he lacks ventilatory response to acute hypoxia at sea level.

If he lacks hypoxic ventilatory drive and has sleep apnea or COPD or both it could help explain his high haematocrit (Hct). It would perhaps be useful to wear a pulse oximeter with inbuilt data recorder during several nights of sleep at low altitude, to determine whether he does desaturate significantly at night. This study could also be done at high altitude using a battery operated pulse oximeter with memory.

His history suggests cerebral thrombosis rather than a bleed with HACE. If his Hct remains that high, clotting could easily recur. If he is determined to go high again, he should consider being bled down to about 50% well before ascent allowing time to replete plasma volume, and also consider daily aspirin. If I were his physician I would advise against further long terms at high altitude. Hard to get a CAT scan and TPA there. He should not consider any thrombolytic therapy at altitude without a scan considering the evidence that petechial bleeding may underlie HACE.

Ken Zafren, USA
The easiest (and possibly best) course would be to tell this guide to stay below 6000 meters. It would be interesting to know whether his hematocrit is high at his European home. If he had developed these symptoms at
moderate or low altitude in the USA, he would probably have had an MRI, duplex ultrasound of his carotid arteries and echocardiography, which might shed some light on subtle long-term changes in the brain and on the source of a presumed thrombus.

I don't think anyone knows the etiology of high altitude neurological syndromes, although in this case, haemoconcentration is an obvious suspect for increased likelihood of thrombus formation. At 7000 meters (or any high altitude), I would treat stroke-like syndromes with immediate descent, oxygen if available, and aspirin. Volume replacement should also be a consideration, especially if one suspects a high haematocrit or volume depletion. The risk of recurrence is unknown as is the incidence of high-altitude stroke-like events. There are certainly many cases

reported. I suspect that most patients do not reascend to extreme altitudes. If the guide decides to reascend, I would recommend daily aspirin (325 mg) avoiding dehydration and monitoring of haematocrit with consideration of haemodilution.

Buddha Basnyat, Nepal
The aetiology in this case could be the dehydration leading to haemoconcentration (HCT 65) and stroke like syndrome. Other possible causes are focal edema leading to vasoconstriction, emboli, thrombus and HACE, although HACE usually presents with global problems and not focal. However given the fact that the patient at high altitude had nausea and vomiting and stroke like symptoms, HACE is certainly in the running and right at the top I would say. The treatment is descent descent and descent. Oxygen will be helpful if available and I would also use dexamethasone.

We have seen a few patients OUTSIDE the setting of AMS at high altitude with focal neurological signs (eg sixth nerve palsy) and for these patients if oxygen is unavailable I would consider rebreathing into a paper bag to raise the PCO_2 and allow more blood flow and oxygen into the brain. Descent would also be instituted at the same time.

The incidence of stroke like events at around 4500m and higher is by a rough estimate around 0.2% based on HRA findings in the Himalayas during the trekking season. I do not know the rate of recurrence although I have known people to do well on subsequent exposure to high altitude. My advice would be: How badly does he want to go up again? If he really wants to go up to high altitude again despite possible risks (this is an unknown area) then I would make sure that lots of oxygen is available and to drink lots of fluids to avoid dehydration, not to go higher than the base camp for starters and to make sure the team doctor and other members know about the past history and the benefits of oxygen and descent in this case. I would certainly re check the HCT again.

Bruno Durrer, Switzerland
With this history of an increased hematocrit of 65, I would recommend regular Hct tests up to the base camp and then decide whether he should continue or not. I had a member of a Cho Oyu expedition in 1994 with a repeated Hct of 65 at base camp. I advised him to drink a lot and not to go higher. Finally he confessed having taken Epo (erythropoeitin) every day, according to the advice of an old doctor back home. He was informed that a very high hematocrit would be helpful to reach the summit….. After stoping the Epo the Hct decreased.

Robert Schoene, USA
Does his Hct return to normal when he comes down? Although he certainly has an obvious cause for his polycythemia, could he have some hyperproliferative disorder? His response just sounds unusually excessive for his brief forays to high altitude. What does the rest of his CBC show? Normal cell morphology and differential on smear? Results of a bone marrow? Coagulation studies.

I think until you have a better feel for his hematologic picture, you can't let him go. Even if it is all normal at sea level, there is just something that is awfully unusual, perhaps acquired over-response from repeated exposures to high altitude. If he does

go, he certainly should monitor Hct.

David Murdoch, New Zealand
Focal neurological deficits of many types have been observed following high altitude exposure. Although several have had strong supporting evidence of thromboembolic phenomena, I suspect many have a migrainous aetiology. Incidence and recurrence data concerning these events are lacking, and very few cases are even reported in the medical literature. Descent should be part of all treatment regimens.

In the case presented, the cause of the neurological deficits is unclear. The persistence of symptoms and signs for a relatively prolonged time after returning to low altitude is a little concerning. Such persistence has been previously described in the context of neurological deficits at high altitude due to both thromboembolism and migraine. I presume his haematocrit returned to normal on descent; if not, this should be investigated further. Otherwise, I would present the current understanding of such events to the guide, in particular the uncertainties concerning aetiology and risk of recurrence in someone who appears in good health. If he decides to go to Cho Oyu, I would caution him to observe carefully for the development of warning symptoms (not just the neurological symptoms he experienced previously) and advise him to descend if they occur.

John English, UK
He risks a serious CVA and should not go. Could try two aspirin and keep his fingers crossed!

Gustavo Zubieta, Bolivia
Increased hematocrit has always been said to be associated with stroke. We have our doubts. In the city of La Paz (3100-4100 m) there is no increased incidence of stroke over 1,000,000 inhabitants with respect to sea level. Stroke is usually associated fundamentally to cardiac arrhythmias, mitral valve anomalies, atherosclerotic disease, and arterial hypertension and these should be ruled out. On the other hand, in many years of practice and seeing thousands of patients with increased hematocrit, we have only seen 2 cases of stroke, similar to the one described in this case, but both with complete recovery.

Strokes in people with high hematocrit seem to recover very well. Our advice, is that the mountain guide have a complete medical check-up, be in good physical condition and to start packing.

Peter Bärtsch, Germany
It appears that this man had an ischemic stroke with an incomplete sensory hemisyndrome on Everest and most likely a second event (cerebellar?) thereafter at home. The second event is rather surprising and may point to a cause that is not necessarily related to high altitude. Nevertheless, he recovered completely and had no further events for the last four years. As he was hospitalized, we can assume that his hematocrit at low altitude is normal. It would be interesting to know the circumstances under which he developed the stroke. Was there extreme dehydration, how much time had he spent at extreme altitude? Under which circumstances was the earlier hematocrit measurement performed?

Before he goes to these altitudes again, one should exclude abnormal cerebral vessels by Doppler-ultrasound and cardiac abnormalities associated with embolism. When such predisposing factors are excluded, I do not see any reasons to prevent him from going to Cho Oyu, given that he has done numerous ascents to these altitudes before, especially when one could establish insufficient hydration as a predisposing cause. I would stress that he should pay attention to an adequate hydration and follow the golden rules for avoid HAPE and HACE. I would explain to him the risks and benefits of taking low-dose aspirin (100 mg). Personally, I would not want to take this drug regularly when going to these altitudes.

Examinations on blood coagulation up to altitudes of 4500 m at rest and submaximum exercise (for review see Bartsch: Hypoxia and Molecular Medicine, JR Sutton, CS Houston and G Coates, eds, p 252 - 258, 1993) suggest no increased in vivo thrombin- and fibrin generation while after one hour of maximal running in normoxia (Weiss, Med Sci Sports Exerc 30: 246-251, 1998) there is a slight increase of markers of in vivo thrombin- and fibrin generation. However, these changes are accompanied by an activation of fibrinolysis. There are, to my knowledge, no data on the effects of a combination of exhaustive exercise and hypoxia or on the effect of very high altitude on homeostasis. The major risk factor for thromboembolic events at very high altitude is most likely dehydration. The fact that reports of such events are relatively rare and the fact that in healthy individuals activation of blood coagulation is always accompanied by activation of fibrinolysis suggests to me that exercise and very high altitude do most likely not severely increase the risk for thromboembolism. We should be careful with inhibition of coagulation in such a setting in which there is a high prevalence of retinal hemorrhages (over 50% at 6000 m) and in which there is an increased risk for head injury.

David Hillebrandt, UK
I will not comment on this case since I gave the patient the facts as I saw them and he is going back to Patagonia on a non commercial trip with me in three weeks time instead. He is a sensible guy (apart from his illogical desire to return to the wet and wind in Chile) and decided that the risk was not warranted in going to very high altitude again but I know that he will value the full comments for future trips.

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