

A Cardiac and a Kidney Transplant Patient Above 6000 Meters in Bolivia

To the Editor:

Over the past 2 decades, survival and quality of life following cardiac transplantation have markedly improved, mainly as a result of advances in immunosuppressive drug regimens.¹ However, the survival rate following heart transplantation remains quite limited and is now approaching 50% at 15 years. The decrease in life expectancy in this population is largely related to associated co-morbidity, such as cardiac-allograft vasculopathy, accelerated atherosclerosis, renal failure, hypertension, and diabetes.¹ As is the case for patients following cardiac transplantation, patients experiencing kidney transplantation are at high risk of cardiovascular events.² The etiology for accelerated atherosclerosis remains largely unexplained, but subclinical inflammation may play a significant role.³ Hypobaric hypoxemia is a potent stimulus for the release of erythropoietin and for the increase in some markers of angiogenesis, such as vascular endothelial growth factor.⁴ Similarly, prostacyclin is a member of the prostaglandin family of lipid mediators, which have potent vasodilator and antithrombotic activities⁵ that increase in response to vascular stress.

Some mountains have been successfully summited by organ transplant recipients. Here we report the experience of 2 climbers, one a recipient of a cardiac transplant and the other of a kidney transplant, on their ascent to Mount Sajama, the highest summit in Bolivia (6522 m). We also explored the changes in selected biomarkers in these climbers before and after the expedition.

Two patients participated in this expedition. The cardiac transplant recipient was aged 35 years and the kidney transplant subject was 41 years. The cardiac transplant recipient received orthotopic heart transplantation at the age of 32 in the summer of 2000. The kidney transplant patient received a kidney from his brother in 1996 at the age of 33 years. Both transplant patients were treated with immunosuppressive agents consisting of tacrolimus, mycophenolate mofetil, or azathioprine. There was no evidence of acute or chronic rejection within 12 months of participation in this expedition. Neither of the climbers experienced any chronic illness prior to departure for the expedition, and both had normal biochemistry profiles. Selected markers of angiogenesis, prostacyclin, and some markers for subclinical inflammation and oxidative stress were measured prior to departure for the Bolivian Altiplano and within 24 hours of returning to sea level. The biomarkers included vascular endothelial growth factor, angiopoietin-2, stable metabolite of prostacyclin (6-keto PGF_{1 α}), interleukin-6,

and thiobarbituric acid-reactive substances. Observations from the transplant recipients were compared with those of 5 healthy climbers aged 42.8 ± 13.2 years. The cardiac transplant patient exhibited a peak oxygen uptake of $48.8 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, while the kidney transplant recipient yielded a peak Vo_2 of $55.4 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ prior to departure for the expedition.

Both cardiac and kidney transplant patients exhibited similar oxygen saturation compared with the rest of climbers who successfully summited Sajama upon arrival in La Paz (cardiac transplant [CTx] = 85%; kidney transplant [KTx] = 84%; Group = 84%). Similarly, saturations were similar at Sajama base camp at an altitude of 4800 m (CTx = 90%; KTx = 82%; Group = 80%).

The cardiac transplant recipient reached an altitude of 6120 m on the foothills of Mount Sajama. He presented with overall exhaustion. His O₂ saturation measured before turning back to base camp was 69%. The kidney transplant recipient reached the summit at an altitude of 6522 m. Saturations were not measured on summit.

Both cardiac and kidney transplant subjects exhibited no significant changes in any of the markers of angiogenesis upon return to sea level. Similarly, there were no changes in plasma interleukin-6 and thiobarbituric acid-reactive substances before or upon return from the expedition.

This expedition was significant in the field of organ transplantation as it highlights the fact that organ transplant is not a contraindication for high-altitude mountain climbing. This is evidenced by the fact that both patients achieved an altitude over 6000 m. Both transplant patients tolerated the high altitude very well and exhibited similar changes in hematology and biochemistry profiles.

Cardiac and other organ transplant recipients have successfully summited high mountains within the last few years. These very special patients exhibited no obvious limitations. Here we report similar biochemistry profiles and adaptations to high altitude compared with other climbers who adapted well to high altitude (3 of the 5 healthy climbers).

The cardiac transplant patient climbed to an altitude of 6120 m and stopped because of overall exhaustion. He presented with no significant cardiovascular or overall health problems in response to high altitude. The low saturation achieved at maximal altitude reached most likely represents a normal response to exercise at such a high altitude.⁶ In addition, neither of the organ transplant recipients presented with infection or any signs of right heart failure.

We found similar levels of selected markers of angiogenesis, subclinical inflammation, and oxidative stress in organ transplant recipients compared with the healthy climbers who summited Sajama. In addition, there were

no significant differences between transplant recipients and healthy climbers.⁷ Normalization of levels of the many plasma markers is not surprising, since measurements were performed 24 hours after a rapid descent to sea level. This likely indicates rapid regulation of the expression of these mediators in response to return to normoxic conditions. These preliminary observations indicate that organ transplant recipients tolerate altitude well and may safely achieve high-altitude challenges. Further investigations are needed to better study the changes in these markers during the process of acclimatization to high altitude in healthy climbers but also in a larger cohort of organ transplant recipients.

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Vipera berus Bite in a Child, With Severe Local Symptoms and Hypotension

To the Editor:

Snakebites are very rare incidents in central parts of Europe (Austria, Germany, and Switzerland), and mortality has been reported to be 0% for adults.¹ The experience of medical personnel with treating snakebites may not be substantial, which at times may lead to an unnecessary time delay in treatment or difficulties in decision making, especially where children are concerned. Viper venoms contain more than 100 components and may cause local symptoms and signs, such as pain and swelling, but also systemic findings, such as gastrointestinal and circulatory disturbances and coagulation disorders. Children and elderly people are more likely to develop severe symptoms.²

We would like to report a 12-year-old boy who was bitten on his right index finger by an adder (*Vipera berus*) in the outskirts of Innsbruck. Almost immediately after the bite, the patient developed signs of shock and complained of gastrointestinal pain and difficulty breathing. Upon arrival of the emergency medical system personnel 10 minutes after the bite, the patient was somnolent, hypotensive (blood pressure: 75/55 mm Hg), and tachycardic (heart rate: 145 beats/min). He had tachypnea with inspiratory stridor and a peripheral oxygen saturation (SpO₂) of 90%. The patient was immediately supported with oxygen and rapid infusion of 1000 mL of a 0.9% NaCl solution. He was given 0.1 mg epi-

Table. Laboratory parameters on admission in a 12-year-old boy bitten by *Vipera berus*. All values are within normal limits.

Parameter	Value	Unit
Partial thromboplastin time	31	seconds
Prothrombin time	96	seconds
Fibrinogen	260	mg·dL ⁻¹
Antithrombin 3	82	%
Hemoglobin	11.5	g·dL ⁻¹
Hematocrit	32	%
Creatinine	0.81	mg·dL ⁻¹
Alanine aminotransferase (GPT)	18	U·L ⁻¹
Aspartate aminotransferase (GOT)	34	U·L ⁻¹
Gamma-glutamyl transferase	13	U·L ⁻¹