

Letters to the Editor

Possible Indirect Adverse Effects to the Eyes in Skiers Wearing Helmets



To the Editor:

Use of helmets among skiers started to increase some 20 y ago, becoming mandatory for children under 16 y old in Austria in 2009.¹ Today, 86% of skiers wear helmets.² Helmet use has reduced the risk of head injuries by 35%.³ Widespread use of helmets implies thorough study of the possible adverse effects of wearing helmets that could offset their protective effects. Helmet use does not increase the risk of neck or cervical spine injury or the risk of compensation behavior.⁴ However, the 2 groups of head injuries, traumatic brain injury and other types of head injuries, are not equally prevented by ski helmets, and the number of traumatic brain injuries is not reduced to the same extent as the number of other types of head injuries.⁵ Over time, the protective effects of helmets have been somewhat reduced.⁶ A direct influence of ski helmets on hearing was investigated and established.⁷

In addition to helmets, usual head gear for skiers includes ski goggles. Their role is to prevent ultraviolet (UV) radiation (wavelengths smaller than 400 nm) from reaching the eye and the surrounding soft tissue; to reduce the total amount of light reaching the eye; to reduce the production of tears; to enhance contrast; and to provide mechanical protection of the eyes and the surrounding soft tissue. Optimal performance depends on ventilation of humid air. Proper ventilation prevents fogging and should not induce the overproduction of tears. Helmets tend to reduce goggle ventilation; therefore, goggles fog much more easily, and when skiers are moving at reduced speed or when they stop moving, fogging becomes almost inevitable. This is probably one of the principal reasons why many helmeted skiers remove their goggles when they stop moving; when using chair lifts, drag lifts, and gondolas; and sometimes during descent.

There are, however, skiers who use an extra pair of sunglasses in situations when goggles are removed, and a certain number who use sunglasses exclusively. All the aforementioned personal observations were confirmed by the inspection of photos from 12 large ski resorts in Austria, Italy, France, and Bulgaria. The largest proportion of skiers, more than 50%, remove their goggles at the base stations of lifts and approximately 30% when using chairlifts, whereas skiers in descent rarely remove them. At the top stations of ski lifts, the percentage of skiers

with removed goggles is nearly the same as among chairlift users. Approximately 10% of skiers were wearing sunglasses and helmets (skiers with removed goggles included). There were no skiers with removed sunglasses. Goggles and sunglasses do not protect the eyes and the surrounding tissue to the same extent because goggles cover a larger area and have a tighter fit.

The effects on the eyes of removing goggles and not replacing them with sunglasses is that they are exposed to increased levels of UV radiation in situations when the UV index is usually very high. As a result, adverse effects of UV radiation are possible. The most notable adverse effects include snow blindness as an acute effect; cataract and macular degeneration are chronic effects.⁸ Owing to increased sensitivity to UV radiation among children, the removal of ski goggles could exacerbate such effects.⁹

Although eye injuries are just a fraction of head injuries, in the only identified study covering skiing, one-third of eye injuries were acute injuries caused by UV radiation.¹⁰ There are no data on the long-term effects of UV radiation to the eyes in skiing.

The almost uniform habit of wearing ski helmets amplifies the importance of addressing possible adverse effects of UV radiation to the eyes of helmeted skiers who remove their goggles while skiing. The proportions of skiers with removed goggles in different situations suggest the possibility that many skiers remove their goggles after descent and put them on just before descent. Information about the high intensity of UV radiation at high altitudes and snow-covered slopes, as well as the need for permanent eye protection, should be promoted. Because chronic effects to the eyes emerge after many years and the effects are not immediately recognized as acute, it is important that skiers be aware of the risk.

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Toxic Brain Edema and Brain Death After Scorpion Envenomation



To the Editor:

Scorpion sting is common in some global regions and mostly causes local envenoming of varying severity. Some estimates suggest that perhaps only 10% of those stung by even the most medically important species develop severe systemic envenomation; however, the frequency of severe envenomation is higher in children. With few exceptions, systemic envenomation by medically important species is characterized by relatively similar neurotoxic stimulation syndromes.¹

Here, we report a 12-y-old girl who developed toxic encephalopathy and lethal brain edema owing to scorpion envenomation (*Leiurus abduhbayrami* [Scorpiones: Buthidae]). She was transferred to our pediatric intensive care unit from another center after 2 d of follow-up. Based on the documents provided by the other medical unit, the patient was intubated 1 to 2 h after the scorpion sting owing to confusion and respiratory distress and was given tetanus vaccine, 1 vial of antivenom (polyvalent antiscorpion antivenom, Refik

Saydam Hygiene Center, Ankara, Turkey), and doxazosin therapy in the initial treating facility. Because the patient experienced cardiac arrest for 5 min, cardiopulmonary resuscitation was performed. When the ejection fraction of the left ventricle was 30% on echocardiography, she was referred to us with a diagnosis of toxic myocarditis.

Immediately after admission to our unit, she was intubated and had decompensated shock findings. Her Glasgow Coma Scale score was 6 of 15. Blood gas findings showed mild acidosis. Laboratory results showed white blood cell levels were $15.73 \times 10^3 \cdot \mu\text{L}^{-1}$, hemoglobin was $11.9 \text{ g} \cdot \text{dL}^{-1}$, platelets was $254 \times 10^3 \cdot \mu\text{L}^{-1}$, serum creatinine was $0.87 \text{ mg} \cdot \text{dL}^{-1}$, blood urea nitrogen was $24 \text{ mg} \cdot \text{dL}^{-1}$, aspartate aminotransferase was $244 \text{ U} \cdot \text{L}^{-1}$, alanine aminotransferase was $71 \text{ U} \cdot \text{L}^{-1}$, creatinine kinase was $8191 \text{ U} \cdot \text{L}^{-1}$, troponin T was $1924 \text{ pg} \cdot \text{mL}^{-1}$ (normal range: 0–14), N-terminal pro-brain natriuretic peptide was $>35 \text{ ng} \cdot \text{L}^{-1}$, and CK-MB was $75 \text{ ng} \cdot \text{mL}^{-1}$ (normal range: 0–3.6); international normalized ratio was 1.59; activated thromboplastin time was 24.9 s; prothrombin time (PT) was 18.4 s; D-dimer was $3789 \text{ ng} \cdot \text{mL}^{-1}$ (normal range: 0–243); and fibrinogen was $5.58 \text{ g} \cdot \text{L}^{-1}$ (normal range: 2–3.93).

To stabilize the patient, we continued with maintenance fluid, milrinone, and doxazosin therapies and dobutamin to correct hypotension. On the first day of admission, the patient was given 1 more vial of Refik Saydam Hygiene Center polyvalent antiscorpion antivenom therapy. On echocardiography, the ejection fraction was 45 to 50%. Cranial computed tomography (CT) was performed on the third day of admission, with decreased alertness and a Glasgow Coma Scale score of 4. The CT report revealed existing widespread hypodense areas in both cerebral hemispheres, loss of gray and white matter separation, and deletion in sulcal structures compatible with edema (owing to possible toxicologic effects) (Figure 1). Because the patient had brain edema, $0.1 \text{ cm}^3 \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$ 3% NaCl was started. The patient's neurologic condition worsened gradually. On the 10th day of hospitalization, neurologic examination revealed absence of all brainstem activity. CT angiography was performed, and the report was compatible with brain death.

It has been reported that after scorpion sting, cortical disruption may develop before other organ disruptions.² One study with 9 fatal cases documented central nervous system (CNS) symptoms before terminal hypotension and cardiac arrest.³ Lethal cerebral edema occurred in a 2-y-old male patient who had diffuse brain edema and ischemic changes on CT after scorpion sting and was later reported to be diagnosed with brain death.⁴ Similarly, our patient had diffuse brain edema