CASE REPORT

Imaging Findings of a Survivor of Avalanche without Any Life Support at Very High Altitude and Extreme Low Temperatures

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Abstract:

Survival at high altitude is very challenging and in spite of adequate training and acclimatization, injuries are frequent. The fate of mountaineers and soldiers at such areas largely depends on the mercy of the climate. An avalanche causes physical trauma, cold injury and asphyxia to the victim. The patient in our report had diffuse cerebral edema, bilateral pulmonary consolidation and pneumothorax. In spite of the best efforts the victim succumbed to the injuries. There are many incidents of high altitude accidents in India. This case report is of a soldier deployed at the high altitude, is a lone ever reported survivor above 5000 meters, under 35 feet snow and below - 45°C for greater than 5 days of exposure to an avalanche

Keywords: High Altitude Cerebral Edema, Pneumothorax, Avalanche

Introduction:

There are many incidents of high altitude accidents in India. There are various types of injuries seen in the avalanche survivors including mechanical injuries, asphyxia, hypothermia, high altitude pulmonary edema, high altitude cerebral edema, and cardiac arrest and nervous system anomalies [1]. Only few of these findings can be radiologically demonstrable.

The pathophysiology behind cerebral edema is hypoxia mediated cerebral vasodilatation, disruption of blood brain barrier possibly due to release of neuromodulators Vascular Endothelial Growth Factors (VECF) and calcitonin gene related peptide [2, 3]. There is also generalized increase in the sympathetic activity due to high ADH (anti diuretic hormone) and aldosterone [2, 3]. The pathophysiology of pulmonary edema is due to blunted ventilator responses to hypoxia, exaggerated hypoxic pulmonary vasoconstriction and reduced production of pulmonary vasodilators 4, 5. The mechanical injuries include bony fractures, CNS and spinal cord injuries, foreign body, aspirations in respiratory tract, chest wall injuries, pulmonary contusions, asphyxia related effects and secondary infections in the underlying lesions [1].

Case Report:

The victim is a 33 year old individual with an occupational deployment with a team of 9 people at about 19600 feet on Himalayan glacier when he faced a massive avalanche. He was rescued on the sixth day after the disaster from 35 feet beneath the snow in -45°C temperature. The victim was found unconscious during the rescue. At the time of hospitalization he had hypotension, pulse fluctuation, hypoxia and hypothermia. Biochemical parameters were deranged including, SGOT, SGPT, bilirubin, serum urea (90mg/dl) and creatinine (7.5mg/dl) during the hospitalization.

At our centre was put on oxygen, thermal and ventilatory supports. He was given IV fluids, broad antibiotic cover and later given ionotropes due to deranged vital parameters. The treatment was primarily supportive and planned for later rehabilitation by various means. After 2 days of rigorous attempts of management he succumbed with the injuries due to multiple organ dysfunction syndrome. The patient did not suffered any fractures, skin changes, any external injuries or any evidence of frost bite [8] or external cold related injuries.

NCCT brain and chest were done as the biochemical parameters of the patient were deranged, immediately after the admission and reviewed by two radiologists, one with 4 years of experience and other with 3 years of experience. MRI brain study was cancelled because the patient was on ventilatory support and the general condition was not good.

The findings in the NCCT brain (Fig. 1) were generalized cerebral edema with loss of the grey white differentiation and effacement of the sulcal spaces. Both lateral ventricles appeared chinked with the edema (Fig. 2). Minimal scalp edema was also noted (Fig. 1). The skull bones were normal. Minimal mucosal thickening was noted in the left maxillary sinus (Fig. 4). The NCCT chest (Fig. 5 and 8) showed a large thick walled irregular cavity at both upper lobes with surrounding ground glass opacities on the right upper lobe. Tree in bud opacities, air bronchograms and nodular opacities (Fig.7 and 8) were also seen in both upper lobes. Left sided pneumothorax with subcutaneous emphysematous changes and passive atelectasis of the left upper lobe was noted (Fig.7 and 8). Bilateral moderate pleural effusion (Fig. 5 and 6) was also noted. A drainage tube is also seen in the left chest below the site of subcutaneous emphysema.

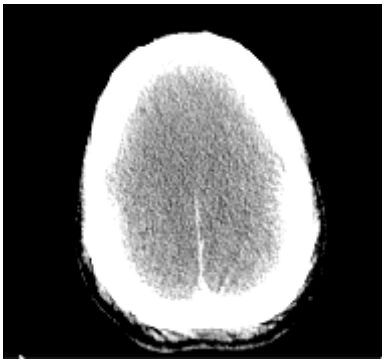

Fig. 1: NCCT Brain Axial Images Demonstrates Mild Scalp Edema at the Parietal Region with Loss of Grey White Differentiation with Diffuse Cerebral Edema

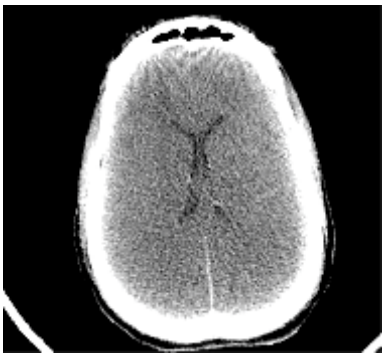

Fig. 2: NCCT Brain Axial Images show Loss of Grey White Differentiation with Diffuse Cerebral Edema and Chinking of the Both Lateral Ventricles

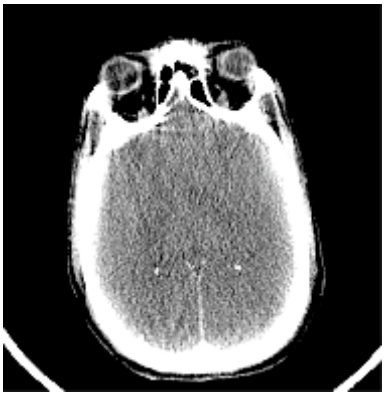

Fig. 3: NCCT Brain Axial Images Show Loss of Grey White Differentiation with Diffuse Cerebral Edema and Effaced Sulcal Spaces

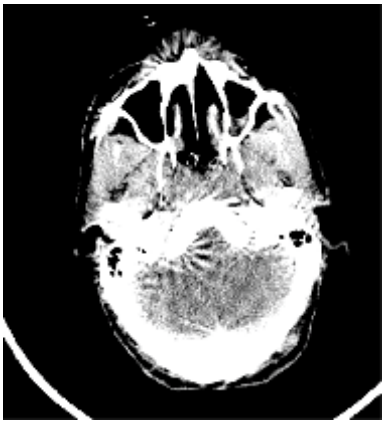

Fig. 4: NCCT Brain Axial Images Demonstrates Loss of Grey White Differentiation in the Posterior Fossa with Diffuse Edema Along With Minimal Soft Tissue Thickening in the Left Maxillary Sinus

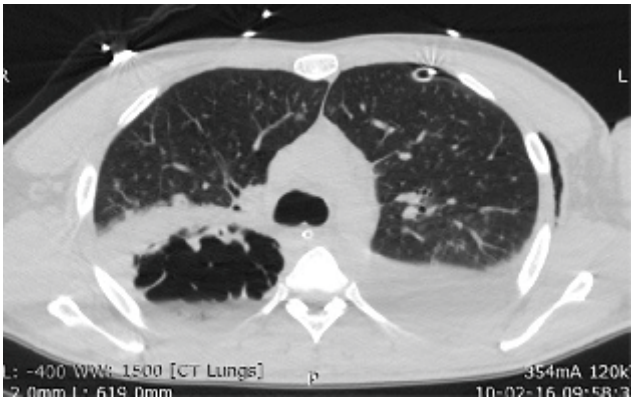

Fig. 6: NCCT Chest Axial Section in Lung Window shows a Large Irregular Walled Cavity Surrounded by Few Areas of Nodular Opacities. Minimal Subcutaneous Emphysematous Changes are also Seen in the Left Chest Wall. Minimal Pleural Effusion is also Seen on the Left Side.

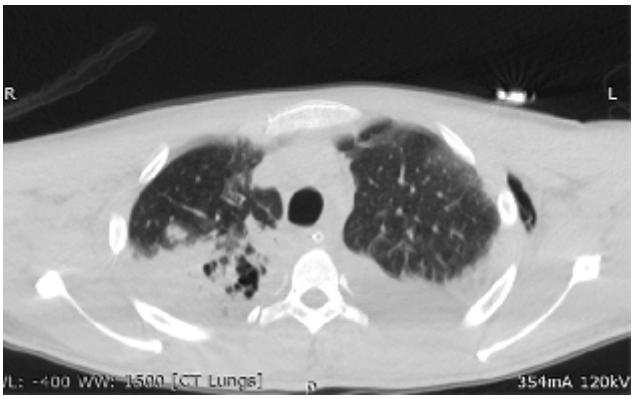

Fig. 5: NCCT Chest Axial Section in Lung Window shows Right Upper Lobe Air Bronchograms, Ground Glass Opacities and Nodular Opacities. Minimal Subcutaneous Emphysematous Changes are also Seen in the Left Chest Wall. Minimal Pleural Effusion Is also seen on the Left Side.

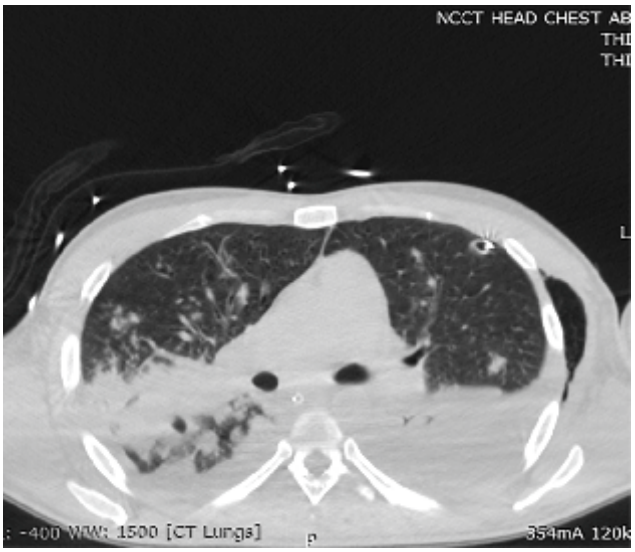

Fig. 7: NCCT Chest Axial Section in Lung Window shows Right Upper Lobe Air Bronchograms along with Tree in Bud, Ground Glass Opacities and Nodular Opacities. Left sided Subcutaneous Emphysematous Changes are also Seen. Left-sided Pleural Effusion and Passive Collapse of Left Upper Lobe is also Noted

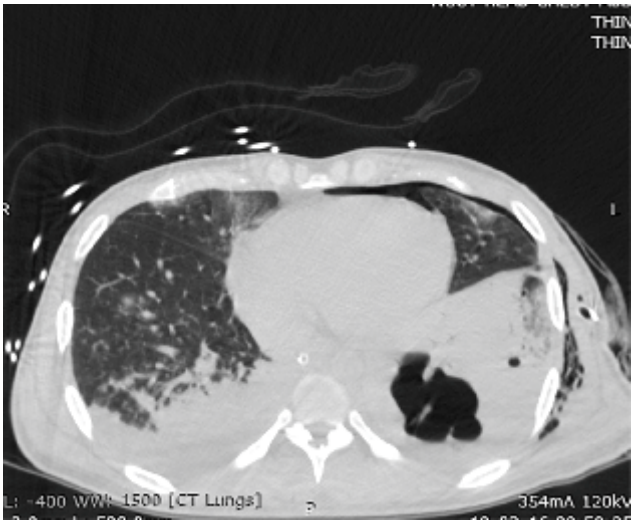

Fig. 8: NCCT Chest Axial Section in Lung Window shows A Large Cavity with Irregular Wall and Associated Left Sided Pneumothorax. Subcutaneous Emphysematous Changes and A Drainage Tube are also Seen on the Left Side. Left sided Passive Collapse of Left Upper Lobe is also Noted. On the Right Lung Multiple Ground Glass Opacities and Tree in Bud Opacities are also Noted.

Discussion:

The prognosis of avalanche survivors is very variable. Many patients have permanent neurological deficit. Victims can have crush injuries and proceed through amputation. Few patients show psychiatric symptoms [6]. A few cases are also found to have peripheral vascular disease and cerebral venous sinuses thrombosis. Survival of these victims also depends on the time of rescue and the conditions of the accident apart of the early diagnosis and management. A need for more sensitive whether alarm system, better search and rescue facilities are required for prevention of such casualities. This case is the rarest example of the human endurance against nature with the radiological findings which are never reported in any patient with a history of survival for more than five continuous days without any support in such an inhabitable environment. The radiological findings help in understanding the mechanism and management of similar cases in future.

The chest and pulmonary findings are more due to mechanical injuries, asphyxia, pulmonary edema and secondary infection in the lung due to extreme conditions. The cavities are an indicator of secondary infections. The tree in bud opacities and air bronchograms are also indicators of active infection in the individual after 5 days of starvation and fight of survival.

The lesions are primarily involving the upper lobes including the apices. The pneumothorax in left lung with subcutaneous emphysematous changes is either mechanical injury due to avalanche or due to iatrogenic procedure.

As compared to study by Schynder *et al.* (1999) [7], our case is partially following the above study with similarity of the patchy consolidation, apices involvement. The mechanical injuries as pneumothorax and subcutaneous emphysematous changes seen in our case are the radiological findings not described in the above study.

In this patient, the diffuse cerebral edema seen with loss of grey white differentiation seen diffusely in the brain is likely due to the release of neuromodulators and cerebral vasodilatation as a result of a response to the injury due to severe hypothermia and hypoxia related to the brain. The edema is so severe that it caused effacement of the sulcal spaces and the chinking of the both lateral and the third ventricles.

Robert *et al.* (2004) [9] study states that the CT images demonstrate attenuation of signal, either diffusely or in the white matter, with compression of sulci and flattening of gyri. The CT findings in our case appears shows resemblance to the CT findings of the above study; however their study is not having any description of the pulmonary CT changes with a difference in the patients history on comparison of cases.

A study of Morales H *et al.* (2006) [10] study states that in patients with high altitude exposure have frontal sub cortical lesions, cortical atrophy and enlargement of Virchow-Robin spaces. On comparison to this study our patient does not show such radiological features. The finding differ as the subjects taken in the above study are victims who had different exposure times on the high altitude areas

Conclusion:

The radiological features of the brain imaging in avalanche survivors are of two types 1st: mechanical injuries like scalp edema, fractures, contusion. However, in this case, the patient had only scalp edema likely due to the preparedness, protection of the professional suit and helmet provided to the individual. 2nd: Tissue related injuries due to hypoxia, asphyxia and cold related increased metabolic rate which if remains uncompensated leads to generalized tissue injury of brain with an increase of the blood-brain barrier in this case seen on NCCT brain scan in the form of loss of grey white differentiation, diffuse cerebral edema with diffuse involvement of the CSF spaces of brain.

The chest findings in this case are primarily due to mechanical injuries as pneumothorax and subcutaneous emphysematous changes leading to secondary change seen in the form of cavities, consolidation, ground glass opacities and tree in bud opacity on NCCT chest. The chest and brain NCCT are transcendental for the investigation of injuries in a critical patient due to the speed of the procedure and better soft tissue appreciation as compared to conventional and digital radiography. MRI is a time consuming procedure and merely for radiological findings. A critical patient with advanced life support and on thermal support cannot be exposed to MRI if enough information can be acquired from NCCT imaging.

References

1. Fieler, Julia. North-Norwegian avalanche victims: a retrospective observational study. Universitetet i Tromsø University of Tromsø 2013-06-03.
2. Jensen JB, Sperling B, Serveringhaus JW, Lassen NA. Augmented hypoxic cerebral vasodilatation in men during 5 days at 3810 M altitude. *J Appl Physiology* 1996; 80:1214–1218.
3. Levine BD, Zhang R, Roach RC. Dynamic cerebral autoregulation at high in: RC Roach, PD Wagner, PH Hackett (Eds.) Hypoxia: into the next millennium. Vol. 474 of Advances in experimental medicine and biology. Kluwer Academic/Plenum, New York; 1999: 319–22.
4. Maggiorini M, Melot C, Pierre, S et al. High altitude pulmonary edema is initially caused by an increase in capillary pressure. *Circulation* 2001; 103: 2078–83.
5. Elser H, Swenson E, Hildebrandt J. Regional distribution of pulmonary perfusion after five hours of normobaric hypoxia in subjects susceptible to high altitude pulmonary edema. *Eur Respir J* 1998; 12: A2328.
6. Flaherty G, O'Connor R, Johnston N. Altitude training for elite endurance athletes: A review for the travel medicine practitioner. *Travel Med Infect Dis* 2016; 14(3):200-11.
7. Gluecker T, Capasso P, Schnyder P, Gudinchet F, Schaller MD, Revelly JP *et al*. Clinical and radiologic features of pulmonary edema. *Radiographics* 1999; 19(6):1507-31; discussion 1532-3.
8. Harirchi I, Arvin A, Vash JH, Zafarmand V. Frostbite: incidence and predisposing factors in mountaineers. *Br J Sports Med* 2005; 39(12): 898–901.
9. Hackett PH, Robert C. Roach. High Altitude Medicine & Biology. 2004, 5(2): 136-146.
10. Fayed N, Modrego PJ, Morales H. Evidence of brain damage after high altitude climbing by means of MRI imaging. *Am J Med* 2006; 119(2):168.e1-6.

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