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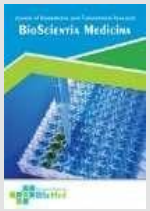

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Challenging the Paradigm: A Clinicopathological Case Series on the Discordance between Cutaneous Burn Severity and Airway Injury Following a Volcanic Pyroclastic Flow Exposure

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A B S T R A C T

Background: Inhalation injury accompanying severe burns is a major driver of morbidity and mortality. The pathophysiology of injury from volcanic eruptions—a unique combination of extreme heat, abrasive particulates, and corrosive gases—is distinct from typical combustion smoke and not well-characterized. Flexible bronchoscopy is the gold standard for diagnosing and grading airway injury, yet its application in this context, particularly in relation to external burn severity, is unclear. **Case presentation:** We present a retrospective analysis of two climbers who were the sole survivors admitted to our tertiary center after exposure to the pyroclastic flow from the 2023 Mount Marapi eruption. A 19-year-old female with 40% Total Body Surface Area (TBSA) deep burns and a 20-year-old male with 30% TBSA deep burns both presented with signs concerning for inhalation injury. Despite the devastating extent of their cutaneous injuries, flexible bronchoscopy in both patients revealed only mild, Grade 1 endobronchial injury (mucosal erythema and edema) based on the Abbreviated Injury Score (AIS) classification. The first patient succumbed to septic shock from her burns, while the second patient showed significant respiratory improvement following therapeutic bronchial lavage and made a full recovery. **Conclusion:** This case series presents hypothesis-generating evidence suggesting that in victims of open-air volcanic pyroclastic flows, the severity of cutaneous burns may be a poor predictor of the degree of underlying endobronchial damage. These findings underscore the potential necessity of a low threshold for early flexible bronchoscopy to accurately diagnose, stratify, and therapeutically intervene in this unique patient population, irrespective of TBSA.

1. Introduction

Burn injuries constitute a significant global public health challenge, with the World Health Organization reporting millions of cases and an estimated 180,000 deaths each year, with a disproportionate burden on low- and middle-income nations.[1] The presence of a concomitant inhalation injury is a critical and ominous comorbidity that profoundly worsens patient outcomes.[2] It dramatically increases fluid resuscitation requirements, escalates the incidence of pulmonary complications such as pneumonia and acute respiratory distress syndrome (ARDS), and is an independent predictor of mortality, with some studies attributing as many as 77% of deaths in burn patients to respiratory failure.[3] The prevalence of inhalation injury among hospitalized burn patients is estimated to be between 5% and 35%, with its likelihood

traditionally believed to increase in concert with the total body surface area (TBSA) of the burn.[4]

The clinical diagnosis of inhalation injury is notoriously complex due to a wide spectrum of presentations.[5] However, a constellation of signs, including facial burns, singed nasal vibrissae, hoarseness, carbonaceous sputum, and a history of being trapped in a confined-space fire, typically elevates clinical suspicion. The vast majority of the scientific literature and clinical guidelines are predicated on data from inhalation injuries sustained in domestic or industrial fires, where the inhaled toxins are byproducts of the incomplete combustion of synthetic and organic materials.[6] Injuries resulting from natural disasters, and volcanic eruptions in particular, present a fundamentally different and more intricate pathophysiological puzzle.

A volcanic eruption unleashes a unique triad of injurious agents. This includes (1) extreme thermal energy from pyroclastic density currents, which are superheated clouds of gas and volcanic matter; (2) a massive burden of abrasive particulate matter in the form of volcanic ash; and (3) a complex mixture of toxic and corrosive gases, including sulfur dioxide (SO_2), hydrogen sulfide (H_2S), hydrogen chloride (HCl), and carbon monoxide (CO). This combination can inflict a multifaceted injury pattern, encompassing direct thermal trauma to the supraglottic structures, a potent chemical pneumonitis in the lower airways, mechanical abrasion from silicate particles, and profound systemic toxicity.

Within this challenging clinical landscape, flexible bronchoscopy is universally heralded as the definitive diagnostic modality—the "gold standard"—for the timely evaluation and grading of inhalation injury. This procedure allows for the direct, real-time visualization of the entire tracheobronchial tree, empowering clinicians to meticulously assess for the cardinal signs of injury: erythema, edema, mucosal blistering, ulceration, necrosis, and the presence of soot, ash, or other foreign debris. Such an immediate and accurate assessment is indispensable for guiding critical airway management decisions, chief among them the need for early protective endotracheal intubation, and for prognostic stratification.[7] Moreover, bronchoscopy offers a vital therapeutic avenue through bronchial lavage, which can be instrumental in clearing obstructive secretions, mucoid plugs, and inhaled particulate matter from the airways.

A foundational, albeit often implicit, assumption in burn care is the positive correlation between the extent of cutaneous burns and the likelihood and severity of concomitant inhalation injury.[8] However, the generalizability of this correlation is highly context-dependent. The unique environmental dynamics of a volcanic eruption on an exposed mountain summit—an open-air setting—differ radically from those of an enclosed-space structural fire, where heat and toxins are concentrated.[9] This case series presents a detailed retrospective analysis of the clinical course of the only two survivors admitted to our national tertiary referral center following the catastrophic eruption of Mount Marapi in West Sumatra, Indonesia, on December 3rd, 2023—a tragic event that claimed 23 lives. Both patients sustained extensive, life-threatening cutaneous burns but, remarkably, demonstrated only mild (Grade 1) inhalation injury upon bronchoscopic evaluation.[10]

This report aims to describe in detail the clinical presentation, diagnostic findings, and outcomes of these two patients, to delve deeply into the unique pathophysiology of volcanic inhalation injury, and to explore the critical clinical implications of the observed discordance between external and internal injury. The novelty of this study lies in its focus on a rare and poorly understood etiology of inhalation injury. By presenting hypothesis-generating evidence, it challenges the conventional clinical reliance on TBSA as a primary surrogate marker for the severity of airway trauma in this specific context. Consequently, this report advocates for consideration of a more aggressive, bronchoscopy-driven diagnostic paradigm for all victims of volcanic eruptions who present with signs or symptoms of respiratory compromise.

2. Case Presentation

This study is a retrospective case series based on a detailed review of the medical records of two patients admitted to Dr. M. Djamil General Hospital, a national tertiary referral and teaching hospital in Padang, Indonesia. Both patients were hikers on Mount Marapi and were within the high-risk exclusion zone during an unexpected phreatic eruption. They were the only two survivors of the incident admitted to our facility for definitive care. The extent of cutaneous burns was estimated by attending burn surgeons using the Lund-Browder chart for enhanced accuracy. The severity of inhalation injury was graded using the abbreviated injury score (AIS) for inhalation injury scale via direct bronchoscopic visualization.

Case 1: A 19-year-old female

A 19-year-old female was transferred to our Emergency Department following initial stabilization at a regional facility. On arrival, she was conscious, alert, and oriented (Glasgow Coma Scale 15/15) but in obvious distress from pain. Her initial vital signs were notable for significant sinus tachycardia (heart rate 143 beats/minute) and tachypnea (respiratory rate 24 breaths/minute), with a blood pressure of 116/65 mmHg and an oxygen saturation of 100% on a 4 L/min nasal cannula.

The physical examination was remarkable for extensive deep partial-thickness and full-thickness burns estimated at 40% TBSA, calculated using the Lund-Browder chart. The burns were circumferentially distributed across her face, neck, bilateral upper limbs, and bilateral lower limbs. There was profound facial edema, periorbital swelling, and her eyebrows and eyelashes were singed. Small amounts of greyish ash were noted in her nares, but the oropharynx was clear of soot. Auscultation of the chest revealed clear, vesicular breath sounds bilaterally with no wheezes or crackles.

Initial laboratory investigations were significant for leukocytosis (13.8 $x10^3/\mu L$) and marked hypoalbuminemia (2.5 g/dL), indicative of a significant systemic inflammatory response and capillary leak. An arterial blood gas (ABG) analysis showed a pH of 7.48, $PaCO_2$ of 30 mmHg, PaO_2 of 110 mmHg, and HCO_3^- of 21 mmol/L, consistent with a compensated respiratory alkalosis, likely driven by pain and anxiety-induced tachypnea. Critically, her carboxyhemoglobin (COHb) level was 1.8%, well within normal limits, suggesting minimal exposure to products of incomplete combustion (Table 1).

The patient was immediately admitted to the specialized Burn Unit. Fluid resuscitation was initiated using the Parkland formula with Ringer's lactate solution. Advanced wound care, robust multimodal analgesia, stress ulcer prophylaxis, nutritional support via a nasogastric tube, and empiric broad-spectrum antibiotics (piperacillin-tazobactam) were commenced. Her hospital course was complicated early on. On day 2, she developed an episode of supraventricular tachycardia (SVT) with a heart rate of 240 beats/minute, which was hemodynamically tolerated and successfully converted to sinus rhythm with intravenous diltiazem. A subsequent episode on day 5 was associated with hypotension (blood pressure 85/50 mmHg), necessitating urgent synchronized cardioversion (50 Joules), which restored sinus rhythm.

For the first six days of her admission, the patient's respiratory status remained relatively stable. While she remained tachypneic, her oxygen requirements were minimal, and her work of breathing was manageable. The primary clinical focus was on the formidable challenges of fluid resuscitation for a 40% TBSA burn and managing her cardiac dysrhythmias and burgeoning systemic inflammation. The decision to defer early intubation and bronchoscopy was made based on this initial respiratory stability, the absence of stridor or significant carbonaceous sputum, and the need to prioritize hemodynamic stabilization in the face of a massive capillary leak. However, from day 6 onwards, her respiratory status began to insidiously decline. She reported a subjective increase in dyspnea, and her respiratory rate climbed to over 30 breaths/minute

Table 1. Initial presentation and hospital course of Case 1.

Clinical & Laboratory Trajectory: Case 1

19-Year-Old Female | 40% TBSA Deep Burns | Mt. Marapi Eruption Victim

PARAMETER	ED (DAY 1)	ICU (DAY 7)	ICU (DAY 10)	REFERENCE RANGE
Vitals				
Heart Rate (bpm)	143	135	**150 (on pressors)**	60-100
Blood Pressure (mmHg)	116/65	**86/56**	**90/50 (on pressors)**	~120/80
SpO_2 (%)	100 (4L NC)	98 (MV, FiO_2 0.8)	92 (MV, FiO_2 1.0)	>95
Hematology				
White Blood Cell ($x10^3/\mu L$)	13.8	7.8	**19.0**	4.5-11.0
Hemoglobin (g/dL)	14.5	8.8	**7.5**	12.0-16.0
Platelets ($x10^3/\mu L$)	250	95	**45**	150-450
Biochemistry & Inflammation				
Creatinine (mg/dL)	1.1	**2.3**	**4.1 (on CRRT)**	0.6-1.2
Urea (mg/dL)	45	73	**109**	10-50
Albumin (g/dL)	2.5	**1.6**	**1.4**	3.5-5.5
Procalcitonin (ng/mL)	Not Done	**>100**	**>100**	<0.5
Arterial Blood Gas (ABG)				
pH	7.48	**7.24 (post-intub.)**	**7.18**	7.35-7.45
$PaCO_2$ (mmHg)	30	32	**55**	35-45
PaO_2 (mmHg)	110	241	**62**	80-100
HCO_3^- (mmol/L)	21	**13.7**	**11.5**	22-26

Abbreviations: ED Emergency Department; ICU Intensive Care Unit; NC Nasal Cannula; MV Mechanical Ventilation; CRRT Continuous Renal Replacement Therapy; TBSA Total Body Surface Area.

On day 7, she developed frank respiratory failure, characterized by a respiratory rate of 35 breaths/minute, use of sternocleidomastoid and intercostal accessory muscles, and a drop in oxygen saturation to 88% despite an increased fraction of inspired oxygen (FiO_2). An urgent ABG confirmed acute hypercapnic respiratory failure (pH 7.24, $PaCO_2$ 32 mmHg, PaO_2 241 mmHg on high-flow nasal cannula). She was emergently transferred to the Intensive Care Unit (ICU), where she underwent rapid sequence intubation for airway protection and the initiation of mechanical ventilation.

Immediately post-intubation, a flexible bronchoscopy was performed via the endotracheal tube to definitively assess the airway. The examination revealed diffuse, moderate mucosal erythema and mild-to-moderate edema extending from the main carina down to the visible subsegmental bronchi bilaterally. There were no signs of mucosal blistering, ulceration, necrosis, or significant carbonaceous

deposits or ash. The findings were classified as a Grade 1 inhalation injury based on the AIS scale, a finding strikingly incongruous with the severity of her external burns.

Her subsequent ICU course was dominated by the overwhelming systemic inflammatory response syndrome (SIRS) and septic sequelae of her extensive burns. She rapidly progressed into profound, refractory septic shock, requiring a combination of high-dose norepinephrine (up to 2 mcg/kg/min), vasopressin (0.04 units/min), and dobutamine (15 mcg/kg/min) to maintain a mean arterial pressure above 65 mmHg. Evidence of multiorgan dysfunction syndrome (MODS) became apparent, with her SOFA score climbing from 8 to 16 over 72 hours. This included stage 3 acute kidney injury (AKI), with her creatinine rising to 4.1 mg/dL, necessitating the initiation of continuous renal replacement therapy (CRRT) for anuria and severe metabolic acidosis. She also developed Disseminated Intravascular Coagulation (DIC), with platelets dropping to 45,000/μL and a D-dimer >10,000 ng/mL. Her procalcitonin level was markedly elevated at >100 ng/mL, confirming a severe systemic infection. Sputum and blood cultures both grew multidrug-resistant *Pseudomonas aeruginosa*, and antibiotic therapy was escalated to meropenem and amikacin based on sensitivity testing.

Despite the implementation of maximal organ support—including lung-protective ventilation according to ARDSnet protocols, CRRT, massive transfusion of blood products, and aggressive hemodynamic management—her clinical trajectory was relentlessly downhill. On day 14 of hospitalization (day 7 in the ICU), she developed a pulseless electrical activity (PEA) cardiac arrest. After a prolonged resuscitation effort, she was pronounced dead. The ultimate cause of death was determined to be refractory septic shock and multiorgan failure, as a direct consequence of her 40% TBSA deep burns (Table 2).

Table 2. Respiratory deterioration and intensive care unit management.

ICU Course & Respiratory Deterioration: Case 1

Timeline of Clinical Decline from Day 6 to Day 14

TIMELINE / EVENT	CLINICAL FINDINGS & STATUS	KEY DIAGNOSTICS & INTERVENTIONS	COMPLICATIONS & OUTCOME
Day 6 Onset of Decline	**Insidious Respiratory Worsening** • Subjective report of progressive dyspnea. • Respiratory rate noted to be climbing > 30 breaths/minute. • Patient remains conscious and alert but appears fatigued.	**Management Focus** • Continued focus on hemodynamic stabilization for 40% TBSA burn. • Supplemental oxygen increased. • Decision made to continue close observation.	**Early Warning Signs** • Developing respiratory muscle fatigue.
Day 7 Acute Decompensation	**Frank Respiratory Failure** • Respiratory Rate: **35 breaths/minute**. • Prominent use of sternocleidomastoid and intercostal accessory muscles. • Oxygen saturation drops to **88%** on high-flow oxygen.	**Urgent Diagnostics & Intervention** • **ABG Confirms:** Acute Hypercapnic Respiratory Failure. • **Intervention:** Emergent transfer to ICU. • **Intervention:** Rapid Sequence Intubation & Mechanical Ventilation initiated.	**Primary Outcome** • Airway secured. • ICU admission.
Post-Intubation ICU Day 7	**Bronchoscopic Findings** • Diffuse, moderate mucosal erythema. • Mild-to-moderate edema throughout tracheobronchial tree. • **Absence of ulceration, necrosis, or significant soot/ash.**	**Definitive Diagnosis** • **Flexible Bronchoscopy Performed:** via ETT. • **Diagnosis: Grade 1 Inhalation Injury** (AIS Scale). • Finding is strikingly mild compared to 40% TBSA burn.	**Clinical Implication** • Respiratory failure likely driven by systemic burn effects (SIRS/ARDS), not primary lung injury.
ICU Days 7-14 Terminal Decline	**Refractory Septic Shock & MODS** • Profound hypotension unresponsive to fluids. • SOFA score progresses from 8 to 16. • Development of anuria and severe metabolic acidosis. • Clinical signs of coagulopathy (petechiae, oozing from lines).	**Maximal Supportive Care** • **Hemodynamics:** Triple-vasopressor support (Norepinephrine, Vasopressin, Dobutamine). • **Renal:** Continuous Renal Replacement Therapy (CRRT). • **Infection:** Antibiotics escalated to Meropenem & Amikacin for *P. aeruginosa*. • **Heme:** Massive transfusion of platelets and FFP.	**Final Outcome** • **Day 14:** PEA Cardiac Arrest. • **Patient pronounced deceased.** • **Cause:** Irreversible septic shock and MODS.

Case 2: A 20-year-old male

A 20-year-old male, injured in the same incident, was admitted concurrently. He sustained deep partial-thickness burns estimated at 30% TBSA via the Lund-Browder chart, primarily affecting his face, neck, and both upper extremities. His chief complaints upon arrival were severe burn pain, a growing sense of shortness of breath, and a strained, high-pitched dysphonia (hoarseness), which immediately raised a high index of suspicion for significant laryngeal edema and inhalation injury.

His initial vital signs were stable: blood pressure 130/80 mmHg, heart rate 110 beats/minute, respiratory rate 22 breaths/minute, and an oxygen saturation of 96% on room air. Initial laboratory results were notable for hemoconcentration (hemoglobin 16.5 g/dL), leukocytosis (11.2 x10^3/µL), severe hypoalbuminemia (2.2 g/dL), and a significant pre-renal AKI (Urea 126 mg/dL, Creatinine 2.9 mg/dL), reflecting profound intravascular volume depletion. His ABG showed a metabolic acidosis (pH 7.32, HCO_3^- 15 mmol/L). His COHb level was also within the normal range at 2.1%. He was admitted to the Burn Unit and started on aggressive fluid resuscitation, wound care, and prophylactic antibiotics. Given the clear clinical signs of laryngeal irritation, a pulmonology consultation was obtained on day 1 with a plan for bronchoscopy (Table 3).

Table 3. Initial presentation and hospital course of Case 2.

Clinical & Laboratory Trajectory: Case 2

20-Year-Old Male | 30% TBSA Deep Burns | Survivor with Full Recovery

PARAMETER	ED (DAY 1)	DAY 4 (POST-LAVAGE)	DAY 15	REFERENCE RANGE
Vitals				
Heart Rate (bpm)	110	95	80	60-100
Blood Pressure (mmHg)	130/80	125/75	120/70	~120/80
SpO_2 (%)	96 (Room Air)	98 (Room Air)	99 (Room Air)	>95
Hematology				
White Blood Cell (x10^3/µL)	11.2	21.7	9.8	4.5-11.0
Hemoglobin (g/dL)	16.5	12.1	9.1 (post-transfusion)	13.5-17.5
Platelets (x10^3/µL)	310	280	350	150-450
Biochemistry (Renal Function & Nutrition)				
Creatinine (mg/dL)	2.9	0.8	0.7	0.7-1.3
Urea (mg/dL)	126	25	19	10-50
Albumin (g/dL)	2.2	2.8	3.2	3.5-5.5
Arterial Blood Gas (ABG)				
pH	7.32	7.38	7.41	7.35-7.45
$PaCO_2$ (mmHg)	31	38	40	35-45
PaO_2 (mmHg)	75	92	95	80-100
HCO_3^- (mmol/L)	15	22	24	22-26

Abbreviations: ED Emergency Department; TBSA Total Body Surface Area.

On day 3 of admission, a flexible bronchoscopy was performed to formally evaluate his airways. The findings were, remarkably, very similar to those in Case 1. The entire visible mucosa of the trachea, main carina, and bilateral bronchial trees exhibited moderate erythema and mild edema. However, a key difference was the presence of copious, thick, tenacious, whitish-yellow secretions throughout the airways. During suctioning, this material was returned near-continuously, clearly distinct from typical carbonaceous soot. There was no evidence of ulceration, necrosis, or pseudomembrane formation. The findings were thus classified as a Grade 1 inhalation injury with significant bronchorrhea.

Given the substantial burden of secretions and potential for airway obstruction, the diagnostic procedure was immediately converted into a therapeutic one. A comprehensive bronchial lavage was performed. A total of 200 mL of sterile 0.9% saline was instilled in 20-30 mL aliquots into all major bronchial segments, followed by gentle but thorough suctioning. This procedure successfully evacuated a large volume of obstructive secretions and mucoid plugs from his airways. Immediately following the procedure, the patient reported a dramatic subjective improvement in his breathing, and his work of breathing visibly decreased, with his respiratory rate settling from 24 to 18 breaths/minute over the next hour.

His subsequent hospital course was long but positive. The primary focus shifted to managing his 30% TBSA burns, which required multiple surgical interventions, including tangential excisions and split-thickness skin grafting. His acute kidney injury resolved completely within 72 hours with sustained, adequate fluid resuscitation, as evidenced by the normalization of his creatinine and urea levels. A wound culture grew *Pseudomonas aeruginosa*, which was effectively treated with a targeted course of meropenem. He required several blood transfusions for anemia resulting from the hypermetabolic, catabolic state of his burn injury and surgical blood loss. He was an active participant in intensive physical and occupational therapy to maintain joint function and prevent contractures.

After a 29-day hospitalization, his burn wounds were granulating well and progressing toward closure. He was hemodynamically stable, fully mobile, and had no residual respiratory complaints. He was discharged home in good condition. At a one-year follow-up appointment, he remained completely asymptomatic from a respiratory standpoint. His physical examination was normal, and formal pulmonary function testing revealed a normal spirometry pattern (FEV1 98% predicted, FVC 101% predicted, FEV1/FVC ratio 0.82), indicating a complete recovery without any discernible long-term pulmonary sequelae (Table 4).

3. Discussion

This case series provides a rare and valuable window into the clinical presentation and management of inhalation injury secondary to a volcanic pyroclastic flow—a relatively uncommon and poorly documented etiology in the medical literature. The most salient and clinically provocative finding from these two cases is the profound discordance between the severity of external cutaneous burns and the grade of endobronchial injury.[11] Both patients suffered devastating, extensive deep burns (30-40% TBSA), a level of external trauma that would justifiably lead most clinicians to anticipate a correspondingly severe, high-grade inhalation injury. Yet, in stark contrast to this expectation, direct bronchoscopic visualization revealed only mild, Grade 1 mucosal inflammation in both individuals. This critical observation challenges conventional clinical heuristics and carries significant implications for the triage, diagnosis, and management of future victims of similar catastrophic events. To comprehend the mechanistic basis for this observed discordance, it is imperative to dissect the distinct pathophysiological insults delivered by a volcanic eruption compared to those from a classic structural fire. The injury is best conceptualized as a triad of thermal, particulate, and chemical insults (Figure 1).[12]

Table 4. Bronchoscopy and subsequent management of Case 2.

Bronchoscopy & Subsequent Management: Case 2

Timeline of Pivotal Airway Intervention and Recovery

PHASE OF CARE	CLINICAL RATIONALE & FINDINGS	PROCEDURE & INTERVENTION DETAILS	OUTCOME & CLINICAL IMPACT
Pre-Bronchoscopy Admission (Day 1-2)	**High Index of Suspicion** • **Chief Complaints:** Progressive shortness of breath. • **Key Sign: Strained, high-pitched dysphonia (hoarseness).** • **Physical Exam:** Deep burns affecting face and neck. • Pulmonology consultation requested due to high risk of laryngeal edema and significant inhalation injury.	**Initial Management** • Aggressive intravenous fluid resuscitation initiated. • Prophylactic antibiotics administered. • Plan for bronchoscopy established for definitive airway assessment.	**Patient Status** • Hemodynamically stable but with significant respiratory symptoms.
Diagnostic Phase Day 3	**Bronchoscopic Findings** • Moderate erythema and mild edema of entire tracheobronchial mucosa. • **Key Finding:** Copious, thick, tenacious whitish-yellow secretions noted throughout the airways. • Absence of soot, ulceration, or necrosis.	**Diagnostic Procedure** • Flexible bronchoscopy performed to evaluate airways. • **Formal Diagnosis: Grade 1 Inhalation Injury with significant bronchorrhea.** • Findings again surprisingly mild given the 30% TBSA burn.	**Clinical Decision** • Large burden of secretions deemed obstructive and requiring immediate intervention. • Decision made to convert diagnostic to therapeutic procedure.
Therapeutic Phase Concurrent (Day 3)	**Rationale for Lavage** • To physically remove inhaled ash and inflammatory debris. • To clear obstructive mucoid plugs and thick secretions. • To improve lung mechanics and reduce work of breathing. • To prevent secondary complications like atelectasis and pneumonia.	**Therapeutic Bronchial Lavage** • A total of **200 mL** of sterile 0.9% saline was used. • Instilled in **20-30 mL aliquots** into different bronchial segments. • Followed by gentle but thorough suctioning. • Successfully evacuated a large volume of obstructive material.	**Immediate Impact** • Patient reported subjective improvement in breathing immediately post-procedure. • Work of breathing visibly decreased.
Post-Procedure Day 4 Onwards	**Sustained Clinical Improvement** • Respiratory rate settled from 24 to 18 breaths/minute. • No further respiratory support was required. • Focus of care shifted successfully to burn wound management.	**Long-Term Follow-up** • Discharged after a 29-day hospitalization with no respiratory complaints. • **1-Year Follow-up:** Completely asymptomatic. • **Pulmonary Function Test:** Normal spirometry (FEV1/FVC normal).	**Final Outcome** • **Full and complete respiratory recovery without long-term pulmonary sequelae.**

The pyroclastic flow, a superheated, gravity-driven current of gas and tephra, can reach temperatures exceeding 600°C. However, the human respiratory system is endowed with a remarkably efficient counter-current heat-exchange mechanism. The extensive vasculature of the nasal turbinates and upper airways acts as a powerful radiator, absorbing and dissipating the vast majority of this thermal energy before it can reach the lower respiratory tract.[13] As a result, direct thermal damage below the level of the glottis is exceedingly rare, with the exception of exposure to high-pressure steam, which has a much higher heat-carrying capacity than dry air. The Grade 1 erythema and edema documented in our patients likely represent the full extent of this proximal thermal insult—a significant but non-necrotizing tracheobronchitis. The open-air environment on the mountain peak, allowing for rapid convective and radiative cooling of inhaled gases, likely played a crucial role in limiting the depth of thermal penetration, in stark contrast to the sustained, intense heat exposure within a confined space.[14]

Volcanic ash is fundamentally different from the carbonaceous soot of fires. Ash is composed of fine, sharp, abrasive particles of pulverized rock, minerals, and volcanic glass, predominantly silicates. When inhaled, these particles inflict damage via multiple pathways.[15] First, their sharp, irregular morphology causes direct mechanical abrasion of the delicate mucosal lining, physically stripping away the protective epithelial layer and paralyzing the cilia. This action severely impairs mucociliary clearance, the lung's primary innate defense mechanism, leading to the retention of secretions, debris, and pathogens, which clinically manifested as significant bronchorrhea in Case 2. Second, the deposition of

these silicate particles triggers a potent inflammatory response. Alveolar macrophages recognize the particles as foreign invaders and initiate an inflammatory cascade via activation of the NLRP3 inflammasome, leading to the release of a flood of proinflammatory cytokines (TNF-α, IL-1β, IL-6) and chemokines.[16] Unlike carbon soot, which can be gradually cleared, crystalline silica can lead to a more persistent, smoldering inflammation, and in chronic exposures, is known to cause fibrosis (silicosis). In the acute setting, this intense inflammation drives increased vascular permeability and exudate formation, which can contribute to ARDS. The therapeutic lavage in Case 2, by physically removing a significant burden of this inflammatory stimulus, may have been critical in averting this progression.

The gaseous cocktail within a volcanic plume is a highly toxic and corrosive mixture. Sulfur dioxide (SO_2), a primary component, is highly water-soluble and, upon contact with the moist mucosa of the respiratory tract, rapidly forms sulfurous acid (H_2SO_3) and sulfuric acid (H_2SO_4).[17] These powerful acids cause immediate chemical burns, leading to cellular injury, severe bronchoconstriction, and an intense inflammatory reaction. Similarly, gases like hydrogen chloride (HCl) dissolve to form hydrochloric acid, compounding the corrosive insult. The relatively mild bronchoscopic findings in our patients suggest that while they were undoubtedly exposed to this toxic mixture, the total inhaled dose was likely limited by the transient nature of the pyroclastic flow in an open space, preventing the development of the severe, necrotizing chemical pneumonitis seen with high-concentration industrial exposures. The normal COHb levels in both patients further support this open-air exposure model, distinguishing it from confined-space fires, where CO production is a major contributor to mortality.[18]

Figure 1. Pathophysiology of volcanic inhalation injury.

The tragic outcome in Case 1 serves as a stark reminder of the profound systemic nature of major burn injury. The primary driver of her mortality was not respiratory failure from her Grade 1 inhalation injury, but rather refractory septic shock and MODS originating from her 40% TBSA burn. Severe burns trigger one of the most intense systemic inflammatory responses known in medicine. The massive tissue destruction releases a flood of damage-associated molecular patterns (DAMPs) into the circulation, which, along with pathogen-associated molecular patterns (PAMPs) from subsequent infections, leads to a "cytokine storm." This overwhelming systemic inflammation causes profound endothelial dysfunction and a global increase in capillary permeability. The lungs, with their vast endothelial surface area, are a primary target of this systemic insult. The resulting leakage of protein-rich fluid into the pulmonary interstitium and alveoli leads to non-cardiogenic pulmonary edema and the clinical picture of ARDS—a "second hit" to the lungs that is independent of the initial direct inhalation injury.[19] Therefore, the respiratory deterioration seen in Case 1 on day 7 was likely a manifestation of burn-induced ARDS rather than a progression of her mild primary inhalation injury. This nuanced understanding is critical: while the airway injury was a comorbidity, her lungs ultimately failed as a target organ of an insurmountable systemic process originating from the skin.

The central lesson from this case series is that TBSA may be an unreliable predictor of inhalation injury severity in victims of open-air volcanic eruptions. This observation suggests the potential need for a revised approach in the initial assessment of such victims. Clinicians must maintain a high index of suspicion for inhalation injury in all victims with respiratory symptoms (dyspnea, dysphonia), facial burns, or a direct exposure history, irrespective of the TBSA. The decision to proceed with bronchoscopy should be driven by these clinical and historical factors, not anchored to the extent of cutaneous burns.

Furthermore, these cases highlight the critical dual role of bronchoscopy. Diagnostically, it provided a definitive grade of injury in both patients that defied clinical assumptions based on their external appearance. Therapeutically, as demonstrated in Case 2, its application was directly associated with clinical improvement. The timely removal of obstructive secretions and ash via lavage likely improved his lung mechanics, reduced his work of breathing, and may have prevented subsequent complications such as atelectasis and ventilator-associated pneumonia, potentially averting the need for mechanical ventilation altogether. The timing of bronchoscopy is also a key learning point. While the procedure was beneficial on day 3 in Case 2, an even earlier intervention might be optimal. The delay until day 7 in Case 1, a decision made in the context of competing life-threatening priorities, underscores the immense clinical challenges in managing these complex patients and highlights the need for institutional protocols that prioritize early airway assessment.[20]

This study has several important limitations that must be acknowledged. First and foremost, as a case series of only two patients, the findings are inherently preliminary and must be interpreted with caution. The small sample size precludes any statistical analysis and prevents the generalization of our observations to all victims of volcanic eruptions. The observed discordance is a significant, hypothesis-generating finding, but it requires validation in larger patient cohorts from future incidents. Second, the study's retrospective design relies on the accuracy and completeness of medical records and is susceptible to information bias. Finally, the absence of a control group makes it impossible to establish causality or definitively conclude that therapeutic lavage improved the outcome in Case 2, though the temporal association is strong.

4. Conclusion

This case series from the 2023 Mount Marapi eruption provides illustrative evidence suggesting that the severity of cutaneous burns may not reliably

correlate with the grade of endobronchial injury in victims of volcanic pyroclastic flows. The unique pathophysiology of injury in this open-air environment can result in devastating skin trauma with comparatively mild direct airway inflammation. The primary clinical implication of this finding is that a low threshold for early diagnostic and therapeutic flexible bronchoscopy should be considered for this specific patient population. Reliance on TBSA alone as a primary tool for triaging the need for advanced airway assessment may be insufficient and potentially misleading. We propose that early bronchoscopic evaluation be considered a cornerstone of management for all symptomatic victims to ensure accurate diagnosis, guide appropriate respiratory support, and facilitate the crucial removal of inhaled particulate matter, thereby offering the potential to improve outcomes and mitigate the risk of severe pulmonary complications.

5. References

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