

When the Odds Seem Impossible: A Case of Severe Craniofacial Trauma, Ethical Dilemma, and Unexpected Recovery

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Abstract

Background: Severe craniofacial trauma in young adults remains one of the most challenging emergencies in modern medicine, combining life-threatening brain injuries with complex facial lesions. It inevitably raises a question that haunts intensive care physicians: how far should we go? When is persistence justified, and when does it cross into futility?

Case Presentation: We present the case of a 26-year-old man with no prior medical history who was brought to our trauma center after a high-speed motorcycle collision. He arrived with a Glasgow Coma Scale (GCS) score of 5/15 -- deeply unconscious and requiring immediate intubation. Imaging revealed extensive comminuted craniofacial fractures, a 12 mm extradural hematoma (EDH), massive subarachnoid hemorrhage, intraventricular bleeding, and right hemispheric hemorrhagic contusions. He underwent emergency decompressive craniectomy and EDH evacuation, followed by a prolonged and turbulent ICU stay marked by convulsive status epilepticus and post-operative bacterial meningitis. Despite everything, he survived -- and walked out of the neurosurgical ward 35 days later.

Conclusion: This case challenges the reflex to equate a low admission GCS with an irreversible prognosis. When early, aggressive, and coordinated multidisciplinary care is delivered -- and sustained through every complication -- meaningful neurological recovery is possible even in the most daunting presentations. The decision to keep going was not reckless; it was reasoned, collegial, and ultimately vindicated.

Keywords: Severe Traumatic Brain Injury; Extradural Hematoma; Glasgow Coma Scale; Neurological Intensive Care; Post-Operative Meningitis; Status Epilepticus; Medical Ethics; Therapeutic Obstinacy

Introduction

Every trauma surgeon and intensivist has faced it -- a young patient arriving barely alive, numbers that look unsurvivable, and the quiet but urgent question hanging in the air: do we push forward, or do we step back? Severe craniofacial trauma forces this question into the open, often within minutes of a patient crossing the emergency room threshold [1].

Road traffic accidents -- particularly those involving motorcycles -- remain the leading cause of traumatic brain injury (TBI) and acquired disability in young adults worldwide. The combination of cranial and facial injuries adds layers of complexity: airway management is

harder, bleeding is harder to control, and the anatomical destruction can be so profound that it skews first impressions toward the worst [2].

The Glasgow Coma Scale has guided our triage decisions for over fifty years. A GCS of 5 out of 15 is, by any measure, severe. It triggers protocols, informs family conversations, and quietly shapes how aggressively a team chooses to fight. But a number, however useful, is not a prognosis -- and this case is a reminder of that.

We report the full clinical course of a 26-year-old man admitted with a GCS of 5/15 following a high-impact motorcycle accident. His story -- from the roadside to the neurosurgery ward -- is a case study in what sustained, multidisciplinary critical care can achieve when the team chooses not to give up.

Case Presentation

The accident and initial presentation

A.A. was a 26-year-old man with no significant medical history. On March 12, 2026, he was involved in a high-velocity collision between his motorcycle and a car. He was ejected from the vehicle on impact -- a detail that, as we will see, carries both clinical and triage significance.

On arrival at the emergency department, he was deeply unconscious. His Glasgow Coma Scale score was 5/15 (E1V1M3) -- placing him firmly in the category of severe TBI and making immediate intubation not just indicated, but mandatory. His vital signs showed a blood pressure of 110/55 mmHg, heart rate of 120 bpm, and peripheral oxygen saturation of 92% on room air. Endotracheal intubation was performed without delay.

Triage and risk stratification

Using the Vittel pre-hospital triage score -- the French standard for identifying patients who require immediate transfer to a Level I trauma center -- our patient satisfied criteria from two separate categories simultaneously: a GCS of 5 and an SpO₂ below 90% (Step 1 physiological criteria), and vehicle ejection (Step 2 mechanism criteria). There was no ambiguity about where he needed to be.

Laboratory findings

Initial bloodwork was notable but not catastrophic. Hemoglobin was 11.5 g/dL, platelets were adequate at 250,000/mm³, and coagulation studies showed a mild coagulopathy (prothrombin time 75%, fibrinogen 1.5 g/L). CRP was elevated at 65 mg/L and albumin was mildly low at 32 g/L -- consistent with physiological stress. Electrolytes and renal function were within acceptable limits. No immediate pharmacological correction was required.

Imaging: The full extent of the damage

The whole-body CT scan performed on March 13 revealed a picture that was, frankly, alarming.

On the cranial side: Multifocal comminuted fractures with depression across the right frontoparietal, temporal, and occipital bones; a 12 mm extradural hematoma (EDH) overlying the fracture sites; moderate-to-severe subarachnoid hemorrhage filling the cortical sulci, interhemispheric fissure, and tentorium; moderate intraventricular hemorrhage; right hemispheric hemorrhagic contusions and petechial lesions; herniation of right parietal parenchyma through the fracture site; and diffuse pneumocephalus.

On the facial side: Fractures of the nasal bones involving the right nasolacrimal duct; comminuted fracture of the right orbital wall with pneumorbital and orbital fat incarceration; a bony fragment in contact with the superior and lateral rectus muscles; extension into the right orbital roof and cribriform plate; ethmoidal opacification; and comminuted fractures of both the lesser and greater wings of the right sphenoid, with air in the right infratemporal fossa.

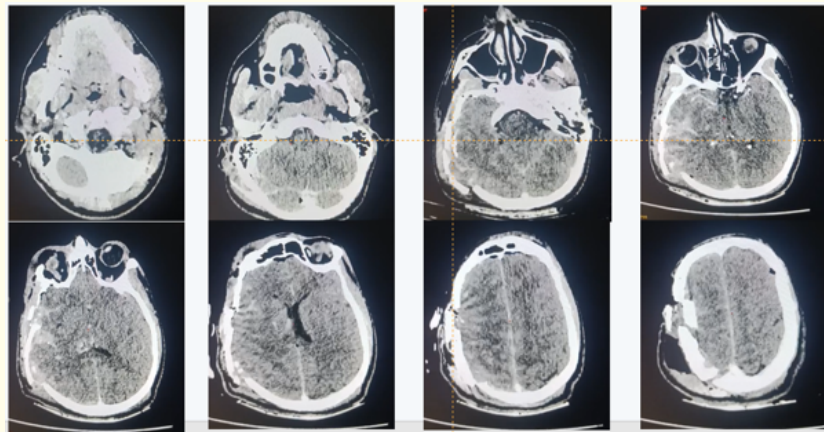


Figure 1

Emergency surgery: March 14

The decision to operate was immediate. On the morning of March 14, the patient underwent evacuation of the extradural hematoma combined with a decompressive craniectomy -- a procedure designed not just to remove the blood, but to give the swelling brain somewhere to expand without being crushed.

Induction of anesthesia required the immediate initiation of norepinephrine, as the patient was in hemodynamic shock at the time of induction. The operation lasted two hours. Intraoperative blood loss was substantial, necessitating transfusion of 2 packed red blood cell units and 3 fresh frozen plasma units, supplemented by human fibrinogen concentrate (Clottafact) and tranexamic acid (Exacyl 1g) for hemostasis.

Intraoperative arterial blood gas analysis showed a mixed acidosis (pH 7.19, pCO₂ 53.1 mmHg, HCO₃⁻ 19.5 mmol/L) with intraoperative hemoglobin of 8.4 g/dL -- evidence of the metabolic toll the surgery was taking.

Post-operative imaging: New concerns

The immediate post-operative CT scan was sobering. While the EDH had been partially evacuated (residual 8.5 mm extra-axial occipitoparagaleal hematoma), the scan revealed: massive subarachnoid hemorrhage now filling the basal cisterns; persistent moderate intraventricular flooding; worsening hemorrhagic contusions with involvement of the right basal ganglia; herniation of right parietal parenchyma through the craniectomy site; and -- most urgently -- early subfalcine herniation with a midline shift of 6 mm. A filling defect in the right transverse sinus raised concern for venous sinus thrombosis, prompting CT angiography.

ICU course: A battle on multiple fronts

The patient was admitted to our surgical ICU on March 15, intubated, ventilated, deeply sedated, and on continuous norepinephrine. For the first two days, his condition was stable but critical.

On day 2 (March 17), when sedation was lightened for neurological assessment, he developed generalized tonic-clonic convulsions. Continuous EEG monitoring confirmed convulsive status epilepticus. Antiepileptic therapy was escalated and optimized, and seizures were gradually controlled over the following days.

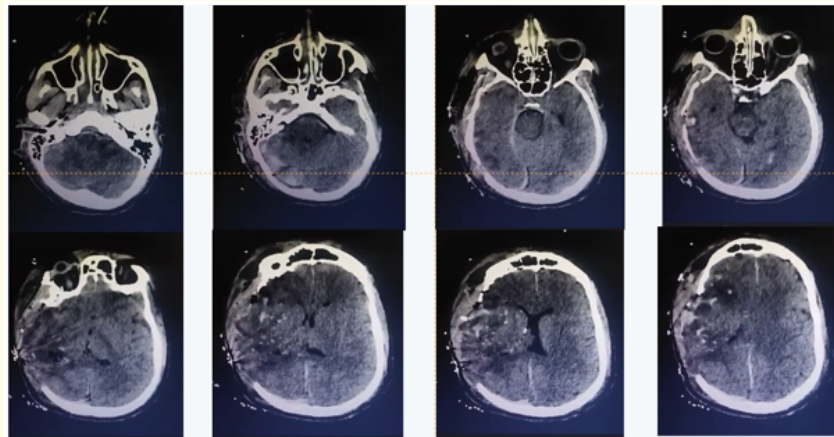


Figure 2

By March 20 (day 5), the first reassuring signs appeared: spontaneous eye opening and purposeful limb movements in response to pain. The team allowed themselves a cautious moment of hope.

Then, on March 23 (post-operative day 9), fever spiked to 39 degrees C. A full septic workup was initiated -- lumbar puncture, bronchoalveolar lavage, urinalysis and culture. The CSF results were unambiguous: 400 white cells/mm³, protein 2.9 g/L, glucose 0.3 g/L. Blood cultures and urine cultures were sterile. The diagnosis was post-operative bacterial meningitis.

Empirical broad-spectrum antibiotic therapy was started immediately: ceftazidime (a third-generation cephalosporin with anti-*Pseudomonas* activity) combined with vancomycin (covering MRSA and other Gram-positive organisms), for a planned total duration of 19 days. Defervescence occurred within 48 hours. By day 6 of antibiotics, the patient opened his eyes spontaneously. By day 8, he was following motor commands.

A follow-up CT scan revealed a new and concerning finding: a large extra-axial hypodense collection with peripheral enhancement along the right frontotemporo-parietal convexity (100 x 47 mm, extending 130 mm) -- consistent with a cerebral abscess. A communicating intraparenchymal collection (43 x 34.8 mm) was also identified, along with mass effect causing early subfalcine and mesial temporal herniation, and thrombosis of the left lateral sinus. The neurosurgical team reviewed the imaging and the patient's trajectory. Their recommendation: continue medical management. No further surgery.

Recovery and discharge

A tracheotomy was performed on March 20 to facilitate gradual weaning from mechanical ventilation. From that point, the rehabilitation team became a constant presence: chest physiotherapy, motor rehabilitation, enteral nutrition, and vitamin supplementation -- day after day, without interruption.

On April 13, the tracheostomy cannula was upgraded to a permanent silver cannula. Three days later, on April 16, 2026 -- 35 days after his arrival unconscious and barely breathing -- A.A. was transferred to the neurosurgery ward in stable condition.

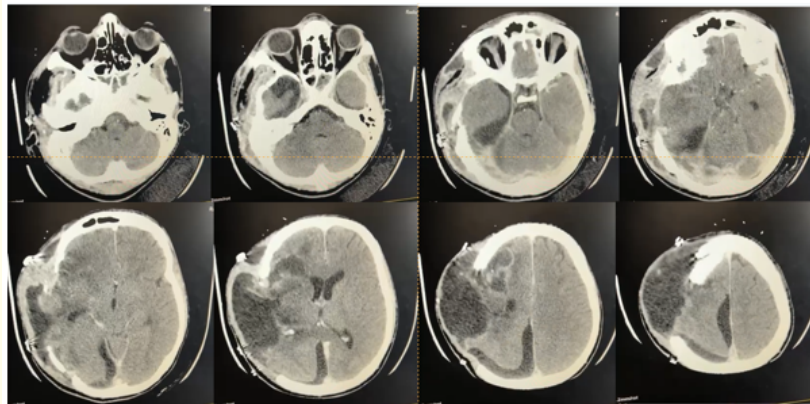


Figure 3

Discussion

Should a GCS of 5 define the prognosis?

The Glasgow Coma Scale has been the cornerstone of TBI triage since Teasdale and Jennett introduced it in 1974. A score below 9 defines severe TBI and mandates intubation; a score of 5 places a patient in the most critical stratum. Families are given grim statistics. Treatment intensity decisions are subtly -- sometimes not so subtly -- influenced.

Yet the evidence consistently tells us that initial GCS alone is an unreliable predictor of long-term outcome in young patients with isolated TBI and no pre-existing comorbidities. Multiple studies have documented meaningful recovery in patients admitted with GCS scores between 3 and 6, provided they received early, high-quality, sustained care [3]. Our patient is one more data point in that argument. A GCS of 5 at admission became a neurologically recovering patient at day 35. The number mattered for triage. It did not determine destiny [4].

The Vittel score and the value of standardized triage

The Vittel score is France's validated pre-hospital triage tool for identifying major trauma patients who require immediate transfer to a Level I trauma center [10]. It evaluates three sequential domains: physiological variables, injury mechanism, and anatomical lesions. The presence of any single criterion is sufficient for Level I referral.

In this patient, two categories were positive simultaneously -- a constellation that demands the most specialized resources available. Early triage to our surgical ICU, with neurosurgical capacity immediately on hand, was almost certainly a prerequisite for the outcome we observed [6]. Standardized triage tools exist precisely to remove hesitation from that decision [7].

Status epilepticus after TBI: The hidden threat

Post-traumatic seizures are a well-recognized complication of severe TBI, occurring in roughly 20 - 25% of patients with intracranial hemorrhage [8]. What makes them particularly treacherous is that a substantial proportion -- up to 25% in some series -- are subclinical: no visible convulsions, just continuous epileptiform activity on EEG quietly consuming neurons and deepening coma.

Continuous EEG monitoring was the tool that caught this patient's status epilepticus. Without it, the episode could easily have been attributed to residual sedation or post-operative obtundation -- and the delay in treatment could have been catastrophic. This case

reinforces what the literature increasingly confirms: in severe TBI patients being weaned from sedation, continuous EEG is not optional [9]. It changes management, and management changes outcomes.

Post-operative meningitis and cerebral abscess: A foreseeable complication

Post-operative bacterial meningitis following neurosurgery is a known risk, particularly when the procedure involves open skull fractures, dural tears, or prolonged operative time. The diagnostic triad -- fever, CSF pleocytosis, elevated protein, low glucose -- was unambiguous here. The absence of bacteriological growth on cultures is not unusual in patients already receiving perioperative antibiotics; the clinical and biochemical picture was sufficient to act on [10].

The empirical antibiotic regimen selected -- ceftazidime plus vancomycin -- followed established guidelines for post-neurosurgical meningitis, targeting the organisms most likely encountered in an ICU setting: Gram-negative bacilli including *Pseudomonas*, and resistant Gram-positive cocci. The rapid clinical response confirmed the appropriateness of this choice [11].

The subsequent development of a large cerebral abscess was the final major complication in an already complex course. The neurosurgical team's decision to manage it conservatively -- without reoperation -- was based on the patient's improving neurological trajectory and the inherent risks of a third intracranial procedure in this context. It was the right call. The patient continued to improve.

The ethics of persistence: When is enough?

This is the question the title poses, and it deserves a direct answer.

The concept of 'therapeutic obstinacy' -- or in its more nuanced legal framing, 'unreasonable persistence' -- describes the continuation of treatments that offer no realistic prospect of benefit and only prolong suffering. Every healthcare system struggles to define its boundaries. In France, the Leonetti Law (2005) and its subsequent amendments provide a legal framework for limiting or withdrawing active therapies in the ICU, requiring collegial decision-making, documentation, and consultation with the patient's family [10].

For A.A., the decision to persist was not made blindly or without reflection. It was grounded in specific clinical reasoning: he was 26 years old with no comorbidities; his injuries, while catastrophic, were traumatic in origin and therefore potentially reversible; he showed early physiological responses to treatment; and at no point did the team observe the clinical signs that might have justified withdrawal -- absent brainstem reflexes, refractory intracranial hypertension, or multi-organ failure incompatible with life.

It is worth acknowledging, with honesty, that this story could have ended differently. The same clinical reasoning applied to another patient with similar initial parameters might have led to survival with severe disability rather than meaningful recovery. That uncertainty is inherent to the work. What matters is that the decision was made thoughtfully, revisited regularly, and grounded in evidence and ethics -- not in reflex or inertia [12].

The lesson is not 'always push forward no matter what.' The lesson is: do not let a number on a scoring scale substitute for clinical judgment. A GCS of 5 is not a death sentence. It is a call to urgency.

Conclusion

A.A. arrived in our emergency department on March 12 with a GCS of 5 and injuries that would have led many clinicians to privately doubt his chances. He left our neurosurgical ward on April 16, alive, neurologically improving, and on a trajectory toward rehabilitation.

His case does not prove that every patient with a GCS of 5 will recover. It proves that some will -- and that the difference is made by the quality, speed, and coordination of the care they receive. Early intubation and hemodynamic stabilization, standardized trauma triage,

emergency neurosurgery, continuous EEG monitoring, prompt treatment of infectious complications, collegial ethical decision-making, and relentless rehabilitation: none of these elements alone was sufficient. Together, they were.

We offer this case not as a triumph, but as a reminder. The most important tool in a trauma resuscitation bay is not a scanner or a surgical drill. It is the willingness to keep asking -- not 'should we stop?' but 'what does this patient still need from us?'

Conflicts of Interest

The authors declare no conflicts of interest related to this article.

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