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Case Report

Severe Traumatic Brain Injury Complicated by Aspiration Pneumonia: When Delayed Intubation Becomes the Prognostic Turning Point: A Case Report

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Abstract

Background: Severe traumatic brain injury (TBI) is a life-threatening emergency whose prognosis depends closely on how early the airway is protected. Aspiration pneumonia is a frequent and largely preventable complication.

Case Presentation: We report a previously healthy 19-year-old man involved in a high-energy road traffic accident (motorcycle, ejection, no helmet), admitted with a Glasgow Coma Scale (GCS) of 11/15 and vomiting. Imaging showed multiple haemorrhagic contusions, intraventricular haemorrhage and early ground-glass lung opacities. Intubation was deferred because the GCS was deemed "non-severe" and was performed only 24h later, after deterioration to 8/15. On day 3 the patient developed severe aspiration pneumonia (Fever 39°C, desaturation to 86% on 100% FiO_2, purulent secretions, a "crazy-paving" pattern) complicated by septic shock.

Outcome: With empirical antibiotics, neuro-ventilatory critical care and a tracheostomy on day 10, the outcome was favourable (18 ventilator-days, 29 ICU-days).

Conclusion: This case shows that the decision to intubate a head-injured patient must never rest on an isolated GCS value, but on global, repeated clinical assessment.

Keywords: *Severe Traumatic Brain Injury; Aspiration Pneumonia; Intubation; Glasgow Coma Scale; Secondary Brain Injury; Critical Care*

Introduction

In severe traumatic brain injury we often speak of the initial lesion as though everything were decided at the moment of impact. In truth, much of the prognosis is settled afterwards, in the hours following admission, where the clinician can still make a difference. That is the whole point of fighting secondary brain insults of systemic origin: hypoxia, hypotension and swings in carbon dioxide worsen the neurological outcome independently of how severe the original injury was [7].

In that fight, airway protection holds a special place. A patient whose consciousness is impaired loses the reflexes that protect the airway; if he vomits, gastric contents reach the lower airways and aspiration pneumonia sets in. This is no rare event: under mechanical ventilation, nearly one brain-injured patient in two develops an acquired pneumonia, far more than the average ICU patient [3].

The case we report illustrates, perhaps better than any lengthy argument, what happens when intubation is deferred on the strength of a single Glasgow score. We felt it worth sharing in order to revisit the indications for intubation in head injury, the price of a delay, and the value of returning regularly to the patient's bedside.

Case Presentation

Patient and circumstances of the trauma

The patient was a 19-year-old student, healthy until then, with no medical history or medication, who neither smoked nor used alcohol or drugs. He was the victim of a road traffic accident: his motorcycle struck a car, he was ejected and was not wearing a helmet. No care was given at the scene; he was evacuated by the fire brigade. With no medicalised handover, the resuscitation-room team knew neither the exact time of the accident, nor the transport delay, nor the patient's neurological state before arrival - a blind spot that would weigh on the interpretation of events.

Admission to the resuscitation room (03/02/2026, 19:09)

At first glance the picture was reassuring: stable haemodynamics, no signs of respiratory distress. One detail, however, should have drawn attention - the patient had vomited on arrival. In someone whose consciousness is already blunted, this is precisely the scenario that heralds aspiration.

Blood pressure	130/80 mmHg
Heart rate	80 bpm
SpO_2	96%
Respiratory rate	18/min
Capillary refill	< 3s
Temperature	Afebrile
GCS	11/15 (M = 5/6)
Pupils	Equal and reactive
Examination	Normal lung auscultation and abdomen; no focal motor deficit

Table 1

Initial imaging (whole-body CT)

Brain: Petechial haemorrhagic contusions in the right frontal, left parietal, pericallosal and periventricular regions; intraventricular haemorrhage (third ventricle and occipital horns of both lateral ventricles).

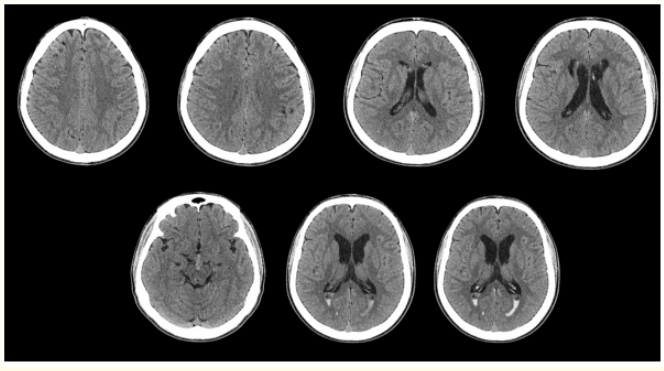

Figure 1: *Initial brain CT (Axial slices): Petechial haemorrhagic contusions (Right frontal, left parietal, pericallosal and periventricular) with intraventricular haemorrhage.*

Chest: Ground-glass opacities of the upper lobes and right postero-basal region, most marked in the apical segment of the right upper lobe; a thin left pneumothorax. These parenchymal changes, present from admission, already pointed to early aspiration, most likely before or during transport.

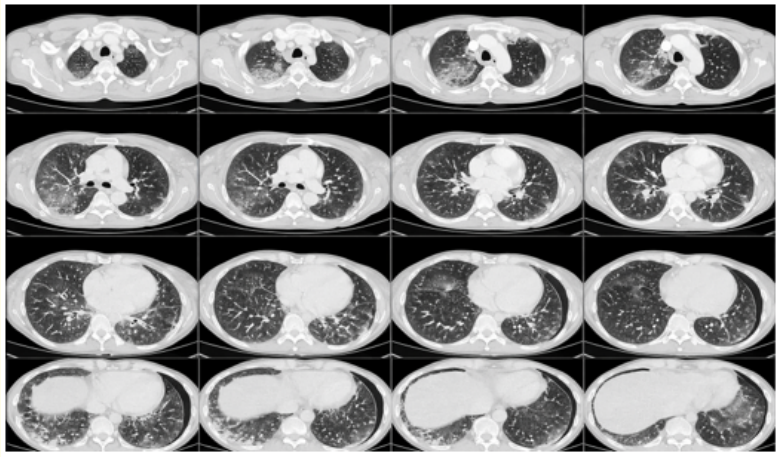

Figure 2: *Initial chest CT (lung window): bilateral ground-glass opacities predominating in the upper lobes and right postero-basal region - the first aspiration lesions.*

Laboratory findings on admission

CBC	Hb 12.7 g/dL; Hct 37.9%; WBC 16,100/mm^3; Platelets 188,000/mm^3
Coagulation	PT 61%; aPTT 34.8/28s; Fibrinogen 1.36 g/L
Electrolytes	Na^+ 139; K^+ 3.7; Cl^- 106
Renal function	Urea 0.27 g/L; Creatinine 7.3 mg/L
Liver/muscle	AST 60; ALT 50; CK 514; LDH 310

Table 2

The leukocytosis and mild coagulopathy (low PT, low fibrinogen) fitted the context of polytrauma and acute brain injury.

Day 1: A deferred intubation

On admission, intubation was put off. The reasoning seemed sound: a Glasgow of 11, reactive pupils, no focal deficit, acceptable haemodynamics and saturation. As long as the figure stayed above 8, the patient was filed under "non-severe." But that figure, taken in isolation, eclipsed everything else: a young man who was vomiting, with haemorrhagic contusions and blood in the ventricles, whose airway remained unprotected. Intubation came only some twenty hours later, once the Glasgow had fallen to 8.

Arterial blood gases drawn just after intubation confirmed that the damage was done: pH 7.26, PaO_2 56.8 mmHg, $PaCO_2$ 40.8 mmHg, lactate 1.4 mmol/L. The acidosis and hypoxaemia made it plain that respiratory involvement was already established by the time the airway was finally secured.

Day 3: Aspiration pneumonia and septic shock

On the third day the respiratory picture tipped over. Fever rose to 39°C and plateaued, oxygen saturation fell to 86% even though the patient was receiving 100% oxygen, and suctioning brought up copious purulent secretions. The diagnosis of aspiration pneumonia was unavoidable.

Follow-up chest CT (06/02/2026) showed foci of left lower-lobe consolidation, right lower-lobe parenchymal consolidations with ground-glass opacities and septal lines producing a "crazy-paving" pattern, a moderate pneumomediastinum and a stable thin left pneumothorax: a worsening bilateral pneumonia, most likely of aspiration origin.

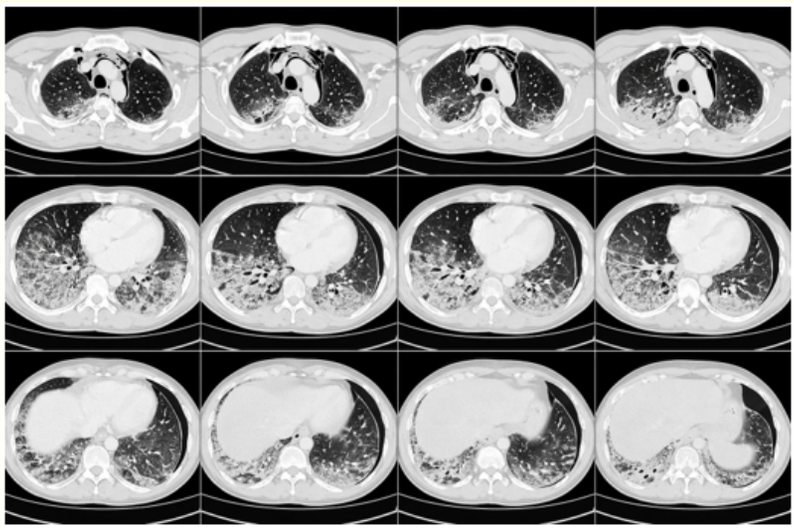

Figure 3: *Follow-up chest CT (day 3): Lower-lobe parenchymal consolidations, ground-glass opacities and septal lines forming a "crazy-paving" pattern - established aspiration pneumonia.*

Follow-up brain CT meanwhile showed multiple oedemato-haemorrhagic contusion foci, moderate subarachnoid haemorrhage, haemorrhagic filling of the right quadrigeminal cistern, and effacement of the cortical sulci reflecting the onset of cerebral oedema. The respiratory complication and the neurological deterioration thus progressed hand in hand, illustrating the hypoxia-secondary-insult vicious circle.

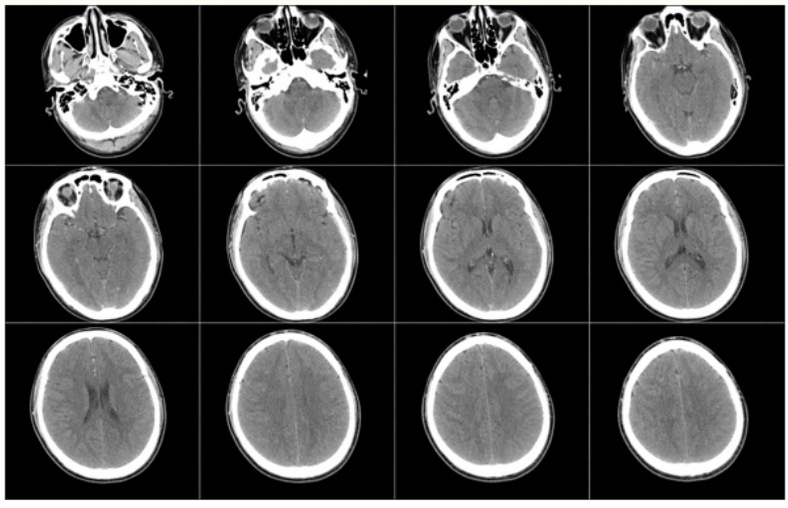

Figure 4: *Follow-up brain CT (day 3): Multiple oedemato-haemorrhagic contusion foci and effacement of the cortical sulci indicating the onset of cerebral oedema.*

Microbiological work-up

A protected distal bronchial sample was taken to document the infection. It came back negative, with no organism at the usual threshold of 10^4 CFU/mL. Such a negative result is not surprising here - it is common once antibiotics have already been started, or when the chemical component of aspiration (Mendelson's syndrome) predominates over bacterial infection. It did not rule out the diagnosis, but it did allow treatment to be scaled back afterwards.

Management

- **Empirical antibiotics:** A broad-spectrum beta-lactam combined with an aminoglycoside - imipenem 1g every 8h IV and amikacin 1.5g every 24h IV - justified by the severity of the picture, for a total of 6 days (stopped after 72h without fever).
- **Neurological critical care:** Appropriate sedation and systematic prevention of secondary brain insults - normoxia (SpO_2 > 95%), normocapnia, MAP ≥ 80 mmHg, normoglycaemia, normothermia and head elevation at 30°.
- **Mechanical ventilation:** Assist-control mode, FiO_2 80%, tidal volume 450 mL, rate 12/min, PEEP 5 cmH_2O, together with daily chest physiotherapy.
- **Adjunctive measures:** Norepinephrine for the initial septic shock, early enteral nutrition, vitamin supplementation (B1, B6, B12, vitamin C, trace elements), prophylactic anticonvulsant, low-dose neuroleptic for agitation, a proton-pump inhibitor for stress-ulcer prophylaxis, prophylactic low-molecular-weight heparin from day 7, and mouth and eye care (honey, eye drops). A tracheostomy was performed on day 10.

Overall course and outcome

In all, it took 18 days of ventilation and 29 days of intensive care to bring this young patient safely through. The septic shock was controlled, neurological recovery was satisfactory, and he was able to leave the unit. A happy ending, then - but one bought at the cost of a month in intensive care for a complication that could probably have been avoided.

Discussion

The trap of the isolated Glasgow score in head injury

That a Glasgow of 8 or below mandates intubation is not in dispute: it is a level-1 recommendation of the Eastern Association for the Surgery of Trauma, a reflex taught from ATLS onwards [1]. The trap lies elsewhere - in the almost automatic reverse reading, which assumes that above 8 one can wait. That overlooks the fact that a Glasgow of 11 in a patient who is vomiting, with haemorrhagic contusions and blood in the ventricles, is anything but reassuring: it is a score waiting to fall. The number is meaningful only in relation to its trajectory and to the lesions underlying it.

Indeed, several situations call for intubation whatever the Glasgow: swallowing difficulties or repeated vomiting, intracranial lesions worsening on CT, respiratory distress, hypoxia or hypercapnia, haemodynamic instability, unmanageable agitation, and seizures [2]. In our patient, the vomiting and the evolving lesions were on their own enough to justify intubation from admission.

Consequences of delayed airway protection

Waiting, here, meant running two risks at once. On the pulmonary side, it allowed aspiration to occur: the ground-glass opacities already visible on the admission CT, and then the "crazy-paving" pattern on day 3, tell the story of a pneumonia first arising and then worsening, with the potential to tip into ARDS and sepsis. On the cerebral side, the resulting hypoxaemia is one of the worst secondary insults that can be inflicted on an injured brain. Yet maintaining normoxia and normocapnia remains one of the most effective levers we have for protecting the neurological prognosis [8].

The blood gases drawn after intubation (pH 7.26, PaO_2 56.8 mmHg) leave little doubt: by the time the airway was finally protected, the lung was already involved. And this complication should not be underestimated. In ventilated brain-injured patients, acquired pneumonia affects between one in three and one in two, prolongs ventilation and length of stay, and in its own right weighs on the risk of severe cognitive sequelae [4].

Chemical pneumonitis or bacterial pneumonia? Reading a negative bronchial sample

It helps, first, to agree on terms. A distinction is often drawn between aspiration pneumonitis - Mendelson's syndrome, a chemical burn of the lung by acidic gastric fluid - and bacterial aspiration pneumonia, a true infection arising from the mouth's own flora [5]. The first heals largely with supportive care, the second requires antibiotics. In practice the two overlap, and it is rarely possible to tell them apart at the outset [10].

It is in this grey zone that our patient's negative bronchial sample should be read. It does not say there was no aspiration; it may mean that the chemical component dominated, that the sample was taken after antibiotics had begun, or that the bacterial load stayed below threshold. Above all, it allowed antibiotics to be stopped after 72 hours without fever - exactly what current thinking recommends: hit hard and early in severe forms, then reassess at 48 - 72 hours and ease off as soon as cultures and the patient allow [12].

Antibiotic strategy

Faced with septic shock on aspiration pneumonia, choosing a broad-spectrum beta-lactam combined with an aminoglycoside, such as imipenem and amikacin, is defensible: it covers Gram-negative bacilli and provides initial synergy. That treatment lasted only six days, stopped once the fever had settled, is in keeping with current practice, which favours short courses to spare the bacterial ecology as soon as the patient improves [10].

The place and timing of tracheostomy

As for the tracheostomy performed on day 10, it falls within a reasonable window. When prolonged ventilation is anticipated in severe TBI, a relatively early tracheostomy shortens ventilation, reduces ICU stay and lowers the risk of acquired pneumonia. The ideal moment, however, is still debated and remains uncodified [9].

The central message: Return to the bedside

If this case had to be summed up in a single line, it would be a matter not of technique but of attitude. In a severely injured brain, deterioration is not a nasty surprise: it is predictable, and therefore to be anticipated rather than endured. A borderline Glasgow is not a green light to wait; it is an invitation to come back and see the patient, often, ideally every hour. Intubating at the right moment blocks aspiration, hypoxia and secondary brain insults all at once. And it all begins there: protecting the airway early already means preventing aspiration pneumonia.

Limitations

Let us stay measured: this is a single case, reported in retrospect, whose prehospital phase is entirely unknown to us. Aspiration may well have occurred before arrival at hospital, so the pneumonia cannot be attributed to the delayed intubation alone. The strength of this observation is educational, not demonstrative.

Conclusion

This young man pulled through, and that is what matters most. But his story - a severe head injury, a severe aspiration pneumonia, septic shock, a month in intensive care - recalls a rule we would be wrong to think self-evident: one does not intubate a number, one intubates a patient. The Glasgow score means something only alongside the rest: what the CT shows, what the patient is doing, what we

fear for the hours ahead. Anticipating deterioration, securing the airway early, and relentlessly hunting down secondary brain insults: nothing new, but everything is there.

Key Messages

1. Never intubate - or defer intubation - on an isolated Glasgow score: assess the patient as a whole.
2. In severe brain lesions, neurological deterioration is predictable and demands anticipation.
3. A borderline GCS requires close, bedside clinical reassessment.
4. Intubating in time prevents aspiration, hypoxia and secondary brain insults.
5. Aspiration pneumonia is preventable: prevention starts with early airway protection.

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