World Journal of Advanced Research and Reviews
eISSN: 2581-9615 CODEN (USA): WJARAI
Cross Ref DOI: 10.30574/wjarr
Journal homepage: https://wjarr.com/

(RESEARCH ARTICLE)

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ACUTE RESPIRATORY DISTRESS SYNDROME AFTER TRAUMATIC BRAIN INJURY: TIMING, ASSOCIATED CLINICAL FACTORS, MANAGEMENT, AND OUTCOMES IN A MOROCCAN INTENSIVE CARE UNIT

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World Journal of Advanced Research and Reviews, 2026, 31(02), 1566–1575

Publication history: Received on 18 July 2026; revised on 26 August 2026; accepted on 28 August 2026

Article DOI: https://doi.org/10.30574/wjarr.2026.31.2.2243

ABSTRACT

Background: Acute respiratory distress syndrome (ARDS) is an important extracranial complication of traumatic brain injury (TBI), but its timing, associated clinical factors, and management are difficult to disentangle from the effects of prolonged mechanical ventilation and neurocritical illness. Data from North African intensive care units are limited. We aimed to describe the incidence, timing, associated clinical factors, management, and short-term outcomes of ARDS occurring after TBI in a Moroccan university intensive care unit.

Methods: We conducted a retrospective, single-center observational study of patients older than 15 years admitted with head trauma to the anesthesiology and intensive care unit of Hassan II University Hospital, Fez, Morocco, from January 2020 through December 2022. Patients were included if they developed ARDS according to the Berlin definition during the ICU stay. Patients with respiratory failure attributed to a cardiac origin were excluded. Patients with confirmed or suspected SARS-CoV-2 infection were managed in a dedicated COVID-19 unit separate from Unit A1 and therefore were not part of the source population analyzed. Clinical, radiological, therapeutic, and outcome data were collected from hospital records and analyzed descriptively.

Results: Among 2,217 ICU admissions, 270 involved head trauma and 45 patients developed ARDS (16.7% of head-trauma admissions, 95% CI 12.7-21.6; 2.0% of all ICU admissions). Mean age was 34.2 years and 84.4% were male. ARDS developed a mean of 7.2 days after trauma and 6.4 days after ICU admission. Mean PaO2/FiO2 at diagnosis was 156 mmHg; ARDS was mild in 20.0%, moderate in 57.8%, and severe in 22.2%. Frequently documented associated clinical factors were ventilator-associated pneumonia (75.6%), sepsis or septic shock (53.3%), associated chest trauma (35.6%), aspiration (17.8%), and pancreatitis (11.1%); categories overlapped. Mean duration of mechanical ventilation was 17.6 days. Inhaled nitric oxide was used in 37.8% and prone positioning in 28.9%. ICU mortality was 66.7%; 24.4% survived with documented severe neurological and/or cognitive impairment at ICU discharge and 8.9% survived without documented severe neurological/cognitive impairment.

Conclusions: In this cohort, ARDS after TBI was usually diagnosed several days into the ICU course and occurred in a population with a high burden of nosocomial infection, particularly ventilator-associated pneumonia. ICU mortality was

very high. These findings highlight the complexity of brain-lung interactions after TBI and the importance of preventing respiratory and infectious complications during prolonged neurocritical care.

Keywords: Acute Respiratory Distress Syndrome; Traumatic Brain Injury; Ventilator-Associated Pneumonia; Neurocritical Care; Mechanical Ventilation; Morocco.

1 INTRODUCTION

Traumatic brain injury (TBI) is a major cause of death and disability and frequently requires prolonged intensive care, invasive mechanical ventilation, and complex management of secondary cerebral insults. Pulmonary complications are particularly important because hypoxemia, hypercapnia, hemodynamic instability, and ventilator-induced lung injury may all interact with cerebral physiology. Acute respiratory distress syndrome (ARDS) therefore creates a therapeutic conflict: the lung benefits from limitation of ventilatory stress and strain, whereas the injured brain requires reliable oxygen delivery, tight carbon dioxide control, and preservation of cerebral perfusion.

The Berlin definition standardized ARDS diagnosis and severity in 2012 [1]. A new global definition published subsequently broadened diagnostic options, including non-intubated ARDS and approaches applicable to resource-limited settings, but the Berlin definition remains appropriate for retrospective classification of patients treated during the 2020-2022 study period [2]. In general ICU populations, ARDS affects approximately one in ten admissions and is associated with mortality that increases with severity [3]. After acute brain injury, reported ARDS incidence varies substantially according to case mix, screening strategy, injury severity, and ventilation practices [4-6].

Two broad, non-exclusive mechanisms are relevant after TBI. Early brain-lung crosstalk may promote neurogenic pulmonary oedema and inflammatory lung injury through sympathetic activation, endothelial dysfunction, and systemic inflammatory signaling. Later in the ICU course, impaired airway protection, prolonged sedation, mechanical ventilation, aspiration, and nosocomial infection may become dominant. Distinguishing these patterns is clinically relevant because they imply different preventive priorities.

Evidence from North African neurocritical care settings remains limited. We therefore conducted a three-year single-center study to describe the frequency, timing, severity, associated clinical factors, management, rescue respiratory therapies, and short-term outcomes of ARDS after TBI in a Moroccan university ICU.

2 METHODS

2.1 Study design and setting

This was a retrospective, non-interventional, single-center observational study conducted in the anesthesiology and intensive care unit (Unit A1) of Hassan II University Hospital, Fez, Morocco, a tertiary university referral Centre. The study period extended from January 2020 through December 2022. Reporting was structured in accordance with the principles of the STROBE statement for observational studies [7].

2.2 Participants

All patients admitted during the study period with head trauma were screened. Patients were included if they were older than 15 years and developed ARDS during their ICU stay according to the Berlin definition [1]. Patients younger than 15 years, patients who died in the emergency department before ICU admission, and patients with respiratory failure attributed to a cardiac origin were excluded. Patients with confirmed or suspected SARS-CoV-2 infection were managed in a dedicated COVID-19 unit separate from Unit A1 and therefore were not part of the source population analyzed in this study. Echocardiographic assessment was used when a cardiogenic origin of pulmonary oedema was considered.

2.3 Data sources and variables

Data were extracted using a standardized case-report form from the ICU admission register, clinical records, paraclinical investigations, and the hospital information system. Recorded domains included age, sex, comorbidities and smoking history, trauma mechanism, admission Glasgow Coma Scale (GCS), pupillary findings, respiratory and hemodynamic status, cerebral and thoracic imaging, laboratory data, ventilatory management, sedation and neuromuscular blockade, rescue respiratory therapies, infectious investigations, ICU length of stay, and outcome.

2.4 ARDS and associated clinical factors

ARDS severity was categorized using the Berlin PaO2/FiO2 strata. The interval from trauma and from ICU admission to ARDS diagnosis was recorded. Clinical factors documented in association with ARDS were assigned using the available

clinical, radiological, and microbiological information. Categories included ventilator-associated pneumonia (VAP), sepsis/septic shock, associated chest trauma, aspiration, and pancreatitis. Because these conditions were multifactorial, potentially overlapping, and their temporal relationship to ARDS could not be established uniformly from the retrospective records, they are reported as associated clinical factors rather than as proven causes or independent risk factors.

2.5 Clinical management

All included patients were invasively mechanically ventilated from ICU admission. The unit protocol targeted tidal volumes of 6-8 mL/kg, plateau pressure below 30 cmH2O, positive end-expiratory pressure (PEEP) of at least 5 cmH2O, and a respiratory rate around 12 breaths/min, with head elevation of 30-45 degrees. Sedation included midazolam, propofol, and fentanyl. Rocuronium was administered at initiation of invasive ventilation, with repeated boluses when required for ventilator adaptation or neurocritical care. Neurological surveillance included repeated clinical examination, cerebral imaging, and daily transcranial Doppler assessment. Inhaled nitric oxide and prone positioning were used as rescue strategies in selected patients with severe hypoxemia.

2.6 Outcomes

Prespecified outcomes were ICU mortality, survival with documented severe neurological and/or cognitive impairment at ICU discharge, survival without documented severe neurological/cognitive impairment, ICU length of stay, duration of mechanical ventilation, renal deterioration, anemia, and use of rescue respiratory therapies.

2.7 Statistical analysis

Data were entered and analyzed using Microsoft Excel 2019. Continuous variables are reported as means with ranges when available, and categorical variables as counts and percentages. The analysis was primarily descriptive.

2.8 Ethics

The study was conducted in accordance with the Declaration of Helsinki and approved by the Institutional Review Board of the University Hospital. It was based on retrospective review of medical records and a computerized clinical database, with no additional intervention or patient contact. The requirement for individual informed consent was waived by the Institutional Review Board because of the retrospective nature of the study.

3 RESULTS

3.1 Study population and incidence

During the three-year study period, 2,217 patients were admitted to the ICU. Of these, 270 (12.2%) were admitted with trauma involving the head, and 45 developed ARDS during the ICU stay, corresponding to 16.7% of head-trauma admissions (95% CI 12.7-21.6) and 2.0% of all ICU admissions (95% CI 1.5-2.7) (Figure 1).

3.2 Baseline characteristics

Mean age was 34.2 years (range 18-83). Four patients (8.9%) were aged 18-20 years, 28 (62.2%) were aged 21-40 years, 10 (22.2%) were aged 41-60 years, and 3 (6.7%) were older than 60 years. Thirty-eight patients (84.4%) were male. Chronic smoking was documented in 26 patients (57.8%). Trauma occurred in an urban setting in 39 patients (86.7%). Road traffic collisions accounted for 34 cases (75.6%), falls for 9 (20.0%), and assault for 2 (4.4%). Among road traffic collisions, 16 patients (47.1%) were motorcyclists, 11 (32.4%) pedestrians, and 7 (20.6%) car occupants.

The mean admission GCS was 9: 20 patients (44.4%) had GCS <=8, 15 (33.3%) had GCS 9-12, and 10 (22.2%) had GCS 13-15. Respiratory failure was present at admission in 40.0%, with a mean oxygen saturation of 91% (range 50-99%). Admission cerebral CT was abnormal in all included patients and showed a spectrum of traumatic lesions including extra- and intraparenchymal hematomas, contusions, skull fractures, and radiological signs compatible with intracranial hypertension.

Table 1: Baseline characteristics of the 45 patients with traumatic brain injury who developed ARDS. Percentages are calculated on n = 45 unless otherwise specified. GCS, Glasgow Coma Scale.

Characteristic	n	%
Mean age, years (range)	34.2 (18-83)	-
Age 18-20 years	4	8.9
Age 21-40 years	28	62.2
Age 41-60 years	10	22.2
Age >60 years	3	6.7
Male sex	38	84.4
Chronic smoking (toxicological history)	26	57.8
Urban location of trauma	39	86.7
Road traffic collision	34	75.6
Motorcyclist among road traffic collisions	16	47.1
Pedestrian among road traffic collisions	11	32.4
Car occupant among road traffic collisions	7	20.6
Admission GCS <=8	20	44.4
Admission GCS 9-12	15	33.3
Admission GCS 13-15	10	22.2
Scalp wound	23	51.1
Respiratory failure on admission	18	40.0

3.3 Timing, severity, and associated clinical factors

ARDS was diagnosed a mean of 7.2 days after the initial trauma (range 3-20) and 6.4 days after ICU admission (range 3-19). Mean PaO2/FiO2 at diagnosis was 156 mmHg (range 51-290). ARDS was mild in 9 patients (20.0%), moderate in 26 (57.8%), and severe in 10 (22.2%).

Table 2: Timing, severity, and associated clinical factors of ARDS. Categories were not mutually exclusive and therefore sum to more than 100%. ICU, intensive care unit; ARDS, acute respiratory distress syndrome.

Characteristic	n	%
Mean onset from trauma, days (range)	7.2 (3-20)	-
Mean onset from ICU admission, days (range)	6.4 (3-19)	-
Mean PaO2/FiO2 at diagnosis, mmHg (range)	156 (51-290)	-
Mild ARDS	9	20.0
Moderate ARDS	26	57.8
Severe ARDS	10	22.2
Associated factor: ventilator-associated pneumonia	34	75.6
Associated factor: sepsis/septic shock	24	53.3
Associated factor: associated chest trauma	16	35.6
Associated factor: aspiration	8	17.8
Associated factor: pancreatitis	5	11.1

ARDS was considered multifactorial. VAP was the most frequently documented associated clinical factor, reported in 34 patients (75.6%), followed by sepsis or septic shock in 24 (53.3%), associated chest trauma in 16 (35.6%), aspiration in 8 (17.8%), and pancreatitis in 5 (11.1%). These categories overlapped. Every patient had at least one documented infection during the ICU stay; because the timing of infection relative to ARDS could not be established uniformly from the retrospective records, these findings should not be interpreted as proof of causation.

3.4 Respiratory management and clinical course

All patients received invasive ventilation using the protective targets described above. Mean duration of mechanical ventilation was 17.6 days (range 7-31). Inhaled nitric oxide was used in 17 patients (37.8%) for severe refractory hypoxemia. Prone positioning was used in 13 patients (28.9%): 10 with severe and 3 with moderate ARDS. Patients undergoing prone positioning received a mean of 2 sessions, each lasting 8-18 hours (mean 12.6 hours). Improvement in oxygenation was observed after positioning in all proned patients; the magnitude of the change in PaO2/FiO2 was not systematically recorded.

During follow-up, 40 patients (88.9%) had at least one hemoglobin value below 10 g/dL, and renal function deteriorated in 11 (24.4%).

3.5 Outcomes

Mean ICU length of stay was 23.5 days overall, with means of 22.6 days for mild ARDS, 23.0 days for moderate ARDS, and 25.4 days for severe ARDS. Thirty patients died in the ICU (66.7%). Eleven patients (24.4%) survived with documented severe neurological and/or cognitive impairment at ICU discharge, and four (8.9%) survived without documented severe neurological/cognitive impairment.

Table 3: Respiratory management, clinical course, and outcomes. ICU, intensive care unit.

Management/course/outcome	n or mean	%
Mean duration of mechanical ventilation, days (range)	17.6 (7-31)	-
Inhaled nitric oxide	17	37.8
Prone positioning	13	28.9
Anaemia during ICU stay	40	88.9
Renal function deterioration	11	24.4
Mean ICU length of stay, days	23.5	-
ICU death	30	66.7
Survival with documented severe neurological/cognitive impairment	11	24.4
Survival without documented severe neurological/cognitive impairment	4	8.9

4 DISCUSSION

4.1 Principal findings

This three-year Moroccan ICU series identifies four clinically relevant features of ARDS after TBI. First, ARDS occurred in 16.7% of admissions involving head trauma. Second, diagnosis was generally delayed, at a mean of 6.4 days after ICU admission, rather than clustered in the immediate post-injury period. Third, infectious complications were prominent among the associated clinical factors, particularly VAP and sepsis. Fourth, global outcome was poor, with two thirds of the cohort dying in the ICU. Together, these findings emphasize the complex interaction between cerebral injury, prolonged mechanical ventilation, pulmonary complications, and systemic infection.

4.2 Incidence and the problem of case definition

The 16.7% incidence observed among 270 head-trauma admissions lies within the wide range previously described after acute brain injury. Earlier cohorts reported substantial variation in ARDS frequency, reflecting differences in TBI severity, mechanical-ventilation exposure, concomitant extracranial trauma, screening intensity, and evolving diagnostic criteria [4-6]. Such heterogeneity makes simple cross-study comparison hazardous. In particular, a cohort restricted to invasively ventilated severe TBI patients is expected to yield a different denominator and risk profile from a general ICU head-trauma population. Recent multicentre work has also focused on early risk stratification for ARDS

after TBI, underscoring continuing interest in identifying high-risk patients while leaving important gaps regarding the clinical course and management of established ARDS in underrepresented settings [8].

The present study used the Berlin definition, which was the accepted standard during the study period [1]. The newer global definition of ARDS, published after completion of the cohort, expands diagnostic pathways by allowing pulse-oximetry-based oxygenation assessment, high-flow nasal oxygen, lung ultrasound, and a resource-limited setting category [2]. For methodological consistency, the present analysis retains the Berlin definition used to identify and classify ARDS during the study period.

4.3 Late timing and brain-lung interaction

ARDS after acute brain injury can arise through overlapping pathways. The initial cerebral insult may trigger sympathetic discharge, systemic inflammation, endothelial activation, and altered pulmonary vascular permeability, creating a biological substrate for early lung injury. Mechanical ventilation, transfusion, aspiration, chest trauma, and infection can then act as additional insults. The mean ARDS onset of 7.2 days after trauma and 6.4 days after ICU admission in this series suggests that, for many patients, the clinically recognized syndrome emerged after several days of critical care rather than during the earliest neurogenic phase.

That temporal pattern does not exclude a brain-driven contribution. The injured brain may remain a susceptibility factor throughout the ICU course, and prolonged ventilation is itself partly a consequence of neurological severity. The appropriate interpretation is therefore not a dichotomy between “neurogenic” and “infectious” ARDS, but a layered model in which initial brain injury, ventilator exposure, aspiration risk, thoracic injury, and nosocomial infection interact over time. This framework is particularly relevant because most trials establishing lung-protective ventilation excluded or underrepresented patients with acute brain injury, leaving major evidence gaps for simultaneous brain and lung protection [9].

4.4 Ventilator-associated pneumonia and the infectious burden

The predominance of VAP among the associated clinical factors is one of the most clinically important observations. VAP was reported in 75.6% of patients with ARDS, sepsis or septic shock in 53.3%, and every patient had a documented infection at some point during the ICU stay. These figures should not be interpreted as the incidence of VAP among all ventilated TBI patients, because the denominator in the present analysis consists only of patients who developed ARDS. Nor can they establish VAP as a causal precipitant in every case, because the retrospective records did not permit uniform reconstruction of the temporal relationship between infection and ARDS. Nonetheless, they demonstrate a very high infectious burden within the ARDS subgroup.

Contemporary neurocritical-care data reinforce the plausibility of this observation. In a 2023 analysis of the international ENIO cohort, VAP occurred in 39.5% of eligible acute brain-injured patients and in 49.5% of the TBI subgroup; VAP was strongly associated with longer ventilation and ICU stay, although it was not independently associated with ICU mortality [10]. A more recent 2026 single-center TBI study reported VAP in 50% of mechanically ventilated patients, with a median onset around day 5 and a high burden of multidrug-resistant Gram-negative pathogens; in that cohort, VAP was independently associated with mortality [11]. The apparent discrepancy in mortality effects across studies illustrates how strongly results depend on case mix, microbiology, severity adjustment, and local ecology. In our series, the predominance of VAP and sepsis among associated clinical factors reinforces the clinical importance of infection prevention during prolonged neurocritical ventilation.

The practical implication is therefore clinically relevant: prevention, early recognition, microbiological documentation, and protocolized management of nosocomial respiratory infection deserve high priority in prolonged neurocritical ventilation. Prospective multicenter studies may further clarify the temporal relationship between infectious complications and ARDS after TBI.

4.5 The brain-lung ventilation dilemma

Management of established ARDS in TBI requires simultaneous limitation of ventilator-induced lung injury and avoidance of secondary cerebral insults. General ARDS guidelines support low tidal volume ventilation in the range of 4-8 mL/kg predicted body weight and limitation of injurious airway pressures [12]. The unit protocol in this cohort targeted tidal volumes of 6-8 mL/kg, plateau pressure below 30 cmH2O, and PEEP of at least 5 cmH2O, while maintaining close neurological surveillance.

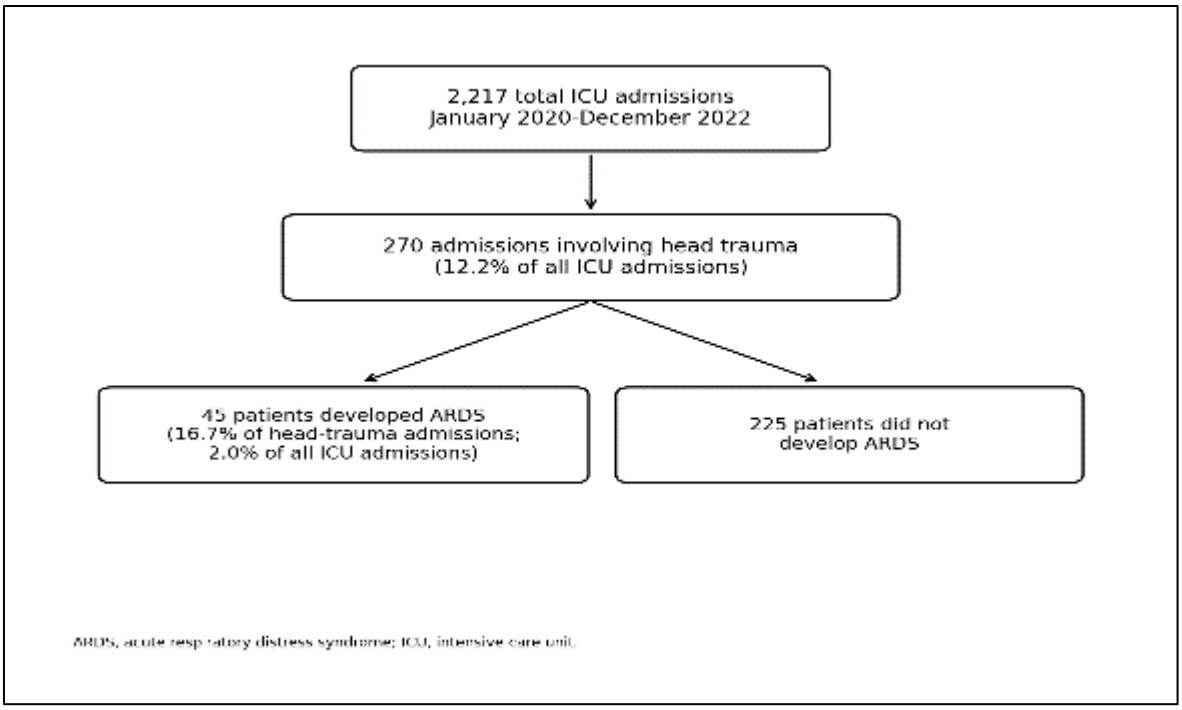

Figure 1: Study flow diagram. ARDS, acute respiratory distress syndrome; ICU, intensive care unit.

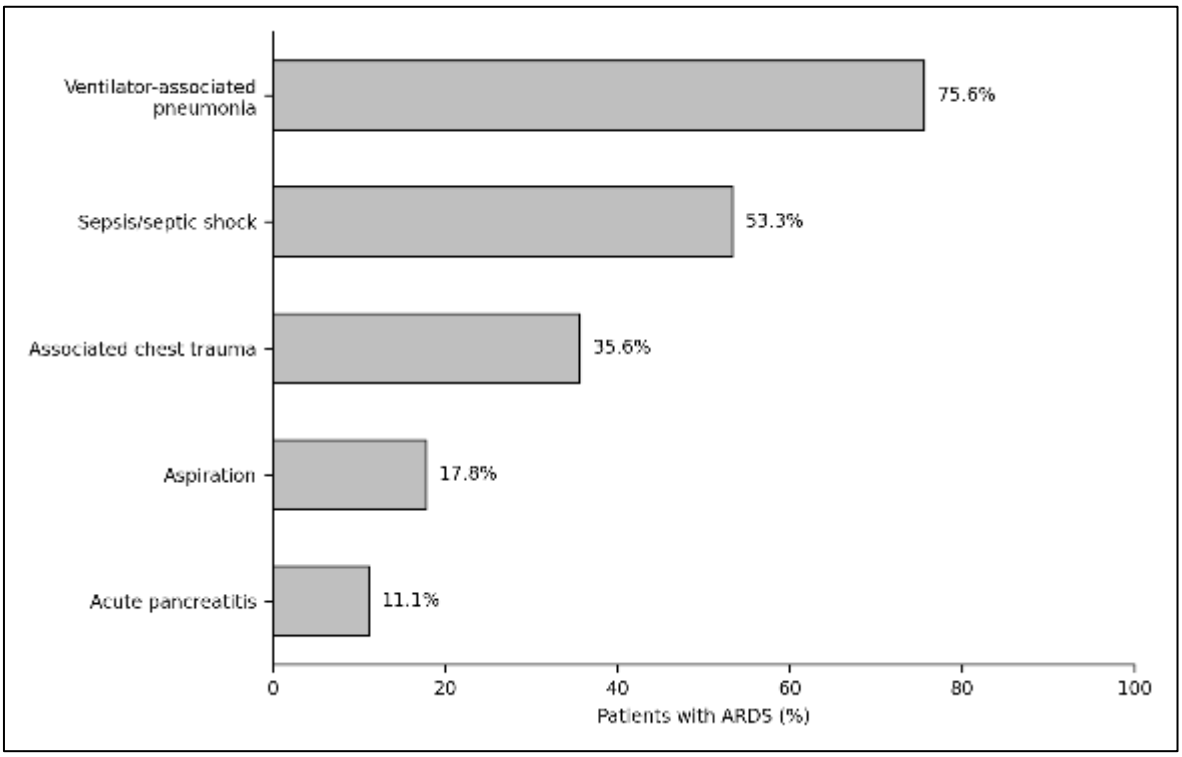

Figure 2: Associated clinical factors documented in patients with ARDS. Categories were not mutually exclusive and therefore sum to more than 100%.

The cerebral consequences of ventilator settings are particularly relevant. Hypercapnia may increase cerebral blood flow and intracranial blood volume, while excessive intrathoracic pressure can impair venous return and cerebral perfusion in susceptible patients. The 2020 ESICM consensus therefore emphasized individualized ventilation in acute brain injury and acknowledged the low certainty of evidence for several commonly used strategies [9]. More recent data have sharpened rather than eliminated this uncertainty. In the PROLABI randomized trial, a lung-protective strategy in severe acute brain injury without established ARDS did not improve the prespecified composite outcome and was

associated with signals of harm; the trial ended early and requires confirmation, but it cautions against uncritical transfer of one-organ ventilation targets to all brain-injured patients [13]. Conversely, the large prospective VENTIBRAIN study showed that protective-range settings are now common internationally, while ventilatory pressures and other parameters remain associated with mortality and vary substantially across countries [14].

For patients with established ARDS, the appropriate lesson is not to abandon lung protection but to individualize it around cerebral physiology. Adequate oxygenation, avoidance of extreme PaCO2 deviations, maintenance of arterial pressure, careful PEEP titration, and monitoring of neurological tolerance are central. In this cohort, repeated neurological assessment, cerebral imaging, and daily transcranial Doppler formed part of routine neurocritical surveillance.

4.6 Prone positioning and rescue therapies

Prone positioning was used in 28.9% of the cohort, including 10 patients with severe and 3 with moderate ARDS. In general ARDS populations, prolonged prone positioning reduces mortality in appropriately selected patients with severe hypoxaemia [15]. Application to acute brain injury is more complex because prone positioning may alter venous drainage, intra-abdominal and intrathoracic pressure, and intracranial pressure. Small neurocritical series suggest that proning can substantially improve oxygenation, but intracranial tolerance is heterogeneous and requires careful positioning and monitoring [16].

Improved oxygenation was observed in all patients who underwent prone positioning. This finding is consistent with the physiological rationale for prone positioning in ARDS, although it should be interpreted within the observational nature of the study. Inhaled nitric oxide was used in 37.8% of patients as rescue therapy for refractory hypoxemia, illustrating the severity and complexity of respiratory management in this neurocritical population.

4.7 High mortality and neurological outcome

The most striking clinical signal is the poor global prognosis. At diagnosis, most patients had moderate ARDS and the mean PaO2/FiO2 ratio was 156 mmHg. Thirty patients (66.7%) died in the ICU. Of the 15 survivors, 11 had documented severe neurological and/or cognitive impairment at ICU discharge and four had no documented severe neurological/cognitive impairment. Earlier TBI studies also found that acute lung injury/ARDS identifies patients with worse outcomes [5,6]. These observations underscore that respiratory failure in severe neurotrauma occurs within a broader syndrome in which the primary brain lesion, secondary cerebral injury, systemic infection, and extracranial organ dysfunction all contribute to prognosis.

This distinction matters. In a neurocritical population, improvement in oxygenation does not necessarily translate into neurological survival because the dominant determinant of outcome may remain the primary brain lesion or subsequent secondary cerebral injury. Conversely, systemic infection and ARDS can worsen cerebral oxygen delivery and hemodynamic stability, thereby aggravating neurological damage. The high mortality observed in this cohort is therefore consistent with a bidirectional interaction between cerebral injury and systemic respiratory and infectious complications.

4.8 Regional and public-health context

The cohort was young, predominantly male, and largely injured in road traffic collisions, with motorcyclists representing the largest subgroup among road traffic victims. This pattern is clinically important because it places the ICU burden within a broader preventable trauma context. It also underscores the value of generating data from middle-income settings, where trauma epidemiology and critical-care organization may differ from those represented in much of the ARDS literature. Prospective regional registries could further integrate trauma severity, neurocritical care, respiratory management, and infection surveillance.

4.9 Strengths and limitations

The study has several strengths. It describes a three-year real-world ICU experience from an underrepresented North African setting; it provides a clear denominator of head-trauma admissions; and it reports ARDS timing, severity, associated clinical factors, ventilatory management, rescue therapies, ICU length of stay, and short-term neurological outcome. The late timing and high infectious burden generate a clinically testable hypothesis for future work.

The principal limitations of this study are its retrospective, single-center design and the limited sample of 45 patients. The statistical analysis was descriptive, and no comparison was made with the head-trauma patients who did not develop ARDS; the study therefore describes a population rather than identifying risk factors. The associated clinical factors were multifactorial and not mutually exclusive. Distinguishing VAP from ARDS is intrinsically difficult, since both rely in part on new pulmonary infiltrates and impaired oxygenation; moreover, the retrospective records did not permit uniform reconstruction of the temporal sequence between infection and ARDS. The neurological outcome categories

were based on documentation available at ICU discharge rather than on a prospectively collected validated functional scale. Finally, the study period coincided with the COVID-19 pandemic. Patients with confirmed or suspected SARS-CoV-2 infection were managed in a dedicated COVID-19 unit and were not part of the Unit A1 source population; nevertheless, pandemic-related changes in ICU organization, staffing ratios, and antimicrobial ecology may have indirectly influenced the burden of nosocomial infection observed in the hospital. These characteristics should be considered when interpreting and generalizing the findings.

5 CONCLUSION

ARDS developed in approximately one in six ICU admissions involving head trauma in this three-year Moroccan cohort. It was generally recognized several days into the ICU course, most cases were moderate at diagnosis, and VAP and sepsis were frequent associated clinical factors. ICU mortality was 66.7%. These findings emphasize the clinical importance of preventing pulmonary and infectious complications while balancing lung-protective ventilation with the physiological requirements of the injured brain. Prospective multicenter studies are needed to validate these observations and refine management strategies for this high-risk population.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

Disclosure of conflict of interest

The authors declare that they have no competing interests.

Availability of data and materials

The data supporting this study are not publicly available because of privacy and ethical restrictions. De-identified study data are available from the corresponding author upon reasonable request and with permission from University Hospital and the institutional ethics committee.

Statement of ethical approval

This study was conducted in accordance with the Declaration of Helsinki and approved by the Institutional Review Board of the University Hospital.

Statement of informed consent

Given the retrospective review of existing medical records and the absence of any additional intervention or patient contact, the requirement for individual informed consent was waived by the Institutional Review Board.

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