

MEETING ABSTRACTS

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P001

A machine learning model for predicting 7-day stroke recurrence using imaging markers in transient ischemic attack (TIA) patients

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Introduction: Predicting early stroke recurrence after transient ischemic attack (TIA) is crucial in emergency settings, where rapid intervention for high-risk patients is essential. While the ABCD2 score is traditionally used for recurrence risk assessment, adding imaging markers may improve predictive accuracy. This study integrates perfusion, hemorrhage after reperfusion marker (HARM), diffusion-weighted imaging (DWI), and stenosis with the ABCD2 score in a machine learning model to enhance 7-day stroke recurrence predictions.

Methods: Data were collected from TIA patients who presented to the ED between 2010 and 2023. Variables analyzed included the ABCD2 score and imaging markers: perfusion, HARM, DWI, and stenosis. After evaluating the association of these variables with recurrence risk, they were integrated into the final model. A random Forest model was developed, with random oversampling applied to enhance the learning of stroke recurrence in the minority class. Model performance was evaluated using accuracy, precision, recall, and F1-score.

Results: The random Forest model combining imaging markers with the ABCD2 score achieved an accuracy of 80.7%, with a precision of 52.8% and a recall of 81.0%. In contrast, the model using only the ABCD2 score demonstrated a lower accuracy of 64.3%, with comparatively reduced precision and recall for recurrence prediction. The imaging markers were identified as significant contributors to short-term stroke recurrence predictions in ED patients with TIA, enhancing the model's predictive capability.

Conclusions: This study suggests that a machine learning model integrating the ABCD2 score with imaging markers effectively predicts early stroke recurrence in TIA patients. With an 81.0% recall, the model enables rapid identification of high-risk patients, supporting timely

intervention and clinical decision-making in emergency settings. This approach may contribute to improved management and preventive strategies for high-risk patients in the ED.

P002

Exploring the link between diastolic blood viscosity and distribution of brain lesion in acute ischemic stroke

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Critical Care 2025, **29**(S1):P002

Introduction: Blood viscosity (BV) plays a critical role in blood rheology and has been linked to microvascular perfusion, contributing to the development of cerebrovascular diseases. However, its influence on the lateralization of brain lesions has not been fully explored. This study aims to investigate the association between BV, particularly dynamic blood viscosity (DBV), and white matter hyperintensity (WMH) burden, as well as the occurrence and lateralization of brain lesions in patients with acute ischemic stroke (AIS).

Methods: We prospectively enrolled 208 patients with AIS within 7 days of symptom onset. BV was assessed using a scanning capillary tube viscometer, with DBV measured at a shear rate of 5 s^{-1} . Based on DBV values, patients were stratified into three groups according to established reference ranges. WMH volumes were quantified and normalized using automated localization and segmentation software. A multivariable logistic regression analysis was performed to assess the relationship between DBV levels and the lateralization of brain lesions in AIS patients.

Results: Lateralization of brain lesions in AIS patients was significantly associated with atrial fibrillation, elevated fasting glucose, and DBV levels. After adjusting for multiple variables, lower DBV (OR 2.036, 95% CI 1.179–3.515) was independently associated with left hemisphere lesions in AIS patients (Table). Additionally, WMH burden was



Introduction: Hyponatremia, defined as serum sodium > 145 mmol/L, is common in critically ill patients. Extreme hyponatremia (> 190 mmol/L) is rare, with high mortality [1]. While typical causes include dehydration and salt ingestion, extreme weight-cutting practices in competitive sports are underreported. This case highlights the challenges and adaptations needed in hyponatremia management.

Methods: A 29-year-old athlete presented with seizures and unresponsiveness after severe dehydration via hot baths and fluid restriction to cut weight for competition. Initial serum sodium exceeded 190 mmol/L (Table), with acute kidney injury and metabolic encephalopathy. ICU management required deviations from standard hyponatremia protocols, including using a hypertonic dialysate in continuous veno-venous hemodiafiltration (CVVHDF). The dialysate initially matched the patient's serum sodium and was gradually reduced by titrating hypertonic saline, ensuring a slower correction rate. Multidisciplinary care addressed systemic complications.

Results: Sodium was cautiously lowered by approximately 8–10 mmol/L/day to avoid osmotic demyelination syndrome. Imaging revealed metabolic brain injury. Despite metabolic stabilization, neurological deficits required prolonged ICU care. Neurorehabilitation resulted in assisted walking after two months, though mild motor deficits persisted.

Conclusions: This case emphasizes the risks of extreme weight-cutting and the critical need for flexible, individualized sodium correction strategies when standard protocols are inadequate. By tailoring CVVHDF, we safely managed sodium correction. Multidisciplinary care was essential to address systemic and neurological sequelae. Greater awareness and proactive intervention are crucial to prevent similar cases.

Acknowledgement: Written consent to publish was received from the patient.

Reference

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Table (abstract P208) Parameters

Marker	On admission	24 h	48 h	7 days
Sodium (mmol/L)	> 190	185	171	140
Potassium (mmol/L)	6	3.9	4.5	4.3
pH	7.17	7.3	7.32	7.33
Anion gap (mmol/L)	Could not compute	26.3	18.4	8.7
Hematocrit (%)	0.64	0.54	0.47	0.28
Urea (mmol/L)	20.7	24.7	21.6	7.5
Creatinine (μmol/L)	642	826	802	358

P209

Prevalence of septic cardiomyopathy in sepsis and septic shock using echocardiography-based tools
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Introduction: Septic cardiomyopathy (SCM) is a reversible myocardial dysfunction in sepsis or septic shock, with a widely variable incidence of 10% to 70% due to the lack of specific diagnostic criteria. This study aimed to evaluate the prevalence of SCM using different echocardiography-based diagnostic tools.

Methods: A single-center, prospective observational study was conducted at a tertiary reference hospital from March to September 2024. Transthoracic echocardiography (TTE) and hemodynamic assessment were performed within 48 h of enrolment. The diagnosis of SCM was made using four diagnostic criteria: left ventricular ejection fraction (LVEF < 50% or ≥ 10% reduction from baseline), cardiac power output (CPO < 0.6 W), afterload-related cardiac performance (ACP < 80%), and left ventricular stroke work index (LVSWI < 33 J/m²) following values reported in the literature.

Results: Seventy patients (mean age 60 ± 16 years; 60% males) were enrolled in the study. The median SOFA score was 10 [9–11], mean APACHE II score was 20 ± 6, mean SAPS II score was 45 ± 14, and median VIS was 40[20–59]. The lowest rate of SCM was observed in patients diagnosed using LVEF, at 16 (22.9%). For CPO-based diagnosis, 21 (31.3%) patients had SCM, while the SCM rate in the ACP group was 41 (61.1%). The highest rate of SCM was observed in the LVSWI group, at 22 (66.6%). LVEF, as the conventional criterion for SCM, was used as the standard for comparison. Fisher's exact test was performed to compare the incidence of SCM diagnosed using LVEF and CPO (*p* = 0.207) and LVEF and LVSWI (*p* = 0.375). Pearson's Chi-square test was used to compare LVEF and ACP (*p* = 0.914). These analyses revealed no significant correlation between SCM diagnosed using LVEF and other diagnostic criteria.

Conclusions: SCM rates vary depending on the diagnostic criteria used. Compared to the conventional LVEF-based criterion, all other methods showed higher and differing rates of SCM diagnosis, potentially reflecting clinically significant factors associated with clinical outcomes.

P210

Transthoracic echocardiography for hemodynamic evaluation and survival prognostication in patients with sepsis and septic shock
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Introduction: Transthoracic echocardiography (TTE) evolved as a readily available, reliable and precise method for hemodynamic evaluation in intensive care. In septic patients, cardiac involvement is evident but poorly described. This study aimed to evaluate cardiac function in septic patients and link it to clinical outcomes.

Methods: A single-center, prospective observational study was conducted at a tertiary reference hospital from March to September 2024. This study included mixed ICU patients with de novo cases of septic shock receiving at least 0.1 mcg/kg/min norepinephrine infusion. TTE was performed within 48 h of enrolment to assess left ventricular ejection fraction (LVEF), cardiac output (CO), cardiac index (CI), cardiac power output (CPO), cardiac power index (CPI), and afterload-related cardiac performance (ACP). Twenty-eight days of survival was selected as a clinical outcome. ROC AUC analysis was performed to determine which TTE parameters can be employed as survival predictors.

Results: Seventy patients were included in the analysis, comprising a critical care cohort with a mean age of 62 ± 16 years, APACHE II score of 20 ± 6, and SOFA score of 10 ± 2. The TTE parameters measured were LVEF (55 ± 11)%, CO (5.0 [3.8–6.8]) L/min, CI (2.1 [1.7–2.9]) L/min/m², CPO (0.86 [0.71–1.21]) W, CPI (0.39 [0.31–0.50]) W/m², and ACP (78 ± 13)%. In ROC-AUC analysis LVEF (*p* = 0.716), CPO (*p* = 0.087), CPI (*p* = 0.117), ACP (*p* = 0.102) and CI (*p* = 0.06) were not significant determinants of survival. However, CO emerged as a determinant of survival, with a ROC AUC of 0.647 (95% CI: 0.51–0.78, *p* = 0.039).

Conclusions: TTE is a valuable tool for assessing hemodynamic parameters in septic patients, with TTE-derived CO showing promise for survival prognostication. Further analysis with a larger sample

group is needed to validate these findings and assess the potential of other TTE diagnostic applications.

P211

Safety, tolerability and pharmacokinetics/-dynamics of the dipeptidyl peptidase 3 antibody procizumab in a first-in-human, placebo-controlled, phase 1 trial

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Introduction: Dipeptidyl peptidase 3 (DPP3), an aminopeptidase that degrades several key cardiovascular mediators, may induce and exacerbate hemodynamic instability during cardiogenic shock. Procizumab is a first-in-class humanized monoclonal antibody, which inhibits DPP3. In placebo-controlled preclinical shock models, procizumab was shown to increase angiotensin metabolite levels, and improve cardiovascular function as well as survival. The present trial evaluated the safety, tolerability, and pharmacokinetics (PK)/-dynamics of procizumab in a first-in-human phase 1 trial.

Methods: Twenty-four healthy male volunteers were enrolled in a randomized, double-blind, placebo-controlled phase 1 trial. Subjects received placebo or one of three doses of procizumab (n=6 per group). Subjects were monitored clinically for 24 h after drug administration, as well as 6 follow-up visits performed during a 28 day period (Figure).

Results: Procizumab exhibited an excellent safety profile, demonstrated by local tolerability, vital signs, laboratory parameters and ECGs. No serious adverse events (SAEs) occurred during the trial. In all three dose groups, the C_{max} was attained at or shortly after termination of infusion. Dose-proportional increases in C_{max} and AUC_{0-∞} were observed. A small volume of distribution indicates that procizumab predominantly remains within the circulation. Procizumab exhibited a short half-life compared to other monoclonal IgG antibodies (terminal elimination T_{1/2} of ~53 h in the high dose group). This short half-life could be a characteristic of the antibody or caused by target mediated drug disposition (TMDD), with procizumab-DPP3 complexes tending towards rapid clearance kinetics, as known for DPP3.

Conclusions: Administration of procizumab was safe and well tolerated in healthy males. Results of this trial will be followed up by a phase 1B safety and optimal dose finding trial in cardiogenic shock patients, which will be initiated in early 2025.

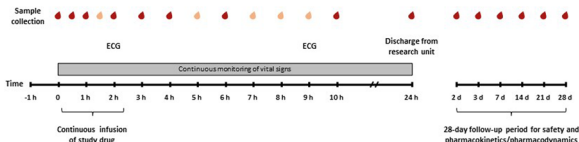


Figure (abstract P211) Summary of all procedures performed in the first-in-man trial. ECG=electrocardiography, h=hour, d=day

P212

Angiotensin II in the management of refractory septic shock

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Introduction: Septic shock may not respond to a single vasopressor. Angiotensin II (AT-II) has been studied as vasopressor in shock since 1960s. ATHOS-3 study evidenced how shocked patients treated with AT-II significantly reached aimed pressure response significantly than controls [1]. Post-hoc analyses of the ATHOS-3 study shed a light on possible best subgroup responders. We aimed at evaluating how AT-II treatment may reduce other vasopressor doses (norepinephrine and vasopressin) and improve hemodynamic profile within 48 h in patients with refractory septic shock.

Methods: Adult septic shock patients, belonging to the REINSURE-ARDS Registry (Prog 1946CESC, Prot 72485), refractory to 2 vasoactive agents, despite adequate volume resuscitation and without cardiac dysfunction, were eligible to be treated with AT-II [GIAPREZA, Viatris]. We asked CTAV authorization for every patient. Patients with arterial ischemic syndrome and/or venous thrombosis within 6 months were excluded. We recorded clinical and hemodynamic variables (TPTD, VolumeView, Edwards Lifesciences) before AT-II infusion, after 2 h from infusion start and then every 8 h.

Results: 9 patients (6 men) were included in this study, median age 64 (58,74) yrs, median BMI 28.3 (24.1,34.2), median APACHE II score 22 (20,30); 5 patients had a history of hypertension and 5 were treated with ACEi/ARB drugs. All patients had sepsis related AKI. AT-II infusion was related to a significant decrease in NE and VP dose (p=0.007 and 0.247, respectively); MAP and SVRI increased (p=0.008 and 0.071, respectively), while HR decreased (p=0.305). Ea increased during the first hrs of treatment, decreasing significantly after 24 h (p=0.025) (Figure). No adverse effects were recorded.

Conclusions: Beside obvious limitations of our study, AT-II demonstrated to act as an interesting tool to treat refractory septic shock and treat adrenergic overload side-effects.

References

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2. Legrand et al. Crit Care 2024;28:389

Variable	G0-T0	G0-T1	G0-T2	G0-T3	G1-T1	G1-T2	G1-T3	p value *
Angiotensin II mg/kg/min	0.00 (0.00, 0.00)	30 (20, 30)	30 (10, 30)	15 (5, 35)	25 (15, 38)	30 (20, 30)	20 (10, 30)	0.014
Norepinephrine mg/kg/min	0.60 (0.60, 0.80)	0.40 (0.30, 0.40)	0.35 (0.30, 0.30)	0.30 (0.30, 0.30)	0.45 (0.25, 0.60)	0.18 (0.05, 0.35)	0.25 (0.15, 0.35)	0.007
Vasopressin U/min	0.03 (0.03, 0.04)	0.03 (0.01, 0.04)	0.02 (0.00, 0.03)	0.00 (0.00, 0.00)	0.03 (0.00, 0.04)	0.02 (0.00, 0.03)	0.01 (0.00, 0.02)	0.347
MAP (mmHg)	3.3 (2.7, 4.4)	3.4 (3.4, 4.7)	3.4 (3.1, 5.1)	3.0 (4.0, 5.2)	3.9 (2.8, 4.7)	3.9 (3.3, 5.5)	4.9 (4.1, 6.0)	0.025
Ea (mmHg)	1.4 (0.6, 1.5)	1.5 (1.3, 1.5)	1.8 (1.2, 3.1)	1.6 (1.5, 1.8)	1.4 (1.1, 1.8)	1.4 (1.0, 1.8)	1.1 (0.8, 1.2)	0.008
HR (b/min)	119.0 (107.0, 128.5)	107.5 (96.5, 127.0)	118.0 (99.0, 136.0)	115.0 (115.0, 115.0)	97.0 (89.0, 116.0)	102.5 (96.0, 110.0)	103.0 (96.0, 108.5)	0.305
MAP mmHg	66.5 (57.0, 71.5)	80.5 (65.0, 87.5)	78.0 (75.0, 86.0)	82.0 (80.0, 94.0)	71.0 (65.0, 80.0)	82.5 (66.0, 86.0)	69.0 (66.0, 80.5)	0.008
SVI ml/m ² /min	37.0 (31.0, 41.0)	40.0 (29.0, 43.0)	32.0 (29.0, 48.0)	43.0 (36.0, 45.0)	39.5 (29.5, 48.5)	38.0 (32.0, 48.0)	42.0 (37.0, 53.0)	0.324
SVRI (dynes x cm ⁵)	942.0	1,132.0	1,372.0	1,024.0	1,213.5	1,333.0	982.0	0.071

Figure (abstract P212) Angiotensin II infusion related changes

P213

Angiotensin 2 in septic shock, non-responders fail to RAAS?

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Introduction: A post-hoc analysis of the ATHOS 3 trial showed an association with a high baseline renin and a reduction in mortality for patients treated with angiotensin 2 [1]. We assessed the renin-angiotensin-aldosterone-system (RAAS) in patients treated with angiotensin 2.

Methods: Patients with septic shock and a noradrenaline dose >0.2 µg/kg/min were included and treated with angiotensin 2 for at least 24 h. Blood samples were drawn before start, after 3 and 24 h. A target MAP of ≥65 mmHg was maintained and patients were