Медичні науки

Asimionese Vitalii
Emergency Medicine Paramedic, Biology Laboratory Engineer
Yuriy Fedkovych Chernivtsi National University
ORCID: 0009-0002-7391-5829

PREHOSPITAL MANAGEMENT OF TYPE 1 DIABETES MELLITUS: A CRITICAL ANALYSIS OF COMMON DIAGNOSTIC AND THERAPEUTIC ERRATA

Summary. *The subject of the present research is the specificity of the prehospital management of acute life-threatening conditions of diabetic ketoacidosis (DKA) and severe neuroglycopenia in patients with type 1 diabetes mellitus (T1DM) within the framework of the American emergency medical services (EMS) system. The clinical situation is analyzed wherein the provision of urgent care under conditions of information deficit and severe time constraints becomes a catalyst for severe iatrogenic complications. In focus are the cognitive biases of providers, the phenomenon of diagnostic obfuscation and systemic contradictions between the complex transcellular biochemistry of endocrine crises and the rigid architecture of field operational algorithms. The objective of the work consists in conducting a comprehensive, pathophysiologically substantiated critical analysis of the most common diagnostic and therapeutic errors committed by emergency medical crews. The research seeks to deconstruct the mechanisms of iatrogenesis occurrence to substantiate the necessity for the profound humanization and conceptual optimization of local EMS protocols. To realize the set objective, an integrative critical review design was applied, combining elements of the retrospective analysis of clinical guidelines and the assessment of epidemiological metrics. Leading medical repositories (PubMed, Cochrane Library, Scopus) served as the information base, encompassing*

specialized publications for the 2010-2024 period. The research synthesizes current consensuses of US associations (ADA, NAEMSP, ACEP) and representative data of the National Emergency Medical Services Information System (NEMSIS). The extracted data array was subjected to thematic content analysis with the stratification of identified deviations into three fundamental categories: diagnostic distortions, osmotic catastrophes of fluid resuscitation and pharmacotherapeutic incidents. The research results convincingly demonstrate that the high frequency of complications in patients with T1DM is a logical consequence of systemic algorithmic dissonance rather than the individual incompetence of medical professionals. To break the vicious cycle of iatrogenesis, the mandatory integration of expanded screening (POCT) into the basic triage protocol, the categorical rejection of the prehospital application of exogenous insulin and the transition to a restrictive, physiologically calibrated tactic of infusion therapy and the gentle alleviation of hypoglycemia (D10W) are critically necessary. The global transformation of prehospital endocrinology requires the abandonment of the rigid "cookbook medicine" concept in favor of adaptive guidelines returning to paramedics the right to scientifically substantiated clinical judgment.

Key words: *type 1 diabetes mellitus, emergency medical services, prehospital care, diabetic ketoacidosis, severe hypoglycemia, iatrogenic complications, fluid resuscitation.*

Introduction. Type 1 diabetes mellitus (T1DM) represents a chronic autoimmune disease characterized by absolute insulinopenia and the total dependence of the patient on exogenous hormone administration. This determines extreme metabolic lability wherein the slightest deviations in the insulin therapy regimen, intercurrent infections or physiological stress are capable of provoking life-threatening acute glycemic excursions. Under conditions of the complete absence of endogenous regulation of carbohydrate metabolism patients with

T1DM continuously balance on a fine line between two polar critical states. These are profound neuroglycopenia leading to irreversible damage to cerebral structures and diabetic ketoacidosis (DKA) accompanied by a cascade of severe disturbances including cellular dehydration, electrolyte imbalance and systemic acidemia.

In the United States of America, the prehospital stage of medical care provision plays a fundamental and often determining role in the survival trajectory of such patients. Emergency medical services (Emergency Medical Services, EMS) annually assume a colossal burden of diabetic catastrophes. According to epidemiological reports of the US Centers for Disease Control and Prevention (CDC) and current extractions from the National Emergency Medical Services Information System (NEMSIS) severe hypoglycemias and hyperglycemic crises account for over 400 000 paramedic calls per year [1]. Approximately 1.9 million Americans live with verified T1DM. Despite the widespread use of continuous glucose monitoring (CGM) systems and hybrid insulin pumps, the frequency of DKA-related hospitalizations in the USA has increased by almost 30% over the last decade, particularly among vulnerable demographic groups.

The specificity of the American EMS model dictates the necessity of making lightning-fast clinical decisions under conditions of severe time pressure and information deficit. The prehospital management of diabetic crises is burdened by the phenomenon of diagnostic obfuscation. The clinical manifestation of DKA including diffuse abdominal pain, intractable vomiting, tachypnea (Kussmaul breathing) and progressive depression of consciousness frequently mimics acute surgical pathology, sepsis or toxic damage to the central nervous system [2]. This polymorphism of symptomatology frequently disorients emergency crews leading to erroneous triage classification. The absence of routine measurement of the capillary glucose level in patients with an altered mental status remains one of the most fatal cognitive errors at the prehospital

stage triggering a chain of incorrect and potentially disastrous therapeutic strategies.

Besides diagnostic traps a colossal threat to the patient is posed by iatrogenic complications induced by incorrect pharmacotherapy and inadequate infusion tactics in field conditions. The aspiration of providers towards the immediate correction of hyperglycemia often prevails over the understanding of the complex physiology of osmotic shifts. The premature administration of exogenous insulin without instrumental verification of the serum potassium level is capable of provoking a rapid intracellular shift of cations which leads to refractory ventricular arrhythmias. Similarly excessive or overly aggressive fluid resuscitation with hypotonic crystalloids especially in pediatric practice creates a significant osmotic gradient forcing the transcapillary transition of fluid into the interstitial space of the brain and provoking severe cerebral edema [3]. Thus, saving the patient from hypovolemic shock the medical worker risks becoming a catalyst for severe neurological deficit.

On the other side of the spectrum lies severe hypoglycemia requiring no less meticulous precision in the dosing of prehospital interventions. The American plan of applying high doses of concentrated dextrose (in particular the D50W protocol) is currently undergoing strict revision. The empirical bolus administration of hyperosmolar solutions not only causes severe phlebitis during extravasation, but also provokes a sharp rebound release of counterregulatory hormones. This inevitably leads to subsequent refractory hyperglycemia closing the vicious circle of metabolic instability. Furthermore, the unjustified application of oral gels in patients with a suppressed gag reflex against the background of profound neuroglycopenia dramatically increases the risk of aspiration pneumonitis [5].

In the context of the described landscape of clinical challenges this article is directed at a comprehensive analysis of the most common and critical errors committed at the prehospital stage of managing patients with type 1 diabetes

mellitus. The research is designed to systematize the etiopathogenetic mechanisms of iatrogenic complications and substantiate the necessity of optimizing local EMS protocols to minimize preventable lethality and invalidization.

Literature Review. The epidemiological basis of the problem is detailedly illuminated in retrospective analyses and large-scale population studies. Thus, the works of Wang et al. (2015) and earlier fundamental observations of Ginde et al. (2008) emphasize the progressing burden of hyperglycemic crises and severe hypoglycemias on the emergency medical services (EMS) system of the USA [5]. They also accentuate attention on the high frequency of life-threatening conditions among vulnerable population groups [6]. At the same time, despite the regular publication of detailed clinical standards, the extrapolation of these inpatient guidelines to the severe conditions of the prehospital stage remains a subject of acute academic debates. This generates a significant array of literature devoted to iatrogenic errors and cognitive biases of paramedics.

A substantial stratum of studies is focused on the problem of diagnostic obfuscation and triage errors. Hallyburton (2022) in his conceptual analysis of the diagnostic obscuration (diagnostic overshadowing) phenomenon visually demonstrates how somatic and neurological symptoms of patients (including the syndrome of altered mental status in T1DM) are critically frequently interpreted incorrectly [7]. The literature emphasizes that the absence of routine capillary blood screening in patients with tachypnea or an unclear genesis of abdominal pain is a powerful trigger for making erroneous preliminary diagnoses such as sepsis or acute surgical abdomen which fundamentally distorts the vector of subsequent intensive therapy and patient routing.

The most polemical aspect of the prehospital management of diabetic ketoacidosis (DKA) remains infusion therapy balancing on the verge between necessary volemic resuscitation and the risk of inducing cerebral edema. This problematic is deeply researched in pediatric emergency medicine. The classic

works of Glaser et al. (2001) for the first time outlined the specific risk factors of osmotic shifts, however, it was exactly the large-scale multicenter study of the PECARN clinical network under the leadership of Kuppermann et al. (2018) that put an end to the debates about the rate and composition of infusion media [8, 9]. The authors convincingly proved that overly aggressive hydration with crystalloids at the prehospital stage triggers a fatal transcapillary gradient. In the context of American EMS-protocols this led to the revision of the "load and go" tactic with uncontrolled bolus fluid administration in favor of strictly titrated infusion directed exclusively at the restoration of peripheral perfusion.

The vector of pharmacotherapeutic errors in the literature is most vividly represented by the analysis of the consequences of premature insulin administration. The fundamental works of Kitabchi et al. (2009) on the pathophysiology of hyperglycemic crises postulate a total intracellular potassium deficit in DKA despite possible normo- or hyperkalemia in the plasma [10]. Relying on this physiological paradigm, the American Diabetes Association (ADA) in the current Standards of Medical Care (2024) strictly regulates the sequence of metabolic correction [11]. This provides the scientific substantiation of the absolute prohibition on the prehospital administration of exogenous insulin. Field insulin therapy without preliminary laboratory verification of kalemia inevitably provokes a rapid intracellular shift of cations converting endocrine instability into fatal arrhythmogenic events.

No less acute stands the issue of arresting severe neuroglycopenia. The protocol of bolus administration of a 50% dextrose solution (D50W) historically rooted in the USA is subjected to rigid well-reasoned obstruction. The comparative studies of Weant et al. (2021) as well as earlier clinical works of Howell & Guly (1997) analyzing the effectiveness of the prehospital application of carbohydrates and glucagon convincingly demonstrate the risks of aggressive pharmacotherapy [12, 13]. The hyperosmolar load of D50W induces a pronounced rebound release of counterregulatory hormones which leads to

refractory posthypoglycemic hyperglycemia and severe tissue damages during extravasation. The modern scientific consensus shifts in favor of the fractional administration of a 10% dextrose solution (D10W) which provides a comparable recovery time of mental status with a radical reduction of iatrogenic risks.

Summarizing the analyzed data array, it is possible to state that modern medical literature provides an exhaustive pathophysiological base for the safe arrest of acute diabetic conditions. Nevertheless, the critical gap identified during the review consists in the dissonance between the accumulated evidence base and the rigidity of local prehospital protocols which dictates the need for the translation of this knowledge into updated directives for paramedics.

Materials and Methods. The relevance of the present research is dictated by the steady growth of the burden of acute diabetic cases on the prehospital emergency medical services (EMS) system in the United States of America. Despite the widespread expansion of continuous glucose monitoring (CGM) systems and hybrid closed loops the frequency of paramedic calls regarding life-threatening glycemic excursions remains critically high [14]. The prehospital stage represents a unique and extremely vulnerable therapeutic window where the cognitive load on the medical worker multiplied by the time deficit and the limitedness of the diagnostic arsenal creates a fertile ground for fatal errors. The incorrect interpretation of compensatory mechanisms in diabetic ketoacidosis (DKA) or the aggressive empirical therapy of severe neuroglycopenia are capable of triggering a cascade of irreversible physiological disturbances even before the arrival of the patient at the emergency department (ED). Consequently, the critical audit of prehospital care provision patterns and the identification of systematic iatrogenic errors is a fundamental task the solution of which determines the reduction of preventable lethality and severe neurological deficit among patients with type 1 diabetes mellitus.

The scientific novelty of the given work consists in shifting the analytical focus from the traditional in-hospital (ICU) stage of intensive care to the

prehospital period which in modern literature is frequently considered merely as transit. An attempt has been undertaken to conduct a deep pathophysiological systematization of iatrogenic errors. The research synthesizes disparate data from toxicology, pediatric emergency medicine and endocrinology forming a unified, structured framework of prehospital therapeutic failures. Such an interdisciplinary approach allows conceptualizing the errors of paramedics as a consequence of a complex dissonance between the basic physiology of osmotic and electrolyte shifts and outdated or rigid local emergency protocols.

To realize the set objective the design of an integrative critical review with elements of the retrospective analysis of clinical guidelines was chosen [15]. Leading international repositories of medical literature including PubMed/MEDLINE, Cochrane Library, Scopus and Web of Science served as the information base of the research. The search was conducted across publications indexed for the period from 2010 to 2024 which allowed encompassing the modern stage of evolution of American EMS-protocols and pharmacological systems. The search strategy was based on the use of combinations of standardized MeSH (Medical Subject Headings) terms and free keywords: "type 1 diabetes mellitus", "emergency medical services", "prehospital care", "diabetic ketoacidosis", "severe hypoglycemia", "iatrogenic disease", "medical errors" and "fluid resuscitation". The inclusion criteria encompassed original cohort studies, systematic reviews, randomized controlled trials (in their presence in the context of EMS) as well as official consensus statements and clinical guidelines of specialized US organizations: the American Diabetes Association (ADA), the National Association of EMS Physicians (NAEMSP) and the American College of Emergency Physicians (ACEP). Besides, aggregated epidemiological reports of the National Emergency Medical Services Information System (NEMSIS) providing representative statistics on real patterns of calls and conducted interventions "in the field" were integrated into the analysis. The exclusion criteria sifted out publications focused

exclusively on the inpatient management of diabetic crises, studies on animal models as well as works considering the pathology of type 2 diabetes mellitus without intersection with the mechanisms of absolute insulinopenia.

The process of data extraction was conducted by means of independent screening of titles, abstracts and full-text versions of the selected articles. The synthesis of the extracted information was carried out according to the method of thematic content analysis during which the entire array of identified prehospital errors was stratified into three fundamental categories: diagnostic obfuscation and triage distortions, iatrogenic catastrophes of fluid resuscitation (osmotic shifts) and pharmacotherapeutic incidents (the incorrect application of exogenous insulin, dextrose and glucagon). The given methodological matrix provided a rigid structural framework for subsequent detailed discussion in the body of the article.

The limitations of the present research are conditioned by the specificity of the analyzed subject area and the chosen methodological design. Firstly, the extrapolation of data obtained from the NEMSIS national registry is associated with the inherent risk of information bias since the quality of the retrospective documentation of paramedics is subject to the influence of the human factor and coding errors. Secondly, the prehospital system of the USA is characterized by pronounced decentralization and unprecedented heterogeneity of local protocols at the level of individual states and counties which complicates the derivation of universal statistical regularities. Finally, in view of strict ethical barriers and the specificity of emergency care provision, an acute deficit of prospective double-blind placebo-controlled trials evaluating specific interventions for T1DM directly "in the field" is observed in modern literature which forces researchers to rely predominantly on observational data and consensus opinions of experts.

Results and Discussions. The conducted integrative analysis of prehospital metrics and clinical outcomes revealed unsatisfactory results. Errors in the urgent management of patients with type 1 diabetes mellitus (T1DM) are

not isolated casuistic incidents, but represent systemic failures deeply rooted in the architecture of the American emergency medical services (EMS). The synthesis of data from the NEMSIS registry and current medical literature demonstrates that the iatrogenic alteration of metabolic status occurs at the intersection of cognitive biases of providers and the rigidity of local operational protocols. The author classified the identified deviations into a single continuum of therapeutic failures which visually illustrates how primary diagnostic inaccuracy triggers a cascade of erroneous pharmacological and infusion interventions exponentially increasing the risk of profound neurological deficit or fatal arrhythmogenic events even before the moment the patient crosses the threshold of the emergency department (ED) (see: Figure 1. Cascade of therapeutic failures in prehospital T1DM management).

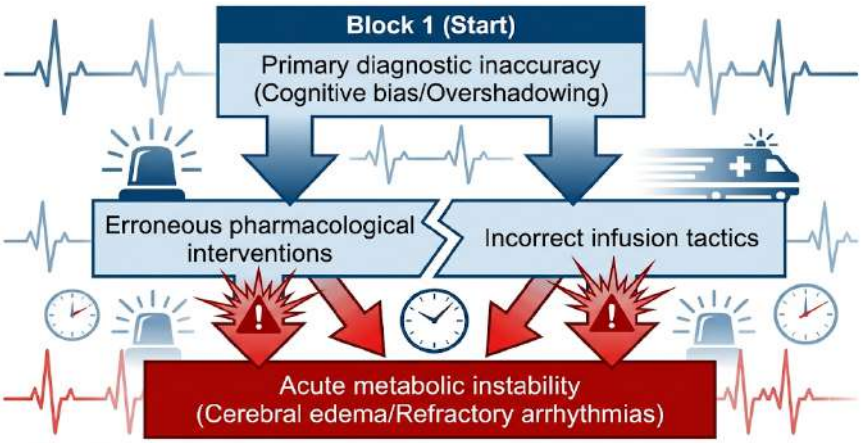

Fig. 1. Cascade of therapeutic failures in prehospital T1DM management

For the adequate contextualization of the obtained results, it is necessary to integrate an understanding of the extreme operational environment in which American paramedics function. The provision of care "in the field" is associated with a colossal cognitive load, an acute deficit of anamnesis data and the imperative of immediate life-saving action. Under these conditions medical workers inevitably resort to heuristic mechanisms of decision-making which makes them vulnerable to the phenomenon of premature closure and diagnostic overshadowing [16]. Encountering a patient with depressed consciousness,

tachypnea or intractable vomiting the paramedic often follows the path of least resistance fitting the clinical picture to more frequent and visually familiar patterns: severe toxicological intoxication, septic shock or acute surgical pathology. The human factor acts here not as a marker of unprofessionalism but as a logical consequence of the absence of rigid algorithmic triggers for the mandatory conduction of point-of-care testing (POCT) in every patient with an altered mental status. Moreover, the stressfulness of the situation is exacerbated by the frequent presence of panicking witnesses who can provide a fragmentary or distorted anamnesis leading the diagnostic search in a false direction (see: Figure 2. Triage overshadowing of acute glycemic excursions).

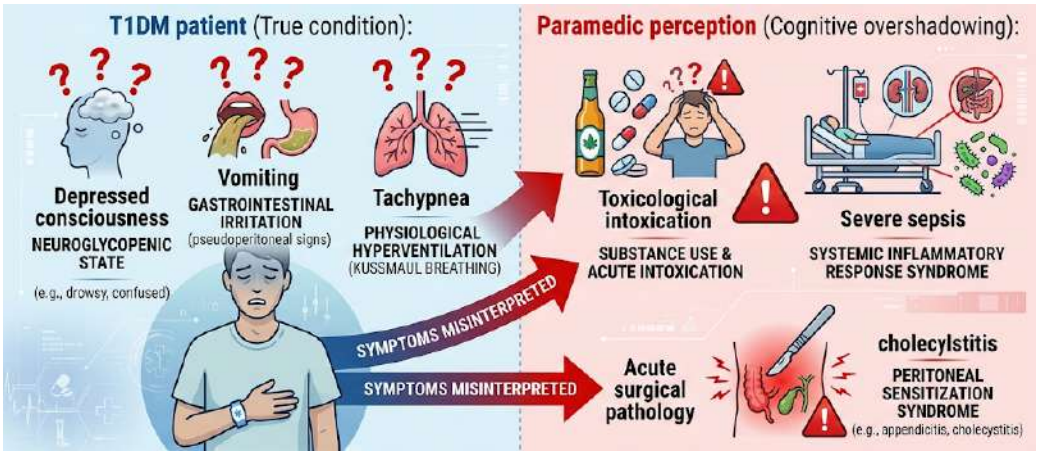

Fig. 2. Triage overshadowing of acute glycemic excursions

The fundamental dissonance identified during the research lies in the deep conflict between the complex biochemical nature of acute glycemic excursions and the linear, immediate result-oriented logic of prehospital protocols. The traditional training of EMS specialists is based on the principles of aggressive resuscitation: the rapid restoration of circulating blood volume in profound shock, the immediate relief of pain syndrome, the urgent normalization of vital signs. However, the pathophysiology of absolute insulinopenia dictates a diametrically opposite approach. Diabetic ketoacidosis (DKA) and severe neuroglycopenia require not a forced, but a meticulously titrated, prolonged intervention. While emergency departments possess the full arsenal of laboratory diagnostics

(including arterial blood gas analysis, precise determination of serum osmolarity and electrolyte level) EMS crews are forced to make decisions relying exclusively on the readings of portable glucometers which inherently possess significant instrumental error at the extreme boundaries of the glycemic spectrum [17]. The aspiration of the paramedic to "correct" the numbers on the display here and now with the help of hyperosmolar boluses of concentrated dextrose or the premature administration of exogenous insulin fatally ignores the complex dynamics of osmotic gradients and intracellular electrolyte shift. It is exactly this translational chasm between molecular endocrinology and field disaster medicine that generates the majority of the analyzed complications.

The deconstruction of the identified therapeutic failures allowed highlighting three critical zones of prehospital risk each of which possesses its own unique pathogenetic mechanism of iatrogenic. Firstly, this is the stage of primary contact and triage where differential diagnostic errors critically prolong the time until the initiation of pathogenetically substantiated therapy. Secondly this is the zone of fluid resuscitation concealing within itself a permanent threat of osmotic demyelination and cerebral edema given an incorrect choice of infusion strategy or crystalloid administration rate. Thirdly this is the area of pharmacotherapeutic incidents including the empirical unjustified or technically incorrect application of insulin, dextrose and glucagon. Finally synthesizing the obtained data, the author identified systemic algorithmic contradictions impeding the full-fledged integration of advanced scientific-medical experience into the routine clinical practice of EMS.

A detailed analysis of each of these aspects presented in the subsequent subsections forms a comprehensive evidence base for the necessary transformation of prehospital endocrinological care (see: Table 1. Systematization of prehospital iatrogenic risk zones and pathogenetic mechanisms).

Table 1

Systematization of prehospital iatrogenic risk zones and pathogenetic mechanisms

Risk zone	Predominant errors	Pathogenetic mechanism of iatrogenesis	Systemic algorithmic contradiction
Primary contact and triage	Diagnostic overshadowing and misattribution of symptoms; lack of point-of-care testing (POCT) application	Misinterpretation of Kussmaul breathing as pulmonary pathology; pseudo-abdominal pain misdiagnosed as acute surgical etiology	Linear algorithm logic vs polymorphic metabolic presentation
Fluid resuscitation	Aggressive, rapid bolus application of isotonic crystalloids; use of hypotonic solutions	significant osmotic gradient; rapid serum osmolarity reduction forcing brain water influx; cerebral edema herniation	Resuscitative logic (shock management) vs titrated physiological replacement
Pharmacothera peutic incidents	Premature insulin administration; excessive hyperosmolar dextrose bolus application	Insulin-induced rapid intracellular potassium shift causing hypokalemia; refractory ventricular arrhythmias; rebound counterregulatory hormone release	"Cookbook medicine" directives vs complex metabolic homeostasis

1. Diagnostic obfuscation and triage misclassification in acute glycemic excursions:

The prehospital stage of emergency medical care provision represents a clinical environment characterized by a high degree of uncertainty, a time deficit and the limitedness of diagnostic instrumentation. In this context acute glycemic excursions in patients with type 1 diabetes mellitus (T1DM) often become a trigger for the phenomenon of diagnostic obfuscation. This is a cognitive bias wherein a true metabolic catastrophe is masked under a spectrum of more conventional urgent conditions. The fundamental problem of the American EMS system is the high frequency of triage misclassification conditioned by the polymorphism and non-specificity of the clinical picture of diabetic ketoacidosis (DKA) and severe neuroglycopenia. Paramedics working under conditions of colossal stress and an information vacuum frequently become victims of the

availability heuristic and premature closure arresting obvious symptoms without the realization of their deep endocrine pathogenesis.

The most vivid illustration of diagnostic overshadowing is served by the clinical manifestation of severe DKA. Profound systemic acidemia associated with massive ketogenesis and electrolyte depletion frequently manifests with diffuse abdominal pain, muscle rigidity and intractable vomiting. Pathophysiologically this pseudoperitoneal syndrome is conditioned by transient mesenteric ischemia against the background of profound hypovolemia as well as local hyperproduction of prostaglandins [18]. However, under conditions of prehospital triage this symptomatology is critically frequently interpreted by emergency crews as a manifestation of an "acute abdomen" (appendicitis, pancreatitis, cholecystitis) or severe toxic infection. An erroneous surgical or infectious vector of the preliminary diagnosis leads to the unjustified administration of analgesics or centrally acting antiemetic drugs which can additionally depress the mental status of the patient erasing the true metabolic picture.

In parallel with gastrointestinal manifestations DKA is accompanied by the development of Kussmaul breathing – a deep, rhythmic tachypnea representing a physiological mechanism of compensatory respiratory alkalosis in response to a critical decrease in blood pH. The insufficient alertness of EMS providers often leads to the fatal misinterpretation of this compensatory pattern as a primary pulmonary pathology such as a severe attack of bronchial asthma, pulmonary embolism or pneumonia. In a number of cases especially in young patients with T1DM Kussmaul breathing is erroneously regarded as psychogenic hyperventilation within the framework of a panic attack. Undoubtedly panic attacks take place and cannot be ignored as a condition which is less critical than for example appendicitis. However, such a triage error entails catastrophic consequences. Attempts of the medicamentous arrest of a panic attack with sedative drugs (for example benzodiazepines) are capable of causing depression

of the respiratory center depriving the patient of a vital mechanism of carbon dioxide elimination and rapidly exacerbating decompensated metabolic acidosis (see: Figure 3. The adverse clinical cascade of Kussmaul breathing misinterpretation in the prehospital setting).

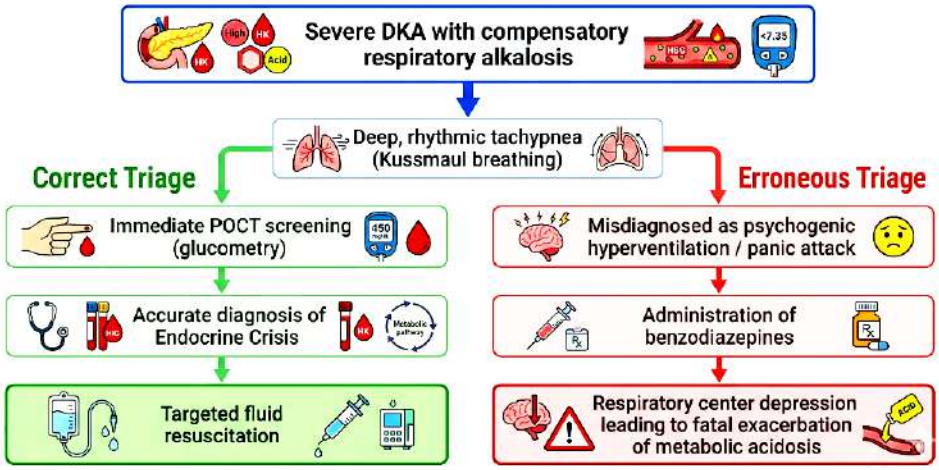

Fig. 3. The adverse clinical cascade of Kussmaul breathing misinterpretation in the prehospital setting

On the opposite pole of the glycemic spectrum is severe hypoglycemia the diagnostic complexity of which lies in the phenomenon of neuroglycopenia. The deficit of the energetic substrate in the tissues of the central nervous system provokes a cascade of neurological and psychiatric disturbances. This includes from aggression, delirium and psychomotor agitation to profound stupor and coma. Not infrequently neuroglycopenia is accompanied by a transient focal neurological deficit (hemipareses, aphasia) mimicking an acute cerebrovascular accident [19]. Under the conditions of American megalopolises, paramedics routinely classify patients with aggressive or inadequate behavior as persons with acute alcoholic or narcotic intoxication or as core psychiatric patients. Similarly focal symptomatology falsely activates the prehospital stroke protocol ("stroke alert"). As a result, the precious therapeutic window closes and neurons are subjected to the risk of irreversible damage due to delayed carbohydrate

resuscitation (see: Figure 4. Algorithmic divergence: empirical symptom-based triage versus POCT-guided assessment).

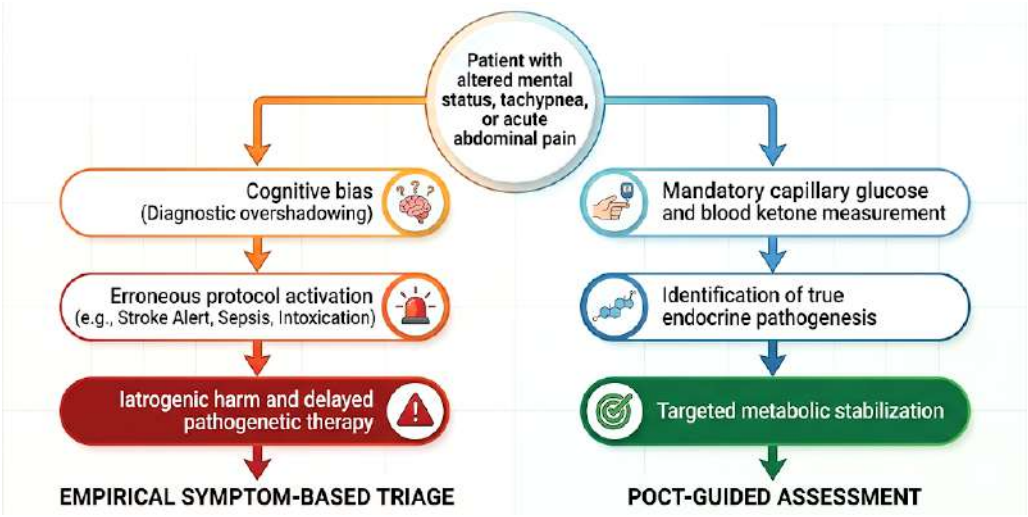

Fig. 4. Algorithmic divergence: empirical symptom-based triage versus POCT-guided assessment

Summarizing the mechanisms of diagnostic failures in acute diabetic catastrophes it is necessary to state that the root cause is the neglect of the imperative of immediate Point-of-Care (POCT) screening. The absence of mandatory measurement of the level of capillary glucose and blood ketones in absolutely every patient with an altered mental status, unclear tachypnea or an acute abdominal syndrome remains a yawning gap in operational algorithms. This diagnostic negligence no matter how psycho-emotionally substantiated it might seem in the chaos of field conditions triggers a chain reaction of iatrogenic complications. Overcoming diagnostic obfuscation requires the rigid incorporation of mandatory glucometry into the basic protocol of vital signs assessment (on a par with pulse oximetry). This will allow leveling the influence of subjective cognitive biases of paramedics and redirecting the triage vector into the channel of adequate pathogenetic therapy.

2. Iatrogenic pitfalls in fluid resuscitation and osmolar shifts:

One of the most complex and paradoxical tasks of the prehospital management of diabetic ketoacidosis (DKA) is the strategy of fluid resuscitation.

The pathophysiological cascade of absolute insulinopenia inevitably triggers massive glycosuria which acting as a powerful osmotic diuretic leads to the profound depletion of the intravascular and interstitial volumes. Patients with type 1 diabetes mellitus in a state of severe crisis can have a total fluid deficit reaching 10-15% of body weight which clinically manifests with pronounced tachycardia, hypotension and signs of tissue hypoperfusion. Under these conditions in emergency medical services (EMS) providers the basic resuscitative reflex naturally triggers -the immediate and aggressive replenishment of the circulating blood volume (CBV) with the aim of arresting hypovolemic shock. However, exactly at this stage a dissonance arises between the standard algorithms for the treatment of traumatic or septic hypovolemia and the subtle biochemical specificity of osmolar disturbances in diabetes.

The classic prehospital "load and go" strategy implying the forced bolus administration of large volumes of isotonic crystalloids (in particular 0.9% sodium chloride solution) conceals within itself a hidden iatrogenic threat in DKA. The intravascular bed of a patient with hyperglycemia is in a state of pronounced hyperosmolarity. Massive infusion not only provokes a sharp drop in serum osmolarity, but is also associated with the risk of developing severe hyperchloremic non-anion (non-anion gap) metabolic acidosis [20]. An excessive chloride load against the background of already existing ketonemia and reduced renal perfusion additionally depletes the buffer capacities of bicarbonate. Also, this prolongs the state of systemic acidemia and complicates the subsequent inpatient monitoring of the metabolic status. Moreover, an incorrect assessment of the degree of dehydration often leads to the erroneous use of hypotonic solutions at the prehospital stage which is categorically contraindicated in view of catastrophic consequences for transcapillary exchange (see: Figure 5. The intersection of DKA pathophysiology and prehospital fluid resuscitation pitfalls).

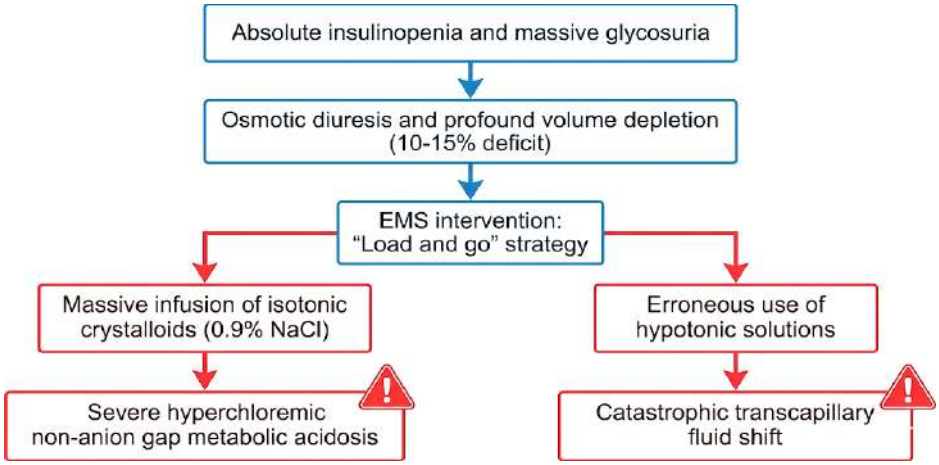

Fig. 5. The intersection of DKA pathophysiology and prehospital fluid resuscitation pitfalls

The most formidable, fatal and unfortunately often iatrogenically-induced complication of irrational infusion therapy is cerebral edema. This pathology possesses a high predilection to the pediatric population and young adults with T1DM acting as the leading cause of preventable mortality in the given cohort. The pathogenesis of diabetic cerebral edema is multifactorial; however a rapid osmotic shift is recognized as the key trigger. Normally the effective plasma osmolarity is calculated by the formula:

$$Osm_{eff} = 2[Na^{+}] + \frac{glucose}{18} \text{ (when measuring glucose in mg/dL) (1)}$$

In response to chronic hyperglycemia the neurons of the central nervous system accumulate idiogenic osmoles – specific intracellular molecules (for example taurine and myo-inositol) designed to prevent brain dehydration [21]. Aggressive and excessively fast prehospital infusion causes a sharp decrease in extracellular osmolarity. As a result, a powerful osmotic gradient directed inside the neurons is created which leads to a massive influx of free water into the brain cells, an increase in intracranial pressure and ultimately to transtentorial herniation (see: Figure 6. Pathogenesis of iatrogenic cerebral edema in DKA: the impact of rapid extracellular osmolarity reduction).

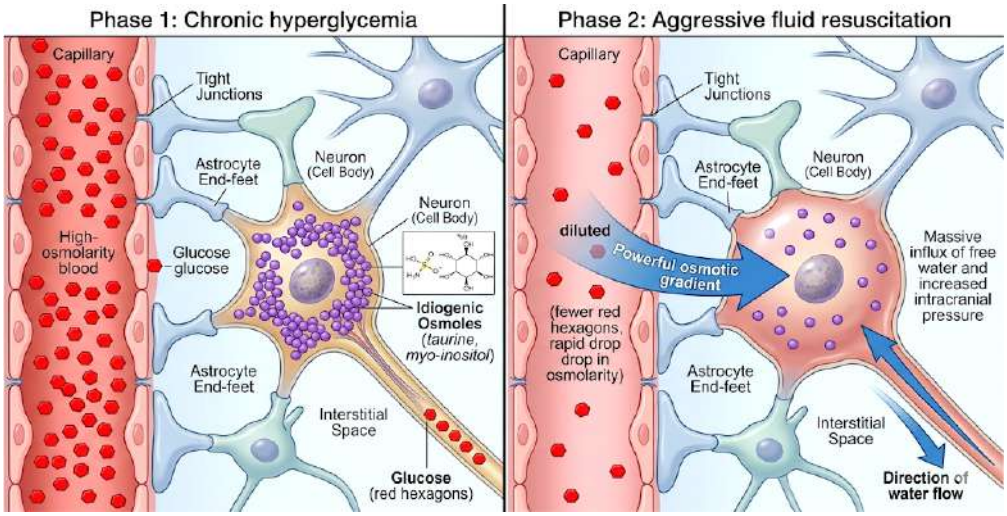

Fig. 6. Pathogenesis of iatrogenic cerebral edema in DKA: the impact of rapid extracellular osmolarity reduction

Modern clinical science dictates an urgent necessity for the conceptual revision of EMS infusion protocols. The focus must be shifted from the "normalization of numbers" to the maintenance of vital functions. Current consensus emphasizes that initial fluid resuscitation "in the field" must be strictly conservative and oriented exclusively at the restoration of adequate peripheral perfusion and hemodynamic stability and not at the immediate replenishment of the entire calculated fluid deficit. The fractional, titrated administration of isotonic crystalloids in small aliquots (for example, 10-20 ml/kg of body weight over the course of the first hour) with the continuous reassessment of mental status, pulse quality and capillary refill is recommended. Such a restrictive, physiologically substantiated strategy allows breaking the vicious circle of tissue hypoxia without subjecting the blood-brain barrier to shock osmotic impact (see: Table 2. Paradigm shift in prehospital fluid resuscitation strategies for acute glycemic excursions).

Table 2

Paradigm shift in prehospital fluid resuscitation strategies for acute glycemic excursions

Clinical parameter	Traditional resuscitative strategy	Modern pathogenetically calibrated tactic
Primary therapeutic goal	Immediate restoration of circulating blood volume and arresting shock	Gradual restoration of peripheral perfusion and hemodynamic stability
Infusion rate and volume	Forced bolus administration of large volumes	Fractional titrated administration in small aliquots of 10-20 ml/kg
Osmotic and metabolic impact	Rapid drop in serum osmolarity and depletion of bicarbonate buffer	Smooth transcapillary equilibration breaking the cycle of tissue hypoxia
Primary iatrogenic risks	Induction of fatal cerebral edema and hyperchloremic non-anion gap metabolic acidosis	Minimal risk of osmotic shock and blood-brain barrier damage

Thus, the prehospital management of volemic status in acute glycemic excursions represents a truly most complex therapeutic art requiring from the provider a deep understanding of the laws of osmosis and cellular metabolism. The minimization of iatrogenic traps is impossible without a rigid rejection of the template, aggressive infusion of fluids in favor of a personalized, pathogenetically calibrated tactic. Only conscious caution in the rates and volumes of infusion therapy is capable of protecting the vulnerable cerebral structures of the patient from irreversible iatrogenic damage at the transportation stage.

3. Pharmacotherapeutic misadventures: the perils of premature insulin administration and glucagon mismanagement:

In the arsenal of prehospital emergency medical services (EMS) pharmacological agents are frequently perceived as a universal instrument for the immediate correction of life-threatening conditions. In the context of type 1 diabetes mellitus (T1DM) the irresistible aspiration of paramedics to normalize critical indices of glycemia "here and now" generates one of the most dangerous

categories of iatrogenic complications – pharmacotherapeutic incidents. The fundamental problem lies in attempts of linear medicamentous intervention into a multicomponent system of disrupted homeostasis. The application of potent endocrine preparations under conditions of a field information vacuum, without preliminary laboratory verification of the electrolyte and metabolic status, turns life-saving therapy into a potentially fatal threat.

The most dramatic example of such a pharmacotherapeutic catastrophe is the premature administration of exogenous insulin at the prehospital stage to patients with suspected diabetic ketoacidosis (DKA). Of course, intuitively hyperglycemia requires insulin therapy, however the pathophysiology of DKA dictates an absolutely different algorithm of actions. Under conditions of absolute insulinopenia and systemic acidemia a massive efflux of potassium ions from the intracellular space into the extracellular one occurs. In combination with osmotic diuresis this leads to the total exhaustion of general potassium reserves in the organism (total-body potassium depletion) [22]. Nevertheless, due to the extracellular shift the primary level of serum potassium, measured upon admission, frequently turns out to be falsely normal or even paradoxically elevated.

If a paramedic, guided exclusively by the exorbitant indices of a portable glucometer, administers a bolus of insulin (subcutaneously, intramuscularly or intravenously) prior to the initiation of adequate infusion therapy and, what is critically important, prior to obtaining the results of a basic metabolic panel, he triggers a deadly plan. Insulin rapidly returns potassium cations back into the cells, simultaneously with this the correction of acidosis with infusion media exacerbates this intracellular transport. The result becomes sudden, profound and refractory hypokalemia. Clinically this iatrogenic electrolyte collapse manifests with life-threatening disturbances of the cardiac rhythm, up to fatal ventricular tachycardias and ventricular fibrillation. Exactly therefore, the modern consensus of American specialized associations (in particular, NAEMSP) impose an

absolute veto on the use of insulin in field conditions. The prehospital imperative always comes down to volemic resuscitation.

On the opposite end of the glycemic spectrum EMS crews face the problems of the medicamentous arrest of severe neuroglycopenia. When reliable intravenous access is impossible due to profound shock, peripheral vasoconstriction or psychomotor agitation of the patient, intramuscular administration of glucagon traditionally acts as the standard of rescue. However, the clinical efficacy of this counterregulatory hormone has rigid physiological limits, the ignoring of which leads to therapeutic failures. The mechanism of action of glucagon entirely depends on the presence of adequate glycogen reserves in the liver (the hepatic pool) [23]. In patients with T1DM, especially in a state of concomitant alcoholic intoxication, after exhausting physical exertions or against the background of preceding episodes of unrecognized hypoglycemia, these reserves can be totally depleted. In such pathophysiological realities the administration of glucagon turns out to be absolutely ineffective, leading to an unacceptable loss of the therapeutic window and the prolongation of the destructive ischemic impact on neurons.

Besides that, the pharmacodynamics of glucagon is inextricably linked with a powerful emetogenic effect. The induction of profuse vomiting in a patient with profound depression of consciousness, convulsive syndrome and an absent protective gag reflex colossally increases the risk of aspiration of gastric contents. This iatrogenic carelessness is capable of instantly transforming an endocrine emergency into the severest chemical pneumonitis. An adjacent problem of the prehospital pharmacotherapy of hypoglycemia remains the use of concentrated carbohydrate solutions. The historical adherence of American paramedics to the D50W protocol (50% dextrose) frequently turns into pronounced endothelial toxicity in the infusion zone and the induction of massive rebound hyperglycemia due to an inadequately high osmolar load. The active transition of advanced EMS systems to the use of a 10% solution (D10W) represents a necessary step towards

the humanization of prehospital care, minimizing post-aggressive metabolic "swings" (see: Table 3. Comparative analysis of hypertonic (D50W) and isotonic (D10W) dextrose solutions in prehospital hypoglycemia management).

Table 3

Comparative analysis of hypertonic (D50W) and isotonic (D10W) dextrose solutions in prehospital hypoglycemia management

Clinical parameter	Hypertonic strategy (D50W - 50% dextrose)	Isotonic strategy (D10W - 10% dextrose)
Osmotic load and safety	Extremely hyperosmolar; high risk of severe endothelial toxicity and phlebitis	Isotonic and physiologically calibrated; safe for peripheral veins
Counterregulatory hormone response	Sharp and pronounced rebound release; closing vicious circle of instability	Smooth transcapillary equilibration; minimized physiological stress
Post-resuscitation glycemic control	Significant risk of massive rebound hyperglycemia	Titrated recovery with predictable glycemic profile; humanized care approach

Consequently, the pharmacotherapeutic management of patients with T1DM at the emergency care stage requires innovations. Medical workers must realize that in an endocrinological urgency a scientifically substantiated abstention from aggressive medicamentous intervention frequently saves more lives than the reflexive use of potent preparations. The rejection of the prehospital application of exogenous insulin in hyperglycemic crises and the critical rethinking of indications for glucagon are the foundations of metabolic safety, guaranteeing the intact delivery of the patient to the stage of high-tech inpatient intensive care.

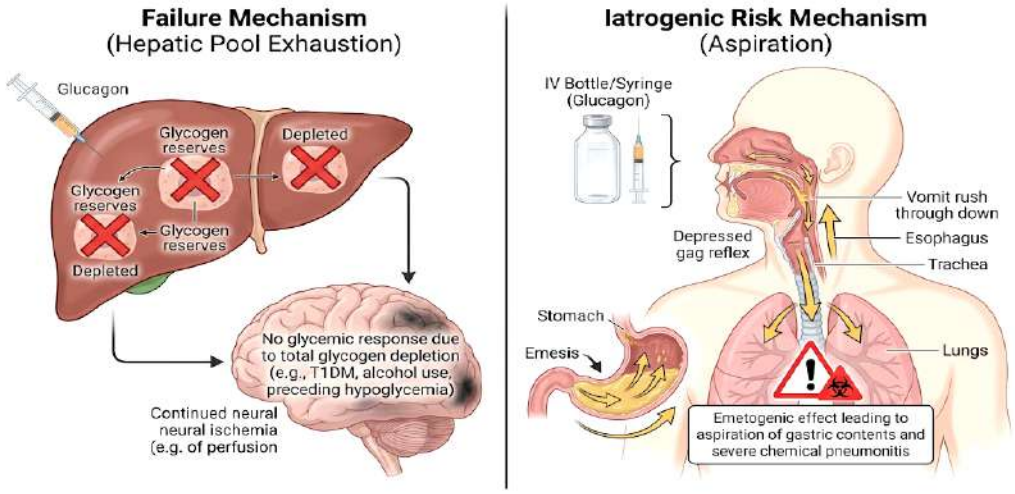

Fig. 7. Physiological constraints and iatrogenic risks of intramuscular glucagon administration in severe neuroglycopenia

4. Algorithmic discrepancies: bridging the gap between clinical guidelines and prehospital execution:

Interestingly, the paradox of modern prehospital management of patients with type 1 diabetes mellitus (T1DM) lies in a deep translational gap between academic endocrinology and field disaster medicine. On the one hand, there exist exhaustive, pathophysiologically calibrated clinical guidelines of specialized organizations (such as the American Diabetes Association – ADA) based on the principles of evidence-based medicine. On the other hand – the harsh operational reality of emergency medical services (EMS) where these ideal theoretical constructs are subjected to forced reduction and transformed into rigid, linear algorithms of actions. This process of translating scientific knowledge into practical directives for paramedics is inevitably accompanied by a loss of clinical nuances which in the context of the most complex metabolic catastrophes such as diabetic ketoacidosis (DKA) or severe neuroglycopenia becomes a catalyst for systemic therapeutic errors.

The architecture of the EMS system in the United States of America is characterized by unprecedented decentralization and heterogeneity. The absence of a unified federal standard of emergency care provision leads to the fact that local medical directors at the level of individual states or even counties

independently form and approve operational protocols (Standard Operating Procedures, SOPs). The consequence of this fragmentation is the coexistence of advanced agencies having already integrated modern concepts of conservative fluid resuscitation and the fractional administration of 10% dextrose with subdivisions continuing to practice outdated "cookbook medicine". Rigid, historically rooted algorithms prescribing aggressive bolus administration of isotonic crystalloids for any tachycardia or the mandatory use of hyperosmolar D50W for hypoglycemia tie the hands of clinicians. This deprives them of the possibility to adapt therapy to the individual physiological profile of the patient (see: Figure 8. The translational gap in prehospital T1DM management: academic guidelines versus EMS operational reality).

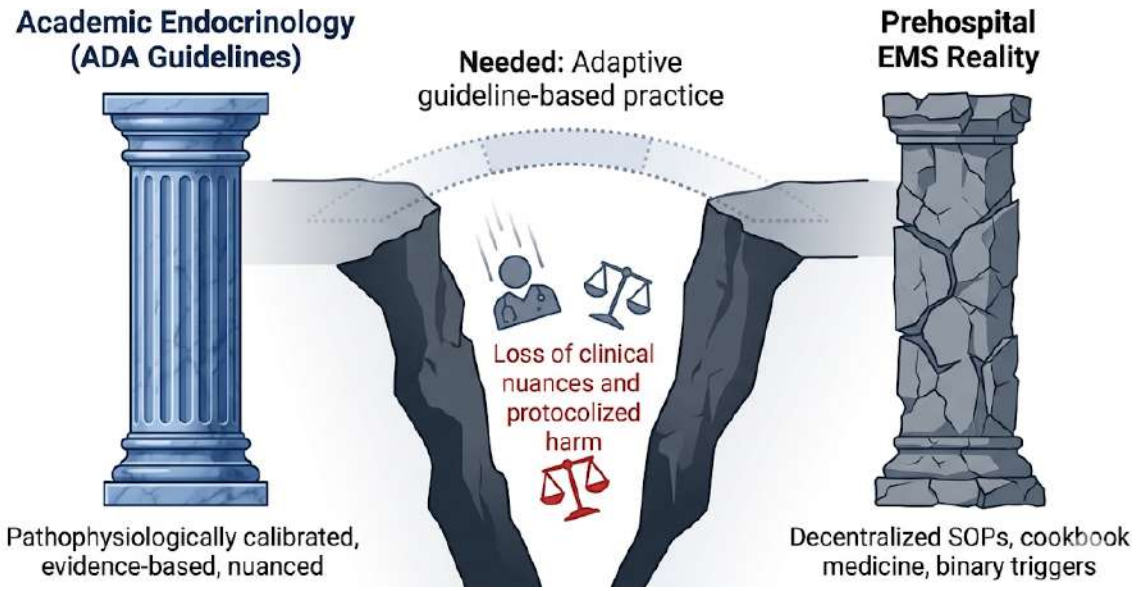

Fig. 8. The translational gap in prehospital T1DM management: academic guidelines versus EMS operational reality

The deep contradiction lies in the discrepancy of the diagnostic instrumentation to the conditions of decision-making. Inpatient clinical guidelines for the management of DKA are developed with the calculation for the controlled environment of the intensive care unit (ICU) where the physician relies on the continuous monitoring of arterial blood gases (ABG), the precise calculation of the anion gap and the hourly assessment of serum osmolarity. In

contrast to this, prehospital algorithms are frequently based on binary triggers (for example, "if the glucose level by glucometer > 400 mg/dL - initiates massive infusion"). Such binary logic fatally ignores the multiphasic dynamics of osmotic and electrolyte shifts. It algorithmically forces the paramedic to treat an isolated numerical indicator on the screen of a portable device and not a complex metabolic crisis which generates the truly lion`s share of iatrogenic complications (see: Table 4. Discrepancies in diagnostic instrumentation and decision-making logic between inpatient and prehospital settings).

Table 4

Discrepancies in diagnostic instrumentation and decision-making logic between inpatient and prehospital settings

Diagnostic environment	Inpatient intensive care unit (ICU)	Prehospital emergency medical services (EMS)
Primary diagnostic instrumentation	Continuous ABG monitoring, exact anion gap and hourly serum osmolarity calculation	Isolated portable glucometer readings with significant instrumental error
Decision-making logic	Multiphasic assessment of osmotic and electrolyte dynamics	Binary linear triggers initiating mandatory massive infusion
Protocol architecture	Pathophysiologically calibrated and nuanced clinical guidelines	Rigid and historically rooted cookbook medicine SOPs
Iatrogenic risk profile	Minimized through continuous physiological feedback and tailored interventions	High risk of protocolized harm due to forced linear medicamentous interventions

This algorithmic dissonance generates a cognitive and moral load on the providers of prehospital care themselves. Experienced paramedics possessing a deep understanding of the pathophysiology of T1DM frequently find themselves in a situation of a professional trap when following a local protocol potentially harms the patient (the phenomenon of "protocolized harm") while deviation from it threatens with serious legal and administrative sanctions [24]. A paradoxical clinical situation arises: the medical worker realizes the high risk of inducing cerebral edema during the rapid administration of fluid to a pediatric patient with unrecognized DKA, but is forced to scrupulously execute the directive of the

universal protocol for the management of undifferentiated hypovolemic shock. Such a dilemma of prehospital endocrinology requires an honest recognition of this conflict and a transition from rigid imperative instructions to flexible, conceptual guidelines (guideline-based practice) which expand the boundaries of the clinical judgment of EMS specialists.

Overcoming the translational barrier and the harmonization of academic clinical recommendations with prehospital practice requires complex changes. The primary step is the technological upgrade of field diagnostics. In particular, the ubiquitous implementation of portable systems (POCT) for the quantitative determination of beta-hydroxybutyrate (capillary ketones) which will allow objectively verifying DKA already at the stage of primary triage. Besides, the establishment of a continuous and transparent feedback loop between inpatient emergency departments and prehospital agencies for the joint retrospective analysis of clinical outcomes is critically necessary. Finally, the system of continuous medical education of paramedics must evolve from the mechanical memorization of tree-like algorithms to the deep study of the pathophysiological mechanisms of endocrine catastrophes (see: Figure 9. Strategic pillars for harmonizing academic clinical recommendations with prehospital execution).

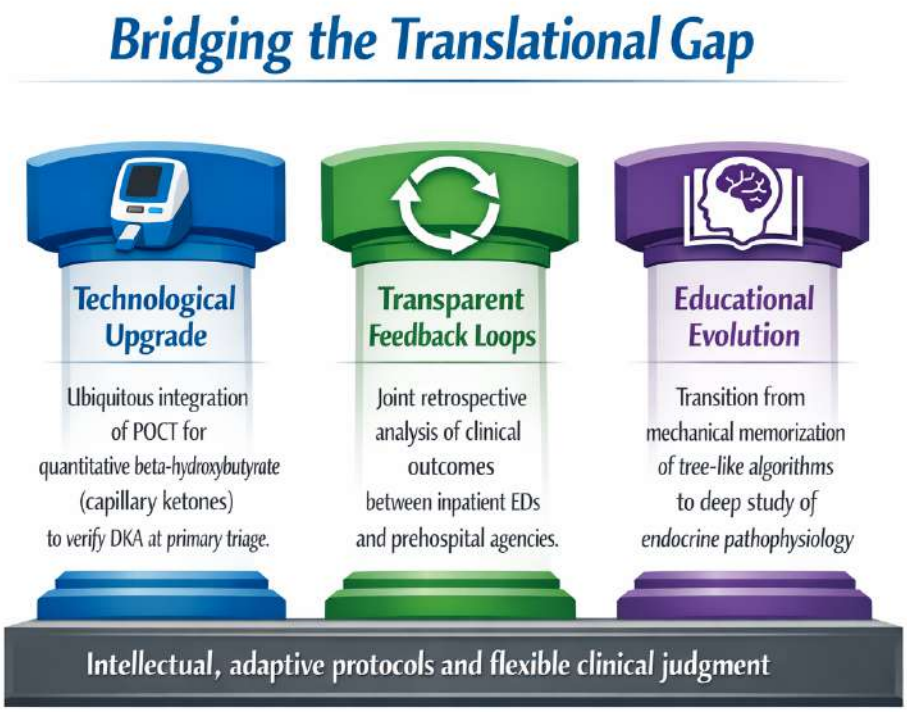

Fig. 9. Strategic pillars for harmonizing academic clinical recommendations with prehospital execution

The creation of intellectual, adaptive protocols supported by the solid biochemical erudition of providers will allow finally overcoming the chasm between high science and the harsh reality of saving lives "in the field". Such an approach also takes into account the human factor and removes too much responsibility from the paramedic leaving the probability and understanding of possible decisions which were made in an emergency situation and possibly do not correspond to idealized protocols.

Conclusions. The integrative analysis of the prehospital management of acute diabetic emergencies demonstrates that the high frequency of iatrogenic complications in patients with type 1 diabetes mellitus (T1DM) is primarily driven by systemic algorithmic limitations rather than individual provider incompetence. The extreme metabolic lability inherent to T1DM conflicts with rigid, historically established emergency medical services (EMS) protocols. These conventional algorithms were predominantly designed for the rapid resuscitation of traumatic or hypovolemic shock, making them unsuitable for the

meticulous, prolonged correction required for complex biochemical and osmolar shifts. Consequently, minimizing preventable morbidity and neurological deficits necessitates a transition from the immediate normalization of clinical parameters to the pathogenetically substantiated restoration of physiological homeostasis.

The foundation of this clinical transformation is the routine integration of expanded point-of-care testing (POCT). The measurement of capillary glucose and beta-hydroxybutyrate levels must be incorporated into the mandatory protocol for vital signs assessment in any patient presenting with an altered mental status.Furthermore, therapeutic interventions require strict, evidence-based revisions. First, the prehospital administration of exogenous insulin must be systematically avoided due to the high risk of inducing severe intracellular potassium shifts and subsequent arrhythmogenic hypokalemia. Second, fluid resuscitation tactics in diabetic ketoacidosis (DKA) must transition to a conservative, strictly titrated replacement of the circulating blood volume to prevent rapid extracellular osmolarity reduction and subsequent transcapillary cerebral edema. Third, the management of severe neuroglycopenia should prioritize minimizing hyperosmolar and emetogenic complications by transitioning to physiologically calibrated protocols, specifically the utilization of 10% dextrose (D10W) solutions.

Ultimately, the optimization of prehospital endocrinology depends on the structural reform of operational algorithms. The prehospital medical community must transition away from rigid procedural directives that are prone to generating algorithmically induced harm. By providing paramedics with adaptive, evidence-based clinical guidelines, supported by modern diagnostic instrumentation and comprehensive education on transcellular physiology, EMS systems can facilitate accurate clinical judgment. Synthesizing academic endocrinology with prehospital practice will effectively reduce iatrogenic risks and establish a safer framework for managing acute glycemic excursions in the field.

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