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Editorial

Cerebral-neurological Manifestations of the Acute Respiratory Distress Syndrome (ARDS)

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Substantial evidence currently exists demonstrating that volumetric overload (VO) complicates fluid therapy (FT) of shock inducing VO shocks (VOS) [1] that cause the acute respiratory distress syndrome (ARDS) [2,3]. These most serious morbidities and mortalities affect hundreds of thousand cases every year all over the world but remained overlooked, unrecognized, and underestimated. Cerebral and neurological manifestations of both VOS and ARDS are the commonest and most prevalent among the clinical features of the multiple organ dysfunction syndrome (MODS) that represent ARDS (Table 1), presenting mainly with deep coma of the highest grade.

Cerebral	Cardiovascular	Respiratory	Renal	Hepatic and GIT
Numbness Tingling SBB[1] COC[2] Convulsions Coma PMBCI[3]	Hypotension Bradycardia Dysrhythmia CV Shock* Cardiac Arrest Sudden Death	Cyanosis. FAM[4] APO)[5] RA[6] Arrest CPA[7] Shock lung ARDS[$]	Oliguria Anuria[8] Renal failure or AKI[9] Urea ↑ Creatinine ↑	Dysfunction: Bilirubin ↑ SGOT ↑ Alkaline Phosphatase ↑. GIT symptoms. DGR[10] Paralytic ileus Nausea and Vomiting.

Table 1: Shows the manifestations of VOS 1 of the TURP syndrome for comparison with ARDS manifestations induced by VOS2. SBB1 : Sudden bilateral blindness COC2: Clouding of consciousness PMBCI3: Paralysis mimicking bizarre cerebral infarctions, but is recoverable on instant use of HST of 5%NaCl and/or NaCo3, and so is coma and AKI FAM4: Frothing around the mouth APO5: Acute pulmonary oedema RA6: Respiratory arrest CPA7: Cardiopulmonary arrest ARDS $: Manifests on ICU later AKI9: Acute kidney injury DGR10: Delayed gut recovery CV Shock*: Cardiovascular shock of VOS reported here as VOS 1 and VOS2. Annuria8: That is unresponsive to diuretics but responds to HST of 5%Ncl and/or 8.4%NaCo3 AKI8: Acute kidney injury Also occurs the excessive bleeding at the surgical site and Leukocytosis occurred in the absence of sepsis and septic shock.

Fluid therapy (FT) was introduced during WW2. Ever since its complications have been frequently reported but notably some serious complications have been overlooked. The role of VO in inducing VOS and causing ARDS has remained unrecognized until recently, first by me and other authors are now catching up. The role of FT complications in inducing VOS and causing ARDS is hard to detect because VOS is a shock that complicates another existing shock being treated and it occurs seamlessly and un-noticed. It took me 40 years to unravel and resolve this dilemma making some new scientific discoveries in physics, physiology, and medicine [4]. It all started with the investigation of a condition known in urology as the transurethral resection of the prostate (TURP) syndrome [5] or acute dilution hyponatraemia. This is induced by large absorption of 1.5%Glycine irrigating solution used for the TURP surgery and/or 5% Dextrose infusion during the one-hour TURP surgery. This sodium-free fluid induces VOS1 that causes ARDS type1. Sodium-based fluids of crystalloids, colloids, and blood induces VOS2 and ARDS2. The difference is that VOS1 has a clear serological marker of hyponatraemia while VOS2 does not have such clear marker.

I attributed these conditions to the faulty rules on FT dictated by the wrong Starling's law that causes many errors and misconceptions of FT [6] which mislead physicians into giving too much fluid during shock resuscitation [7]. The research findings on the wrong Starling's law and how it has been corrected based on the hydrodynamic of the porous orifice (G) tube [8,9] and the newly recognized VOS [1] and the new patho-aetiology and therapy of ARDS were reported [2,3] and are highlight ed here. The errors on current FT and its corrections are given [6]. The original clinical studies on VOS causing ARDS [2,3,5] and the physics study that provide the replacement for the wrong Starling's law [8,9] were reported years ago and finalized recently. The precise volumetric overload is

shown in (Figures 1 and 2) which is statistically significant for the patho-etiology of VOS and ARDS with (P = 0.0001).

Figure 1: Shows the means and standard deviations of volumetric overload in 10 symptomatic patients presenting with shock and hyponatraemia among 100 consecutive patients during a prospective study on transurethral resection of the prostate. The fluids were of Glycine absorbed (Gly abs), intravenously infused 5% Dextrose (IVI Dext) Total IVI fluids, Total Sodium-free fluid gained (Na Free Gain) and total fluid gain in liters.

Figure 2: Shows volumetric overload (VO) quantity (in liters and as percent of body weight) and types of fluids. Group 1 was the 3 patients who died in the case series of 23 patients as they were misdiagnosed as one of the previously known shocks and treated with further volume expansion. Group 2 were 10 patients from the series who were correctly diagnosed as volumetric overload shock and treated with hypertonic sodium therapy (HST). Group 3 were 10 patients who were seen in the prospective study and subdivided into 2 groups; Group 3.1 of 5 patients treated with HST and Group 3.2 of 5 patients who were treated with guarded volume expansion using isotonic saline.

Recently, other authors have reported research in support of the concept that VO induce VOS and cause ARDS but stopped short of recognizing both conditions. Professor Hahn extensively studied volume kinetics in human volunteers and patients [10,11]. He demonstrated that increasing volume cause the morbidities of MODS. The huge prospective multicenter trials reported 3-10 liters of retained fluid in surviving ARDS patients with statistically significant (p = 0.001) but missed documenting VO in mortality cases [12,13]. Recent authors' prospective studies in adults and children reported the high significance of VO in relation to the morbidity and mortality of ARDS [14-16]. A brilliant future for the precise diagnosis and successful therapy for VOS and ARDS has already started.

Bibliography

1. Ghanem AN. "Volume Kinetic (VK) Shock or Volumetric Overload Shocks (VOS) In Surgical Patients". *Journal of Biomedical Science and Research* 2.3 (2020): 128.

2. Ghanem AN. "Volumetric overload shocks cause the acute respiratory distress syndrome: The plenary evidence on patho-aetiology and therapy". *Journal of Bio-Science* 1.4 (2020): 1-9.

3. Ghanem AN. "Volumetric Overload Shocks Cause the Acute Respiratory Distress Syndrome: Building the Bridge Between Physics, Physiology, Biochemistry, and Medicine". *Biomedical Journal of Scientific and Technical Research* 29.1 (2020).

4. Ghanem AN. "Update on Ghanem's new scientific discoveries in physics". *Physiology, and Medicine, Research in Infectious Diseases and Tropical Medicine* 2.1 (2020): 52-57.

5. Ghanem AN and Ward JP. "Osmotic and metabolic sequelae of volumetric overload in relation to the TURP syndrome". *British Journal of Urology* 66 (1990): 71-78.

6. Ghanem AN. "The Adult Respiratory Distress Syndrome: Volumetric Overload Shocks in Patho-Etiology, Correcting Errors and Misconceptions on Fluid Therapy, Vascular and Capillary Physiology". *Surgical Medicine Open Access Journal* 2.2 (2018).

7. Ghanem AN. "What are Misleading Physicians into giving too much Fluid During Resuscitation of Shock and Surgery that Induces ARDS and/or AKI?". *Asploro Journal of Biomedical and Clinical Case Reports* 3.1 (2020): 90-98.

8. Ghanem AN. "Magnetic field-like fluid circulation of a porous orifice tube and relevance to the capillary-interstitial fluid circulation: Preliminary report". *Medical Hypotheses* 56.3 (2001): 325-334.

9. Ghanem KA and Ghanem AN. "The proof and reasons that Starling's law for the capillary-interstitial fluid transfer is wrong, advancing the hydrodynamics of a porous orifice (G) tube as the real mechanism". *Blood, Heart and Circulation* 1.1 (2017): 1-7.

10. Hahn RG. "Adverse effects of crystalloid and colloid fluids". *Anaesthesiology. Intensive Therapy* 49.4 (2017): 303-308.

11. Hahn RG. "REVIEW ARTICLE Understanding volume kinetics". *Acta Anaesthesiologica Scandinavica* (2020): 1-9.

12. Rowan KM., *et al*. "PRISM Investigators, Early, Goal-Directed Therapy for Septic Shock - A Patient-Level Meta-Analysis". *The New England Journal of Medicine* 376.23 (2017): 2223-2234.

13. Huang DT., *et al*. "ProCESS/ARISE/ProMISe Methodology Writing Committee, harmonizing international trials of early goal-directed resuscitation for severe sepsis and septic shock: methodology of ProCESS, ARISE, and ProMISe". *Intensive Care Medicine* 39.10 (2013): 1760-1775.

14. Jones DG., *et al*. "Crystalloid resuscitation in trauma patients: deleterious effect of 5L or more in the first 24h". *BMC Surgery* 18.1 (2018): 93.

15. Coons BE., *et al*. "High volume crystalloid resuscitation adversely affects paediatric trauma patients". *Journal of Pediatric Surgery* 53.11 (2018): 2202-2208.

16. Schrier RW. "Fluid administration in critically ill patients with acute kidney injury". *Clinical Journal of the American Society of Nephrology* 5.4 (2010): 733-739.