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Introduction:
Most scales(GCS,NIHSS) don’t consider the pathway of secondary acute brain failure (sABF). Neuron-specific-enolase (NSE) could be usefull in diagnostic and treatment pts. with sABF[1,2].

Methods:
Prospective study incl. 35 pts. with ABF. Pts. were identical in condition, age and comorbidies. In main group, NSE examed and choline alfoscerate was used, pts. was divided into 2 subgroups Ia (n=12) with acute ischemic stroke(AIS) and Ib (n=10) pts. with posthypoxic encephalopathy. The control group (n=13) pts. with AIS treated by Loc.Protocol №602. Clinical, laboratory, and imaging variables were fully compared. Pts. examed by ABCDE algorithm, GCS and NIHSS. Brain CT, Carotid Doppler performed. Considering criteria:NSE(days 1,3,5), neurological status, length of stay in ICU (ICU LOS). "SS-6.0"was used.

Results:
The baseline NSE was higher and correlated to NIHSS(16.3±2.2, ÷2=1.08)in all pts. In Ia, Ib sbgroups NSE decreased for 3-5 days vs. control group 10-12days (÷2=7.93) and correlated with regression neurological deficit. ICU LOS in main group was 3.8±0.9 days vs. control group 5.9±0.9 days. Sensitivity and specificity of NSE as a marker of brain injury in pts. with AIS were 65 and 83% and in posthypoxic pts. were 90 and 90%, respectively, which showed NSE as eligible diagnostic criterion of posthypoxic cerebral edema. In Ia (AIS) pts. and Ib (posthypoxic edema) were confirmed by increasing NSE in 4-fold and 9-fold respectively more vs. pts. who had only Brain CT at first day. NSE also correlated with regression neurological deficit and improving of the neurological status. Although, two pts. In IIb group died with NSE 150-220 ig/ml

Conclusion:
1.NSE is an effective marker of the severity of damages even in the sABF, and shoved efficacy efficacy of treatment.
2.Negative outcome can be in pts. with sABF and more 3-fold increasing NSE and increasing up to 150-220 ig/ml is a mortality predictor.
3. We included NSE in local protokols