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Targeting Neuroinflammation by Vascular Versus Metabolic Acting Drugs, in a Preventive or Therapeutic Approach, Using an Experimental Model of Sporadic Alzheimer's Disease [sad].

O. NAYEL[1], W. El Hadidy[2]

[1]Pharma Dept. Medical Collage, King Saud University, Riyadh, Saudi Arabia ; [2]Pharma Dept Medical Research Institute, University of Alexandria, Alexandria, Egypt

Introduction; Neuroinflammation, is a final consequence of neuro-vasculo-metabolic uncoupling, common to all major neurodegenerative disorders,as sAD. One cause is brain insulin resistance (BIR).

Objectives; Comparepreventive or therapeutic potentiality of vascular versus metabolic actingdrugs

Aims; Their ability to suppress CSF neuroinflammatory markers, in STZ-induced, rat model of sAD.

Methods; 63 rats were injected i.c.v. STZ (3mg/kg/10ul) to develop BIR that induces sAD, then grouped into: 21 as untreated-sampled on (7^{th}/28^{th}/90^{th}) days / 28 as preventive-sampled after receiving oral dailysildenafil (10mg/kg), telmisartan (10mg/kg), rusovastatin (10mg/kg), orpioglitazone (10mg/kg) from 1^{st} till 28^{th} days / 14 astreated-sampled after receiving telmisartan or pioglitazone from 28^{th}till 90^{th} days. Sampled CSF from cisterna magna was analyzed for IL-6,TNF-a, TGF-b, MDA, and p-tau.

Results; In untreated group; inflammatory markerswere detected on 7^{th} while p-tau on 28^{th} day. Theirsignificant elevation was more on 28^{th} and 90^{th} days respectively. By prevention; p-tau was not detected withany drug, while inflammatory markers were significantly reduced by telmisartanand pioglitazone > rusovastatin, tipping % reduction in favor of metabolicacting drugs. By treatment, % reduction in p-tau and neuroinflammation was moresignificant by telmisartan > pioglitazone, when compared to untreated group.

Conclusions; Neuroinflammation was bettercontrolled in prevention by the collectively used metabolic than vascularacting drugs but the reverse was detected by those selected for treatment; denoting thatfinal outcomes are rather delineated by the drug's inherent ability to targetneuroinflammation, irrespective of its primary vascular versus metabolic action.

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