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ACETYLCYSTEINE AS AN IMPORTANT PANACEA.

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Acetylcysteine also known as N-acetyl-L-cysteine (NAC) is pharmaceutical drug and nutrition supplement used primarily as a mucolytic agent and in the management of acetaminophen (paracetamol) poisoning. It is on the WHO’s list of Essential Medicines. Some of the numerous pharmacological effects of acetylcysteine are antioxidant, antiradical, membrane stabilizing, detoxicating, immunomodulatory, anti-inflammatory actions.

So the target of our work is a justification of the pharmacodynamic effects of acetylcysteine at various urgent conditions in medicine.

When large doses of acetaminophen is taken, N-acetyl-p-benzoquinone imine accumulates in the body and it is usually conjugated by glutathione, but due to its excess, the reserved glutathione in the body is not sufficient because of the excess of it to inactivate it, making it free to react with hepatic enzyme leading to the damage of hepatocytes. When acetylcysteine is administered, it acts to maintain or replenish depleted glutathione reserves in the liver and enhance non-toxic metabolism of acetaminophen. In conditions like chronic obstructive pulmonary disease (COPD), emphysema, pneumonia, bronchitis, tuberculosis and bronchiectasis with production of excessive and thick mucus, inhaled acetylcysteine is normally used for mucolytic therapy as an adjuvant for the above respiratory conditions, due to splitting disulphide bonds linking proteins present in the mucus (mucoproteins) to reduce the symptoms, exacerbations and the accelerated lung function decline.

In diseases like neurodegenerative disorders, down syndrome, multiple sclerosis, Parkinson’s disease, Huntington’s disease, Alzheimer’s disease, focal cerebral ischemia, subarachnoid haemorrhage and traumatic brain injuries an acetylcysteine exerts its action by increasing the level of glutathione peroxidase, free radical scavenging and by increasing the level of superoxide dismutase.

Acetylcysteine inhibits homocysteine enhanced expression of an oxidized-LDL receptor, inhibits metalloproteinase-9 (gelatinase B) activity and expression in lipid-laden macrophage-derived foam cell by 60%, thereby demonstrating a potential for antioxidant to stabilize vulnerable atherosclerotic plaques.

Oral acetylcysteine is used for the prevention of radiocontrast-induced nephropathy (a form of acute renal failure). Acetylcysteine protects patients with moderate chronic renal insufficiency from contrast-induced deterioration in renal function after coronary angiographic procedures, with minimal adverse effects and at a low cost. Years of studies shows acetylcysteine’s ability of treating and preventing the cause of chronic generalised periondontitis due to its antioxidant, antiradical and membrane stabilizing properties.

In conclusion acetylcysteine has a broad spectrum of actions and possible applications across multiple conditions and systems. As a drug, it represents the ideal xenobiotic, capable of directly entering endogenous biochemical processes as a result of its own metabolism. So further scientific investigations should be directed in new findings of possible acetylcysteine pharmacodynamics effect in case of different urgent medical problems.