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Review Article

Acute Lymphoblastic Leukemia

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INTRODUCTION

Leukaemia as we know is a malignant cancer of the bone marrow and blood which is characterized by an uncontrolled accumulation of abnormal blood cells which leads to the inhibition of normal blood cells functions and many times death. It causes more deaths than any other form cancer among children around the age of twenty [1]. Acute lymphoblastic is a cancer of white blood cells which fight against infection characterized by the excessive multiplication of malignant and immature WBC (lymphoblast) in bone marrow. This cancer can be treated by chemotherapy, steroids, radiation therapy and intensive combined treatments including bone marrow or stem cell transplants. The drugs which are commonly used for this cancer are prednisolone, dexamethasone, vincristine, L-asparaginase, daunorubicin, cyclophosphamide,cytarabine, etoposide, thioguanine, mercaptopurine, hydrocortisone, etc. There are variety of drugs available today but their efficacy in treatment of cancer at third and fourth stage is doubtful. But scientist believe that treating the patient suffering with this type of cancer should be treated with this enzyme L-asparaginase and not by chemotherapy because the enzyme is more safer to use .

Types Of L-asparaginase :-

This enzyme has two related families which are designated as type 1 and 2 . These types are according to the terminology in E.coli. L-asparaginase-1 (AnsA) is a low affinity enzyme which is found in the cytoplasm. The synthesis of cytoplasmic L-asparaginase 1 is constitutive whereas L-asparaginase-2(AnsB) has ahigh affinity periplasmic enzymewhich is activated during anaerobiosis. AnsB is changed by aeration, carbon source and variation of available amino acids. If we go further only the type-2 enzyme shows the substantial anti-tumour activity [2]. periplasmic enzymewhich is activated during anaerobiosis. AnsB is changed by aeration, carbon source and variation of available amino acids. If we go further only the type-2 enzyme shows the substantial anti-tumour activity [3].

If we come down to the evaluation part of L-asparaginase from different bacterial strains so each one differ in their biochemical and therapeutic properties.Currently two bacterial strains are commonly used one is E.coli and second is Erwinia chrysanthemi [4].

They both possess different immunological specificities and offer an alternative therapy if patient becomes hypersensitive to one of the enzymes. When comparison is between the both then E.coli can be used as the first line of therapy and Erwinia chrysanthemi can be reserved for allergic patients as it is less toxic. Erwinia chrysanthemi has a shorter life span than E.coli [5].

MECHANISM OF ACTION OF L-ASPARAGINASE:-

The mechanism of this enzyme explains that lymphocytic leukemic cells are deficient in L-asparagine synthetase so that they can make their own asparagine. When these cells are treated with L-asparaginase which catalyzes the conversion of L-asparagine to L-aspartate and ammonia.as a result of which the growth of cells is critically affected with the rapid depletion of L-asparagine from the blood supply and the surrounding tissue [6].

As we know that cells require a continuous supply of amino acid L-asparagine so that they can build proteins and many cells need the enzyme asparagine synthetase so that they can make their own asparagine. The enzyme takes L-aspartate and addition of an amine takes place which forms the characteristic amide group of asparagine. L-asparaginase works in an opposite manner, by taking L-asparagine and pulling of its amine and in turn releases l-aspartate and ammonia.In our human body L-aspartate plays a major role by acting as a precursor of ornithine in the urea cycle. It also takes part in transamination reactions by forming oxaloacetate in the gluconeogenic pathway leading to glucose formation. But if we introduce a large dose of this enzyme into the blood then it will circulate and continually break done all the asparagine it finds which ultimately starves the cells that rely on blood-borne supply. This enzyme cuts off the supply of L-asparagine in the blood and the cancer cells die as they are not able to build the proteins. When L-asparaginase is given it also prevents free diffusion of L-asparagine from the surrounding tissues to the cancerous cells.If there is a high pressure in the blood stream, then these enzyme molecules may passintothe intracellular spaces from fine capillaries and they will catalyze the hydrolysis of L-asparagine [7] .

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