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Research Article

IN VIVO STUDY OF IMMUNOMODULATORY ACTIVITY OF VIDARIKANDA

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ABSTRACT:

An attempt was made to assess antifungal immunomodulatory activity of vidarikanda the experiment is carried under delayed hypersensitivity (DTH) model after screening we can say that vidarikanda has immunopotentiating and immunomodulatory action when it given in formal dose (dose 3 to 6 gm/kg) Thus vidarikanda can be used in a) Auto immune diseases. b) immuno deficiency syndrome.

KEY WORDS: *Pueraria tuberosa*, immunomodulatory, immunopotentiating.

INTRODUCTION:

Now a days the immunity of the individuals in the society is decresing and individuals were becoming more prone to various diseases.

Due to bad dietary habits, physical and mental stress and other factores togetherly acting and affecting on the health and immunity of individuals in the society.

A cure or even management of immunosuppressive disease or degenerative disease has been found to be an extremely difficult task by the modern medicine.

But in Ayurveda the Rasayana theraphy is very much helpful for immunosuppressive diseases. It also results in delaying the ageing and also slowing down the degernative process.

MATERIAL AND METHODS:

Method: This in vivo study carried out under delayed type hypersensitivity (DTH) Model.

Materials: 1) Swiss Albinomice
2) Sheep Red blood cells.

Drugs:

1) Vidarikanda (Pueraria taberosa)
2) Cyclophosphomide
3) Alsevar sol
4) Distilled water

Instruments:

1) Digital vernier callliper
2) Autoclave chamber
3) Centrifuging Machine
4) Centrifuge tube
5) Oral feeling needle
6) Microscope

Method Delayed type hypersensitivity (DTH):

DTH is a reaction of cell of cell mediated immunity and becomes visible only after 16-24 hours.

Groups of 10 mice per treatment are immunized by injecting 20 ul of 2x10 SRBC/ml.s.c. into the right footpad. Seven days later, the thickness of the left hind foot pad is measured using Vernier caliper reading to 0.01 mm and the mice are then challenged by injection of 20 ul of 5x10 SRBC/ml into the right foot pad. Severn days later, the thickness of the left hind foot pad is measured using vernier caliper reading to 0.01 mm and the mice are than challenged by injection of 20 ul of 5x10 SRBC/ml. i.d. into the left hind foot pad (0 time) Foot thickness is measured again+24h and +48h after this challenge. The difference between

the pre and post challenge foot thickness expressed in mm was taken as a measure of DTH.(Doherty:1981)

For this experiment 8 (eight) groups are taken into consideration.

F1 - Control (C)
F2 - Cyclophosphamide Group (CP)
F3 - Vidarikanda Low Dose 3 gm
F4 - Vidarikanda Medium Dose 6gm
F5 - Vidarikanda Higher Dose 18 gm
F6 - Cyclophosphamide + Vidarikanda Low Dose 3 gm
F7 - Cyclophosphamide + Vidarikanda Medium Dose 6 gm
F8 - Cyclophosphamide + Vidarikanda Higher Dose18 gm

Experimental Design- Delayed type hyper-sensitivity moded (DTH) in mice.

Day 0	Groups F2,F6.F7,F8	C.P. by IP rout, 2hrs before Sensatization
	Groups F1------- F8	Sensatization with 1x10 SRBC's
Day 1 to 5	Groups F1 Groups F2 Groups F3 & F6 Groups F4 & F7 Groups F5 & F8	Normal diet Normal diet Lose dose fo V.K. Medium dose of V.K. Higher dose of V.K.
Day 5	Groups F1------F8	Measurment of paw Thickness. Challenge with 1x10 SRBC's
Day 6 to 9	Groups F1------F8	Measurment of paw thickness.
Day 10	Groups F1------F8	Sacrifice and collection of Blood for WBC and Total plateletcount

C.P. – Cyclophosphamid, I.P. - Intraperitonical
SRBC – Sheep Red Blood Cells, V.K. – Vidarikand.

RESULTS:

In Control group W.B.C. Count is 8333+727.24 which is regarded as base line for comparism.

In group F2 i.e. cyclophosphomide 20mg/kg. showed reduction in W.B.C. count to 3200+702.37 indicating cytotoxic and immunosuppressant action of the drug.

In group F3 i.e. 3 gm/kg of vidarikanda showed increase in W.B.C. count i.e.10750+1251.33.

C.P. treated animals receiving vidarikanda 3gm /kg showed increased in w.b.c. count i.e. 10533+528/46 which is statistically significant.

C.P. treated animals receiving vidarikanda 6gm/kg showed increase in w.b.c count i.e. 10550+629.15 which is statistically significant.

Hb%

Hb % in control i.e. F1 group is 13.21+9004 which is regarded as base line for comparism.

In group No.F2 C.P.20 mg/kg. showed increase in Hb% count i.e. 10.49+0.9234 due to immunosupresant action of cyclophosphamide.

Group F3,F4,F6, F7 showed stasticaly no significant effect with Hb% more or less same which respect to control group.

In to RBC :

RBC count for all various formulation compaire to cyclophamide treated group were improve- significantly to my closappressed group.

Effect of Vidarikanda on D.T.H.

The results obtained in D.T.H. model are given in table,

The change in mean foot pad oedema is plotted against time in graph

In all groups odema increased up to first 48 hours and then started to decrease.

In groups F2 rate by decrease in oedema was low as compare to other groups.

In Groups F3 and F4 rate of decrease in oedema was higher than group F1 &F2.

In group F6 And F7 showed the remarkable decrease in means foot pad oedema.

In group F5 and F8 all albinomice were found dead due to high dose toxic effect of vidarikanda.

DISCUSSION:

Discussion of haemogram changes:

W.B.C. count:

As discussed earlier, leucocytes mediates the immune response in animals and human being.

In Control group F1 leucocytes count is within indicates natural immunity of mouse.

In group F2i.e. Cyclophosphamide group leucocyte count is comparatively very low due to immunosuppressive action of cyclophosphaminde.

In group F3 & F4 i.e. vidarikanda 3gm & 6gm without C.P. optimal increase in count indicating immunopotentiating activity compaired to that of control group.

In group F6& F7 leucocytes count showed significant increase which proves immunomodulating action of Viderikanda.

Haemoglobin:

Hb% is not direct indicator of immunity, but it is a good indicatior of general health.

Values of Hb % for all the groups are found to be more are showed not significant variations.

Shich indicates vidarikanda had no effect on Hb%.

RBC:

RBC count for all various formulations compaire to cyclophosphaminde trated group were improve significantaly to mylosappress group.

Platelet : Count :

Platelets are not direct indicatory immune response. In Group F2 there is significant decrease in platelet count.

In group F3& F4 there is increase in platelet count as compare to group F1.

In group F6 & F7 showed modete increase in platelet count after immunopression.

D.T.H. Model :

Formulation were checked for their immunomodulatory potential by using C.P. induced. Delayed type Hypersensitivity model in mice.

The decrease in mean foot pad oedema was seen.

As compare to control group C.P. treated group showed minimum decrease in foot pad oedema.

The rate of decrease in mean foot pad oedema in F3& F4 group was higher than the other control group.

CONCLUSION:

By Studying all the results we can say that vidarikanda has immunopotentiating and immunomodulatory action. When given in formal dose (Dose3-6 gm/kg)

Thus vidarikanda can be used in

a. Auto immune disease
b. Immuno deficiency syndrome

In high dose it is said to be toxic, For detail information about toxic effect of high dose of vidarikanda further study will be needed.

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