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Short Communication

Serum electrolytes and glycoprotein changes during penicillic acid toxicosis

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Abstract

Serum electrolytes and glycoproteins were assayed during penicillic acid toxicosis and were found to be increased in the experimental animals suggesting the interaction of penicillic acid with the cell membrane and its components thereby altering the integrity and permeability of membranes.

Key words: Penicillic acid toxicosis, electrolytes, glycoproteins.

1. Introduction

Penicillic acid, 3-methoxy-5-methyl-4-oxo, 2,5-hexadienoic acid (an α, B-unsaturated conjugated lactone), is a secondary metabolite produced by several food-borne fungi. Its potential health hazard was suggested when it was isolated from agricultural products. Penicillic acid has a variety of biological activities including antibacterial, antidiuretic, antiviral and antitumour properties. It has also been reported to be carcinogenic in rats and mice, cytotoxic in cultured cells and hepatotoxic in mice[1]. Several enzymes such as lactic and alcohol dehydrogenases[2], aldolase[3] and ATPases[4] have also been inhibited by penicillic acid.

Mycotoxins like patulin[5] (hepatotoxin and carcinogen) and rubratoxin-B[6] (potent hepatotoxin) elaborated from several species of *Penicillium* have also been known to possess lactone ring in their structures and have received attention as potential health hazard.

In the present investigation, the serum electrolytes and glycoprotein components were estimated. The changes of these parameters are indicative of and can suggest the occurrence of tissue damage.

2. Materials and methods

Penicillium cyclopium (This strain was isolated in our laboratory from a fungal-contaminated feed and was confirmed by IARI, New Delhi. It has been found to

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produce penicillic acid as a major secondary metabolite) was grown in 1 litre of Raulin-Thom medium at 20 to 22°C for 14 days and penicillic acid was extracted from the culture filtrate by the method of Bentley and Keil[7]. Its purity was tested by NMR, IR and UV spectral analyses along with an authentic sample (A gift from Dr. E. B. Lillehoj, Agricultural Research Southern Region, Louisiana, U.S.A.). Contaminated diet for experiments was prepared by growing *P. cyclopium* in bread sterilized at 20 to 22°C for 14 days. The fungus was then inactivated by the addition of chloroform and later removed completely by drying. The bread was then powdered and mixed with normal rabbit diet in the ratio of 1:2 (w/w) (contained 1 mg penicillic acid per 10 g of bread).

A six-month long feeding trial was carried out using albino female rabbits weighing 650–850 g. The rabbits were divided into three groups of six animals each; the control group received normal diet, the second one was fed with contaminated diet and the third was injected intraperitoneally, with the pure toxin dissolved in saline, at a dosage level of 2 mg/kg body weight on every alternate day. Water was given *ad libitum* to all the animals, which were pair-fed.

After the experimental period, blood was collected by ear vein puncture and serum was separated. In the serum the components total hexose[8], hexosamines[9], sialic acid[10], fucose[11], mucoproteins[12], non-aminopolysaccharides[8], sodium[13], potassium[13] and calcium[14] were assayed.

3. Results and discussion

The concentrations of glycoprotein components are increased significantly in both contaminated diet-fed and toxin-treated animals (Table I). Elevation of serum protein-bound polysaccharides reflects the occurrence of tissue damage[15] resulting in the release of substances derived from complex tissue carbohydrates into the circulation[16–17]. Shetlar *et al*[18] have suggested local liberation of glycoproteins from an injured area. Since penicillic acid has been reported to be hepatotoxic[1] the increased glycoprotein components in serum may be due to interaction of penicillic acid on the membranes resulting in the escape of these components into the surrounding plasma.

Table II depicts the increased concentration of serum electrolytes during penicillic acid toxicosis. Electrolytes are essential for many vital processes and a decreased uptake of K^+ ion by erythrocytes has been observed during patulin toxicosis[19]. Thacker and Carlton[20] have reported a slight elevation of sodium during citrinin mycotoxicosis in guinea pig. The increased sodium and potassium levels observed in the present investigation suggest an altered membrane permeability during penicillic acid toxicosis. The elevated levels of serum calcium may be due to increased removal from the bones. Hypercalcaemia similarly occurs in secondary carcinomatosis involving the bones[21]. It has already been established that penicillic acid is a carcinogen[1].

The increase in the levels of serum glycoproteins and electrolytes are due to the leakage of these components from the affected tissues.

Table I

Serum glycoprotein components and mucoproteins of normal and experimental rabbits

(Values are expressed in mg/dl serum and are the average of six individual experiments in duplicate ± S.D.)

Groups	Hexose	Hexosamines	Fucose	Sialic acid	Non-amino polysaccharides	Mucoproteins
Control	125.70 ± 10.12	54.08 ± 4.51	8.84 ± 0.69	51.08 ± 3.93	131.01 ± 9.98	142.23 ± 10.09
Contaminated feed fed	148.33 ± 10.99***	66.49 ± 4.36**	10.63 ± 0.59**	62.86 ± 5.91**	166.91 ± 16.82**	166.24 ± 12.10***
Toxine-treated	153.91 ± 12.02**	68.47 ± 4.35*	11.23 ± 0.81*	66.49 ± 4.50*	170.44 ± 11.04*	170.81 ± 10.35**

Statistically significant variations as compared to normal rabbits are indicated by $*P < 0{\cdot}001$; $**P < 0.01$; $***P < 0.02$.

Table II

Serum electrolytes of normal and experimental rabbits

(Values are the average of six individual experiments in duplicate ± S.D.)

Groups	Sodium (mEq/L)	Potassium (mEq/L)	Calcium (mg/dl)
Control	144.80 ± 5.10	4.72 ± 0.32	4.54 ± 0.38
Contaminated feed fed	153.62 ± 6.18****	5.21 ± 0.24****	5.90 ± 0.41*
Toxin-treated	155.72 ± 7.10****	5.36 ± 0.38****	6.59 ± 0.81*

Statistically significant variations as compared to normal rabbits are indicated by $*P < 0.001$; $****P < 0.05$.

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