LETTER TO THE EDITOR

EFFECT OF NIFEDIPINE ON HISTOLOGY OF HEART LIVER, KIDNEY AND PANCREAS IN RATS

(Received on October 19, 1992)

Sir,

It has been reported that nifedipine raises the blood sugar level in human volunteers (1). The effect of nifedipine on blood sugar is not dose dependent (2). Experiments have therefore been performed to investigate the long-term effect of nifedipine in large dose on the histology of the heart, liver, kidney and pancreas as well as to ascertain whether morphological changes of the beta cells of the pancreas, caused by alloxan, could be prevented/reduced by prior treatment with nifedipine in rats.

Groups of 3 albino rats were treated as follows: Group 1 was treated with nifedipine 0.5 mg/kg, ip for a period of 3 weeks and sacrificed. Pieces of heart, kidney, liver and pancreas were removed and kept in 10% formalin, second group received saline ip for the same period and served as control. They were processed for histological studies and stained with eosin and haematoxylin. Group 3 and group 4 were treated with nifedipine as above, followed by alloxan 150 mg/kg, sc for 2 days (gr. 3) and 6 days (gr. 4) and scarificed.

Results show that on microscopic observation there was no change in cellular architecture of tissues from heart, kidney, liver and pancreas after 3 weeks of treatment with nifedipine 0.5 mg/kg compared with control. In this observation nucleus and cytoplasm appeared deep blue and pink respectively.

Microscopic study with alloxan for 2 days showed necrosis of pancreatic beta cells. However, presence of nucleus in the cytoplasm of some of the cells indicate that viable cells are still there, whereas animals treated with alloxan for 6 days, caused almost total destruction of beta cells. Because of disappearance of nucleus and cytoplasm, only cellular membrane looked pink in colour. Nifedipine pretreatment for three weeks, did not alter alloxan induced changes.

It has been shown that nifedipine pretreatment significantly reduces the insulin secretion and increases the blood sugar level in the rat even after complete destruction of beta cells of the pancreas (4) suggesting other factors might contribute to hyperglycaemic effect of nifedipine. In view of the fact that calcium channel antagonists enhance epinephrine induced gluconeogenesis (5) reduced insulin secretion might cause epinephrine release which may contribute to hyperglycaemia caused by nifedipine.

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REFERENCES


