Catecholamine and Acetylcholine Levels of the Kidney in Weanling Rats Fed a Choline-Deficient Diet

M. A. Rossi and R. S. Costa

Department of Pathology, Laboratory of Experimental Pathology, Medical School of Ribeirão Preto, Brasil

With 2 Figures

Received June 15, 1979

Summary

A disturbed renal circulation due to an imbalance between vasoconstrictor catecholamines and a vasodilator such as acetylcholine, caused by a decrease in acetylcholine, has been postulated as the basic mechanism of hemorrhagic degeneration of the kidneys in choline deficiency. To explore this hypothesis further a group of male weanling Wistar rats was fed on a choline-deficient diet for 10 days (group CD). A control group was fed on the same basal diet supplemented with choline (group CS). Food intake and body weights were registered. The kidneys of choline-supplemented and choline-deficient rats were studied grossly and histologically. The levels of catecholamines (noradrenaline and adrenaline) and acetylcholine were determined. Pathological changes of the kidneys were present in 30 out 57 choline-deficient rats, permitting the separation of data obtained from deficient rats into those not associated with renal injury (CDa rats) and those associated with renal injury (CDb rats). A marked increase in the levels of renal catecholamines (noradrenaline and adrenaline) and acetylcholine were determined. On the other hand, the content of acetylcholine remained unchanged. It is noteworthy that the changes in tissue catecholamine levels occurred before there were changes in kidney weight and morphology. The findings support the concept that an imbalance between sympathetic and parasympathetic systems plays an important role in the pathogenesis of the renal injury of choline-deficient weanling rats; and this imbalance would be the result of an excess of catecholamines in the kidneys.
Since the description by Griffith and Wade (1939) of marked hemorrhagic enlargement and degeneration of the kidneys in choline-deficient weanling rats, the study of this pathological change has attracted much attention. Its basic mechanism, however, is still obscure. It has been suggested the possibility that a renal phospholipid deficiency may play a role in the pathogenesis of this condition (Patterson and McHenry, 1944; Baxter and Goodman, 1955; Monserrat et al., 1974). On the other hand, Wolbach and Bessey (1942), in view of the function of acetylcholine as a neuromuscular mediator, advanced the hypothesis of a neurovascular cause underlying the renal injury in choline deficiency. Several reports available seem to favour this hypothesis of a neurovascular mechanism. Dessau and Oleson (1947) have shown that the renal damage due to choline deficiency is considerably reduced by prior renal decapsulation, appearing that this effect would be a result of disturbing renal nerves. A similar observation was described by Baxter (1952). Baxter (1953) has found that while dibenamine, a blocker of $\alpha$-adrenergic receptors, afforded protection against the renal injury of choline deficiency, the use of dibenzyline, another adrenergic blocking agent, was ineffective. However, because of the severe local irritation and induration of tissues caused by dibenamine, and the negative results with dibenzyline, he interpreted the protective effect of dibenamine as resulting from an "alarm reaction" and not from an adrenergic blocking effect. Nagler et al. (1968) have found that 5 days of choline deficiency resulted in a 50--75% decrease in kidney acetylcholine concentration which lead them to suggest that a hypersensitivity to catecholamines results from this insufficiency with subsequent vaso-spasm and ischemic damage. Later, Nagler et al. (1969) have reported an increased sensitivity to adrenaline of mesoappendicetal circulation of rats fed a choline-deficient diet as compared to controls. More recently, Bruce et al. (1976) could demonstrate that choline-deficient rats had a higher excretion of urinary catecholamines than control animals supplemented with choline. Furthermore, they could shown that deficient rats injected with reserpine, which depletes the stores of noradrenaline in both central and peripheral nervous systems, had significantly less renal necrosis and excreted less catecholamines than choline-deficient animals.

No information exists in the literature, however, on the levels of both sympathetic and parasympathetic transmitters in the kidneys in choline deficiency. Therefore, the current investigation was undertaken in order to estimate the levels of catecholamines and acetylcholine