CARDIOVASCULAR DISEASE

CASE REPORT ON HYPERTENTION

BY
Dr. Moses Kayode Omole, Pharm D.

Department of Pharmaceutical Microbiology and Clinical Pharmacy, Faculty of Pharmacy College of Medicine, University of Ibadan, Ibadan, Nigeria.

This is the case report of a 58 year old black female that was admitted to the University College Hospital on 9/11/83, with the chief complaints of Edema, Hypertension, Diabetes and Anemia. She reported having severe Dyspnea and othopnea a day prior to admission.

The patient is a chronic alcoholic who started drinking at the age of 13 years. She reported having Diabetes for 10 years and recognition of hypertension for the same length of time. She had had 3 hospitalizations in the University College Hospital for the same complaints. The interval between the third and fourth admission was 2 years.

During these 2 years, she was engaged severely in drinking and smoking. On physical examination, she was slightly wasted, alert in mild to moderate distressed but not jaundiced. She had edema, 2+, Ascites 2+, no lymphadenopathy. Pulse = 96/minute regular; B.P. = 180/110 (standing).

There was A.V. nicking and enlarged prostate. She had had 10 years history of gout and essential hypertention. The lung showed vesicular breadth sounds and few scattered rales heared in the bases.

In the abdominal region, there was ascites +, no capus medusae, liver 5cm and nontender. No hernias, bowel sounds were normal.

X-ray revealed left ventricular hypertrophy, blood laboratory data revealed Uric acid =8-10mg. per cent, serum creative 4,3, serum potassium 5.5, creatinine clearance 15 ml/mint. Secondary postprandrial 146mg.%. Urinalysis shows the presence of protein, RBC, WBC and Bacteria.

LABORATORY DATE

Blood	Patient	Normal value
HCT*	35	40% for women
HGB*	10.1	12-16gm% (for women)
WBC	10,000	5,000-10,000
SUGAR*	250	60-100mg.%
UREA - NITROGEN	15	10-20MG.%
URIC - ACID*	9.6	2-5-8mg.%
MAT*	134	135-152meg/L
K+	5.6	3.7-5.1meg/L
CL-	105	95-105meg/L
CO2	15.4	24-32meg/L
CA++	9.4	8.5-10 mg%
Bit Total	0.8	0.15-11.0mg%
A1K PO4*	116.	30-85 mu/ml
Total prot*	7.2	6.8mg%
Abumin*	3.3	3.5-5 mg%
SGOT	24.	10-24 mu/ml.
LDH*	218	90-200 mu/ml
URINE		
Protien		
RBC		
S1Bc		
Bacteria.		

* Indication of abnormalities.

The purpose of the above laboratory tests was to show all the diseases that might associate with her hypertension and possible complications.

TREATMENT

1. Bed Rest
2. Diet
3. Aldomet^{r} (Methyldopa)
4. Chlorothiazide (Diuril)^{R}1

METHLDOPA (Aldomet^{r})

Methyldoppa effectively inhibits the decarboxylation of both dopa and 5-hydroxytryptophan in vitro and invivo. In addition, it decreases the concentration of 5-Ht, dopamine and morepinephrine in the CNS and in most peripheral tissues, but not in the adrenal medulla.

When Methyldopa is administered orallly, 50% or less is absorbed. It appears rapidly in the urine, predominantly as the administered drug and its conjugates, but small amounts of decarboxylate derivatives have been identified. Both the total quantity absorbed and the distribution of metabolities in the urine can vary considerably in different individuals and in the same patient from day to day. Methyldopa and the metabolites react in the standard chemical tests for catecholamines, and their presence in blood and urine can cause false positive tests for pheochromocytoma for several days after therapy is discontinued.

CHLOROTHIAZIDE (DIURIL^{R})

The dominant action of thiazides is to increase the renal excretion of sodium and chloride and an accompanying volume of water. This results from inhibition of the tubular mechanisms of electrolyte reabsorption. Unlike both the mercurials and carbonic anhydrase inhibitors, the renal effect is virtually independent of alterations in acidbase balance.The thiazides also evoke a significant augmentation of potassium excretion in amounts sufficient to produce hypokalemia. The thiazides vary widely in their potency as inhibitors of carbonic anhydrase.

The thiazides are absorbed from the gastrointestinal tract and owe their usefulness largely to their effectiveness by the oral route. Absorption is relatively rapid, since most agents show a demonstrable diuretic effect within an hour after oral administration. Chlorothiazide is distributed throughout the extracellular space and does not accumulate in tissues other than the kidney. The drug passes readily through the placenta barrier to the fetus. All thiazides probably undergo active secretion in the proximal tubule. The extent of this process may vary as may the degree of subsequent reabsorption.

The renal clerances of the drugs are therefore high and may be either above or below the rate of filteration. Most compounds are rapidly excreted within 3 to 6 hours.

Hydrochlorothiazide does not undergo metabolic alterationin the body.

REFERENCE

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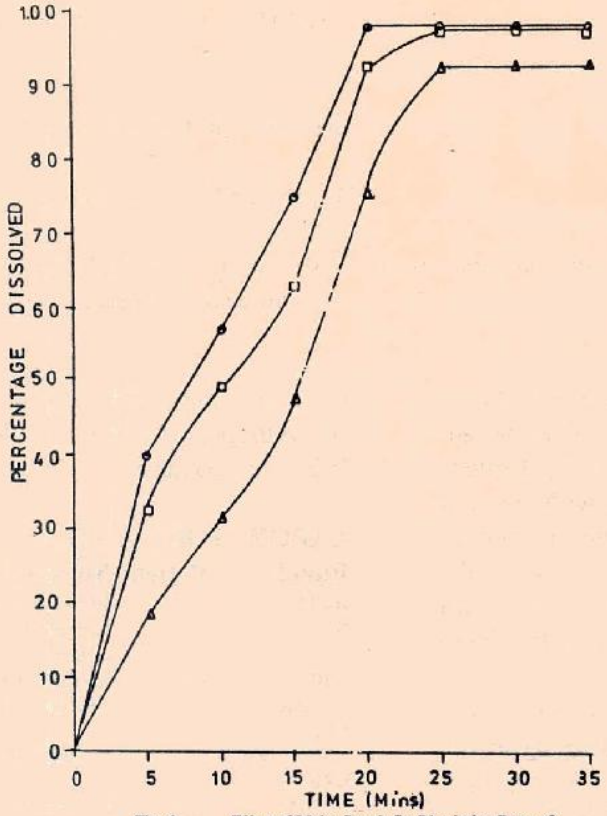

Fig. 4: Effect of Maize Starch On Dissolution Rates of Acetyl Salicylic Acid Tablets.

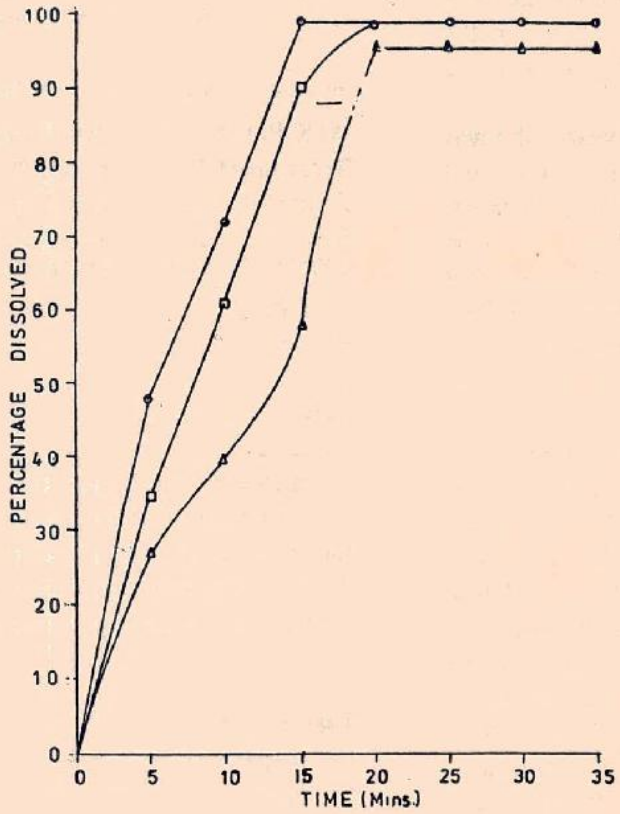

Fig. 3: Effect of Sorghum Starch Binder on Dissolution Rates of Acetyl Salicylic and Tablets.