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Case Report

MANAGEMENT OF CONGESTIVE HEART FAILURE (CHF): A CASE REPORT ON DIGOXIN

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A case report on the management of Congestive Heart Failure is presented with emphasis on the use of DIGOXIN.

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INTRODUCTION

Congestive heart failure (CHF) can be defined as a clinical syndrome that is characterized by congestion of both the pulmonary and systemic circulation. Despite many controversies of the term congestive heart failure (CHF), it was not until recently that the term was established to be appropriate as almost all patients exhibited the typical signs of pulmonary congestion such as dyspnoea on exertion (DOE). It is now known that some patients may not have congestive symptoms. (Raeh1 & Nolan 1992). Patients with primarily diastolic dysfunction may have dyspnoea only with exertion. Therefore, the term FAILING HEART SYNDROME (FHS) may replace the older term congestive heart failure. Another definition of FHS is "a syndrome in which cardiac dysfunction is associated with reduced exercise tolerance, a high incidence of ventricular arrhythmia and shortened life expectancy (Cohn 1988).

The incidence is correlated with age. It is estimated that above 50 years, 2 to 5 people in every 1000 will bee diagnosed as having CHF. Males are more predisposed to developing CHF by age of 60 and above than females at a ratio 3: 1 (Raehl & Nolan 1992).

The case of a 61-year-old adult Nigerian reported and discussed in this paper is peculiar in the sense that she really had Failing Heart Syndrome. Her CHF is not a complicated one, since in most cases, Patients with CHF has complications such as Cardiac arrythmias, Hypercholesterolemia and/or Coronary artery disease. (Castlemen & Page 1984). These complications make diagnosis and therapy very difficult and patients die of Cardiac arrest. (Raehl & Nolan 1992). She also belongs to the relatively few Female adult Nigerians who has CHF which is not chronic or complicated.

Digoxin, despite many controversies on its use and other Cardiac glycosides in the treatment of CHF is still predominantly used in clinical medicine as the most rational drug whether the CHF is chronic (Complicated) or not.

Case History

A sixty-one year old Nigerian woman was admitted to University College Hospital Ibadan.

The patient had four previous admissions at University College Hospital, Ibadan, Nigeria and the past history revealed that she had diabetes mellitus for nine years, congestive heart failure for one year, chronic renal failure and shortness of breath (SOB) a day prior to the 5th admission. Her SOB became progressively worse on the day of admission as she complained that she could not breathe properly. Her SOB was so severe that she could not sleep, and she was awaken with dyspnoea. Whenever, she sat upright in

bed, the SOB would be relieved, but not completely.

She denied any recent episode of paroxysmal nocturnal dyspnoea (PND) but did admit sleeping on two pillows. She admitted experiencing SOB in walking around in the house and when performing daily activities. She admitted history of SOB on mild exertion while walking short distance. She said that she did not walk upstairs and stated that during the last few weeks dyspnoea was more often and occurring with less exertion. She denied associated chest pain on exertion at any other time.

The volume of urine passed was small and the patient admitted to the accumulation of fluid in lower extremities. She stated that she first noticed fluid five days prior to admission and the leg became progressively larger. The patient had cough associated with dyspnoea but cough was non-productive and dry.

She denied the use of drugs, cigarette or alcohol. She revealed that during her childhood, she had chicken pox, measles, mumps and whooping cough. She was allergic to penicillin.

Physical Examination

Her physical state was well nourished and developed. There was no visible signs of cyanosis, tremor or jaundice. In the neck, she had slight venous distention bilaterally with pulsation 4cm. above angle. The neck was supple, her thyroid was not palpable, and the funduscope showed the arteriovenous necking of 1.5cm in the left eye with tortuosity of vessels. There was ptosis of the right lid. Conjuctiva in each eye was pale showing soft exudates and punctual hemorrhage. Both nostrils were hyperemic with septum occurring in the mid line. There was inflammation of the throat with ovula occurring in the mid-line. The breadth sounds were coarse, shallow and rapid with hearing of bilateral rales.

The heart was maximally enlarged and the 5th Intercoastal space (ICS) was 1 - 2 cm on outside the mid clavicular line. First and second heart sounds were normal but third heart sound showed the presence of gallop. Tachycardia was present.

The abdomen was obese and flabby. The liver was slightly enlarged span of 19cm. The stool home test was negative. She had a left lower leg amputation and the right leg to the knee and scar occurred above the knee joint 2-3+

The neurological examination was negative; there was no peripheral neuropathy. The oral temperature was 33°C, the pulse was 104 and the respiratory rate 28. The blood pressure was 160/100 mm Hg.

Laboratory Investigations

Laboratory blood data showed Hematocrit (HCT) 29%, hemoglobin (HBG) 9.6 mg%, sugar 270gm%, Blood urea nitrogen (BUN) 63mg%, Uric acid 10.1mg%, Alkaline phosphatase 116mu/ml, Total protein 7.2mg%, Lactic acid dehydrogenase 254mu/ml. Urinalysis showed Proteinuria (Protein in Urine). (Table 1).

Management

To digitalize an adult such as the elderly woman in question, she was given 1.0mg Digoxin tablet initially digitalization. Since the Absorption, distribution, Metabolism and Excretion by Digoxin are rapid, this permits vigorous therapy without the danger of prolonged toxicity. The appearance of earlier gastrointestinal symptoms such as Anorexia, Nausea and vomiting in the patient in question served as a warning of toxicity of digitalization with digoxin. This warning also lessened the danger of serious arrhythmia's and Cardiac damage. Auer *et al* 1992).

Treatment of Digoxin Toxicity

The patient had a chronic renal failure associated with hyperkalemia. Thus Potassium chloride was not used in treating the digoxin toxicity. The patient was given atropine sulfate 0.5mg IV bd. the following day to treat digoxin toxicity and heart block, after which the manifest symptoms of digoxin toxicity of gastrointestinal symptoms gradually disappeared.

The following drugs could also be used to treat digoxin toxicity, but only drugs listed 1 - 4 could be used in these patients. Drugs 5 & 6 should be avoided

1. Propranolol, 2. Lidocaine 3. Kilating Agent (EDTA) 4. Diphenylhydantoin 5. Procainamide 6. Quinidine

TABLE 1.
LABORATORY DATA

A. BLOOD INVESTIGATION

BLOOD	PATIENT
HCT*	29% (40)
HGB*	9.6gm% (12-16)
WBC	7,000 cells/cu mm (5,000-10,000)
Sugar	270mg% (60-100)
Blood Urea nitrogen*	63mg% (10-20)
Uric Acid*	10.1mg% (2.5-8)
Na+*	134Meq/L (135-152)
K+*	5.6Meq/L (3.7-5.1)
CL-*	106Meq/L (95-105)
CO_2*	15.8 Meq/L (24-32)
Ca++	9.4mg% (8.5-10.5)
Bil. Total	0.8mg% (0.15-1.0)
Alk. Phos.*	116mu/ml. (30-85)
Total Protein*	7.2mg% (6-8)
Albumin	3.5mg% (3.5-5)
SGOT (AST)	24mu/ml (10-24)
LDH*	25mu/ml (90-200)

B. URINE INVESTIGATION Urinalysis indicated Proteinuria. Protein in form of Albumin and Globulin in the urine.

NOTE. * Indication of Abnormalities. Figures in parentheses indicate the normal values

Drugs such as Diphenylhydantoin is very effective in suppressing digitalis induced ventricular and supra ventricular arrhythmia. It has the advantage over propranolol and lidocaine as it does not depress conduction. Diphenylhdantoin should be administered slowly (not more than 100mg given intraveously in 5 minutes) to prevent hypotension. This dosage may be repeated after 10 to 15 minutes. The oral loading doze of 1g may be given on thee first day and this may be decreased to 600 to 300 mg per day depending on the clinical improvement of the patient. Diphenylhydantoin could be used in the patient in question to treat Digoxin toxicity.

Propranolol has been used and it is sometimes effective in digitalis induced arrhythmia. For intravenous administration, it has to be given at a dose of 1mg per minute. An oral dosage of 10 to 40mg four times a day is usually employed. With intravenous administration of propranolol and Lidocaine, it is mandatory to use ECG monitoring to ascertain the effects of thee drugs. Quinidine and procainamide both depress cardiac activity, making these drugs dangerous to use in cases of digitalis induced arrhythmia thus cannot be used in this patient. Atropine has been used in severe cases of Tachycardia to reduce the vaginal component of the toxicity, thus confirming its rational for use in this patient in the treatment of Digoxin toxicity. (Aguwa 1996).

DISCUSSION

Knowledge of Pathogenesis of congestive heart failure (CHF) (FHS) allows one to easily predict the signs, symptoms and Laboratory findings. Increase in extracellular and plasma volume lead to congested pulmonary and systemic circulation (Cynthia *et al* 1992).

Pulmonary congestion is manifested by varying degrees of shortness of breadth (SOB) (Falase 1997). These include:

1. Dyspnoea on exertion (DOE) even at mild exertion as with the case of the patient reported in this paper.

2. Orthopnoea - Dyspnoea that occurs in the recumbent position and requires levation of the head with one or several pillows to prevent its reoccurrence; as with case of the patient reported in this paper.
3. Paroxysmal nocturnal dyspnoea (PND) - an exaggerated form of orthopnoea that occurs when the patient is abruptly awakened at night with a feeling of suffocation.
4. Dyspnoea at rest 5. Pulmonary Edema - Fluid accumulation within the alveoli.

Only 1 - 3 outlined above were clearly manifested in the elderly patient under discussion. This can help to also confirm that the CHF is actually Heart failing syndrome (HFS), it is acute, it is not chronic or complicated.

Rales and dullness to percussion, usually over the bases of the Lung are common pulmonary findings. Systematically, inadequate perfusion of the skeletal muscles leads to easy Fatigability and weakness. PND could result from redistribution of blood flow to the kidney during recumbence in early course of CHF. Oliguria may occur later as the failure worsens. A host of cerebral symptoms may also be observed and can include impairment of memory, confusion, Insomnia and anxiety. Auer *et al* 1992).

A number of cardiac and systemic physical findings are observed with varying degrees of frequency. An early diastolic third heart sound (S3) is believed to be related to impaired diastolic relaxation of the ventricle and suggests an elevated systolic and diastolic blood pressure. A resting sinus Tachycardia is often present, as in the case of the elderly patient under discussion. Hyperuricemia and diabetes may as well present (Auer *et al* 1992).

Digoxin was mainly used here to treat CHF (FHS). Other digitalis glycosides have also been used as inotropic drugs for the treatment of CHF and other congestive states.

Digoxin when given orally is rapidly absorbed. About 50% or more of the drug is absorbed. Rate of absorption is rapid and fairly complete (Benjamin & Lot 1984).

Digoxin is distributed widely throughout all the body tissues. The drug is less rapidly excreted and this serves as a warning to the patient taking Digoxin. If the patient has been given digoxin during the previous week, the dose should be reduced accordingly (Benjamin & Lot 1984). Because of impaired renal function and excretion in elderly patient as in the case of the patient under discussion, they frequently require lower than the recommended doses. (Ilena *et al* 1992).

The rate of elimination of digoxin like other Cardiac glycosides is closely correlated with the duration of action. Elimination rate is rapid and it is 2 0r 3 days (Auer 1992).

However considerable controversy still remains over the true benefit of Cardiac glycosides such as digoxin in the management of CHF (FHS) particularly in the chronic management (Auer, 1992). The impression and problems list such as Azotemia, Anemia, Chronic renal failure, Hyponatremia, Hyperkalemia, Hyperchloronemia, Hyperorpnea, Hyperphosphatemia, Proteinuria, Lactic Acidosis and Hypertension have been established to be associated with congestive Heart Failure (CHF) or failing heart syndrome (FHS) (Cynthia *et al* 1992).

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