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Case report

Reversible uremic cardiomyopathy - A Case of a young lady with squamous cell cervical carcinoma

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Summary

Dilated Cardiomyopathy (DCM) in adults is mostly an idiopathic disease with a progressive and irreversible course. It usually carries a poor prognosis [1]. Rarely, a reversible metabolic etiology amenable to specific therapy is identified [1]. There are few cardiovascular abnormalities that are commonly encountered in patients with renal impairment; these include LVH, LV dilatation, and LV systolic and diastolic dysfunction [2]. Here, we report a case of 36 year-old female with reversible uremic cardiomyopathy due to obstructive uropathy due to moderately differentiated squamous cell type metastatic cervical carcinoma which completely responded to bilateral ureteric stenting.

Keywords: systolic/diastolic dysfunction, dilated cardiomyopathy, uremia

Background

Dilated cardiomyopathy (DCM) is an idiopathic condition that results from impaired ventricular systolic function, leading to progressive cardiac remodeling and dilatation [1][3]. A reversible form of DCM can develop due to alcohol abuse, pregnancy, hypothyroidism, hyperthyroidism, uremia, illicit drugs, and other endocrine dysfunctions [1] [4] [5]. We report a case of 36-year-old female with uremic Dilated Cardiomyopathy in the background of metastatic, moderately differentiated squamous Cervical Carcinoma. When the cardiomyopathy got reversed with bilateral ureteric stenting, the patient became suitable for palliative chemotherapy.

Case report

A 36-year-old female, diagnosed with moderately differentiated squamous carcinoma of Cervix (FIGO 111a), underwent radical hysterectomy and treated with adjuvant concurrent chemoradiotherapy with cisplatin followed by intracavitatory brachytherapy. After 6 months, she presented with recurrence of the tumour with invasion of bladder and ureters bilaterally. There were secondary deposits in the anterior abdominal wall and mesentery. She developed dyspnea, palpitations and chest discomfort with orthopnea over 2days. There were no other clinical features to suggest neither pneumonia nor pulmonary embolism. There was no history of cardio toxic chemotherapy.

She was tachypnic, but not pale, with the pulse rate of 132bpm. The rhythm was regular with blood pressure of BP 140/90mmHg. JVP was elevated with bilateral pitting ankle edema. There was no calf tenderness. Oxygen saturation was 94 % on air. S1 and S2 were normal; S3gallop was present. There were no murmurs. Bilateral fine crepitations were present. ECG findings were compatible with sinus tachycardia with diffuse T inversions, Troponin I was 0.226 ng/ml. Chest radiograph showed pulmonary edema. 2D echo findings confirmed global hypokinesia with ejection fraction of 30% with no RA, RV dilation, and no pulmonary hypertension with grade 2 MR without any pericardial effusion. The Blood urea was 23.6 mmol/L, serum creatinine was 692 µmol/l with Na^+ 136 meq/dl, K^+ 5.2 meg/dl. Transaminases were high with SGOT – 342 IU/Land SGPT- 365IU/l.

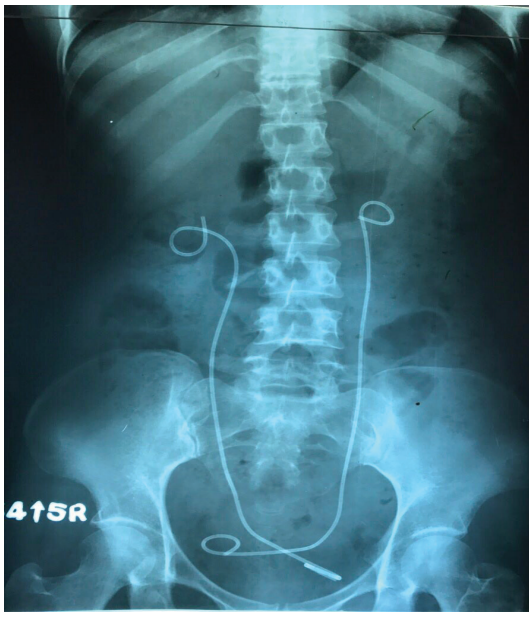

Figure 1: X ray abdomen after placement of bilateral stents.

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Diuretics were administered and the patient has undergone a right ureteric stenting and Percutaneous nephrostomy (PCN) followed up by a similar procedure in the left. Normalisation of renal function was followed up by clinical improvement. Repeat 2 D echo findings in 3 weeks were absolutely normal with EF of 65%. She was planned for a palliative chemotherapy as she was given maximum radiotherapy in the recent past.

Discussion

Dilated cardiomyopathy is defined as decreased systolic function due to dilatation of the heart especially the left ventricle, shown by global hypokineasia and low ejection fraction and sometimes with some diastolic dysfunction [3][6]. It is one of the important causes of congestive cardiac failures. Patients can present with chest pain, heart failure, fainting attack, sudden cardiac arrest. It can be also an incidental finding [3][6][7]. It can be idiopathic or secondary to infections, illicit drugs , myocardial infarction, toxic metabolites, alcohol, endocrine dysfunction, chronic tachycardia and pregnancy[1][3][5] [8]. Some of those causes of DCM can be reversible with interventions, medications. It also can spontaneously resolve or progressively worsen and become irreversible [4].

Prevalence of cardiac abnormalities are very high among uremic patients[9]. Uremic cardiomypathy occurs as a consequence of hemodynamic factors such as volume overload, pressure overload, and anemia[2][9]. Myocardial hypertrophy occur due to volume overload and pressure overload. It is associated with a reduction in the capillary density[18]. It cause ischemia due to an imbalance between supplies and demands of the heart[13] [18]. Ischemia induce apoptosis of myocytes, as well as extracellular matrix and collagen deposition. It leads to interstitial fibrosis and LV stiffness. As a result LV filling pressure increase which leads to impaired diastolic filling, and finally diastolic dysfunction[2][3][14][15]. Increased preload due to volume overload leads to the development of LV dilatation by accumulation of sarcomeres[16] [18]. Also a number of nonhemodynamic factors such as hyperphosphatemia ,uremia itself , excess angiotensin 2 and aldesterone and sympathetic overactivity involve in the development of cardiomyopathy[18][19]. As a result,progressive changes are accumulated in cellular and molecular level and damage the heart. Those are catalytic iron-dependent oxidative stress, inflammatory changes and stimulation of fibrogenic factors such as cardiotrophin-1, galectin-3, transforming growth factor-β(TGFβ) and fibroblast growth factor-23(FGF), and pro-hypertrophic factors[12][19]. The steroid hormone, bufadienolides circulating in the blood and excreted in urine are accumulated in renal failure. It inhibits Na^+-K^+-ATPase. As a result volume expansion and hypertension occur [12].

Impaired diastolic function may occur early in CKD, even without LVH[12][13] . Uremic cardiomyopathy can be reversible with intervention causing normalization of renal functions[11]. In this patient there was no LVH but dilation of heart. Other reversible causes of DCM are hyperthyroidism, hypothyroidism, Cushing syndrome, hypoparathyroidism and alcoholism. They can be reverted with appropriate treatment [7][8][10][12][17].

Learning Points

- Local recurrence of carcinoma of uterine cervix can present with obstructive uropathy
- Renal impairment secondary to obstruction can be reversible with intervention
- Dilated cardiomyopathy can occur in renal failure due to uremia
- Normalisation of renal function would restore the cardiac function

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