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Case Report

Use of SGLT-2 Inhibitor in Decompensated Heart Failure Presenting with Bilateral Resistant Pleural Effusion

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ABSTRACT

Chronic diseases concomitantly decrease quality of life and lifespan expectancy. Novel medications, including Sodium-glucose-co-transporter-2 inhibitors (SGLT-2i), affect both diabetes, heart failure, and diabetic kidney disease. These multisystemic effects give patients with mono systemic and multisystemic diseases new treatment options. Recent studies have shown that SGLT2i may benefit heart failure patients without diabetes. In this case report, we presented a 65-years old patient who was admitted to the emergency room with shortness of breath. Bilateral pleural effusion was observed in the chest X-ray. After etiological studies, empagliflozin was initiated for pleural effusion, which is thought to be related to heart failure. The patient's symptoms declined on the fourth day of the treatment, and the control lung X-ray revealed that the effusions declined.

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Introduction

Chronic diseases constitute a significant group that reduces the quality of life, especially in the middle-advanced age group. New treatment options provide a better quality of life in hypertension, diabetes, kidney, and heart failure. Multiple chronic diseases result in a higher risk of cardiovascular events and death.[1] Treatment options, particularly in low ejection fraction heart failure, include improving health status (symptoms, physical function, and quality of life) reducing the rate of hospitalization and mortality. Sodium-glucose-co-transporter-2 inhibitors (SGLT-2i), introduced as an oral antidiabetic, have been shown to reduce hospitalization rate and improve quality of life regardless of diabetes in patients with heart failure.[2] SGLT-2 inhibitors have proven to be very effective for glycemic control and have adjunctive effects in managing heart failure, hypertension, diabetic nephropathy, and even weight loss.

SGLT2i are effective osmotic diuretics on proximal tubules that maintain heart function by reducing volume load and blood pressure. It affects partly by reducing atrial-natriuretic-peptide and diuretic resistance and inhibiting Na-H modifiers and sympathetic tone.[2] It has also been reported that SGLT2i have anti-inflammatory and anti-fibrotic effects and reduce oxidative stress. Herein, we reported the management of a 65-year old female patient with decompensated heart failure.

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Case Report

A 65-year-old female patient with a history of diabetes, hypertension, chronic renal disease, and tuberculosis was admitted to the emergency room with shortness of breath. She had no fever (36.8 °C), tachycardia (75 beat/min), hypertension (130/80 mmHg), or hypoxemia (SaO_2: 88%). Respiratory sounds were decreased in bilateral middle and lower zones. Chest X-ray shows bilateral pleural effusion extending to the middle zones (*Image 1A*). Computed tomography reports pleural effusion up to fissure level in the right and left lungs. Pleural fluid studies, including; cell count, total protein albumin, glucose, lactate dehydrogenase (LDH), adenosine deaminase (ADA), acid-fast bacilli (AFB), and cytology is, consisted of a transudate. The cytology was reported as reactive inflammatory effusion. Bilateral chest tube drainage was applied for the management of massive effusion.

Repeated AFB and ADA results were negative for tuberculosis. The echocardiographic evaluation of the patient was compatible with heart failure (left heart cavities were dilated, left ventricular global hypokinesis, mild mitral insufficiency, and ejection fraction calculated as 30%). Since the patient's pleural effusion did not regress with diuretic therapy, SGLT2i (empagliflozin 10 mg/day) was started. Pleural effusions decreased, and symptoms were declined in the control lung x-ray of the patient on the fourth day of the treatment. After room air sPO2 was determined as 95%, the patient was discharged with empagliflozin treatment. The outpatient control chest X-ray shows significant regression in pleural effusion (*Image 1B*).

Discussion

SGLT2i in heart failure has been shown to reduce hospitalizations in the acute condition and the chronic period. Ipragliflozin, the first SGLT-2 inhibitor approved in Japan, treatment was started for a patient who had a history of hospitalization at least four times in the last five years. For two years, no hospitalization for heart failure was required during the ipragliflozin treatment.[3] As in our case, signs, and symptoms were regressed with empagliflozin treatment. Bilateral pleural effusion was resolved entirely within three weeks under empagliflozin treatment.

In the analysis of the EMPA-REG study, involving 7020 patients with a high risk of cardiovascular disease, which compared the placebo with empagliflozin (10 mg and 25 mg) groups, it was reported that the need for furosemide was significantly decreased in patients using empagliflozin.[4] Our patient's diuretic requirement was finished entirely after the sixth week of empagliflozin treatment.

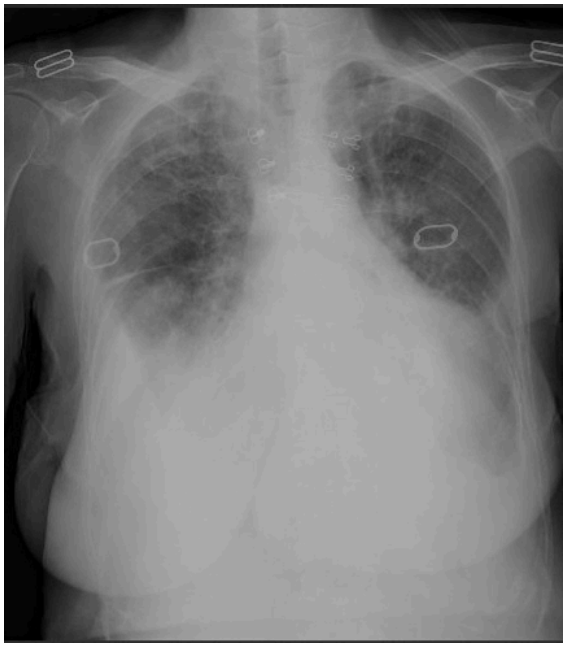

Image 1A. Before the use of SGLT-2 inhibitors.

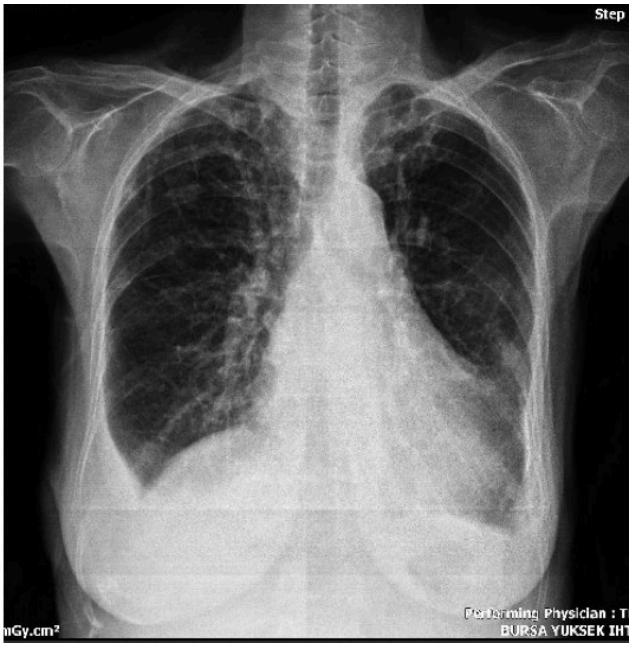

Image 1B. After the use of SGLT-2 inhibitor.

In the RECEDE CHF study conducted with 23 participants, a significant increase in 24-hour urine volume was found on the third and sixth weeks when the empagliflozin group was compared with the placebo group. Empagliflozin with a loop diuretic treatment results in a significant increase in 24-hour urine volume without increasing urinary sodium.[5] Our patient did not require the addition of loop diuretics six weeks after treatment. Since a minimal glomerular filtration rate (GFR) decrease was experienced in the first week, the GFR turned back to the expected value within two weeks.

Acknowledgment

This study has been presented in 18th Uludag Internal Medicine National Winter Congress, 7th Bursa Family Medicine Association National Congress, 12th Uludag Internal Medicine Nursing Congress, 3-6 March 2022, Bursa, Turkey.

Conflict of interest

The authors declared that there are no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Authors' Contribution

Study Conception: NK, BT, EK, UA; Study Design: NK, BT, EK, UA; Supervision: NK; Funding: NK; Materials: NK, BT, EK, UA; Data Collection and/or Processing: NK, BT, EK, UA; Statistical Analysis and/or Data Interpretation: NK, BT, EK, UA; Literature Review: NK, BT, EK, UA; Manuscript Preparation: NK, BT, EK, UA; Critical Review: NK.

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