Heart failure(HF) is a serious condition that develops when heart doesn’t pump enough blood for whole body’s needs. According to the data from CDC, more than 6 million adults in the United States have heart failure. [1]The New York Heart Association (NYHA) functional classification is the most commonly used classification system for HF, based on the limitation of physical activities, the patients were categorized into four classes. The dataset we used is focused on patients of NYHA class III and IV, the most two advanced HF stages, which indicate they are limited or unable to carry on physical activity without discomfort.

Renal dysfunction, also known as worsening renal function is a common complication of heart failure. Generally speaking, the reduced cardiac output and the consequently renal under-perfusion is the main pathophysiology cause of renal dysfunction because it leads to low renal blood flow and increased renal venous pressure.[2] Besides, neurohormonal activation(renin–angiotensin–aldosterone and  sympathetic nervous system), inflammatory activation and diuretic treatment are also mechanisms leading to renal dysfunction in patients with heart failure. [3]On the contrary, renal dysfunction may cause heart failure through mechanisms such as inflammatory activation and anaemia (caused by a depression of renal erythropoietin production). [4]

Therefore, lots of studies have shown worse outcomes in patients with concomitant renal dysfunction and HF, the markers of renal function, such as serum creatinine and blood urea nitrogen have been confirmed as powerful predictor of mortality.[2, 5]

Creatinine is a chemical compound produced from energy-producing processes in human muscles and excreted into the serum. Serum creatinine can be filtered out of the blood by health kidneys. Hence, the amount of creatinine in blood is relatively stable for healthy people. Serum creatinine could be used to calculate the glomerular filtration rate, which is an important indicator of the kidney function. [6]Serum creatinine level is tested through venous blood, and have become routine clinical practice because of efficiency and easy procedures.

We also regard the serum sodium and platelets as secondary exposure.

In some studies, low serum sodium (hyponatremia) is a prognostic marker in heart failure. Hyponatremia is associated with activation of vasoactive neurohormonal systems. [7] Activation of these system leads to systemic vasoconstriction and an increase in heart rate and cardiac contractility. However, the constant activation of these systems is a causal effect of HF through excess oxygen consumption and a promotion of myocyte loss and fibrosis.[8]

Platelet abnormalities in HF have been well discussed,  there is an increasing platelet aggregation in heart failure patients’ blood. An increase in vascular resistance in heart failure may cause abnormal platelet aggregates. Besides, the activated sympathetic nervous system is also a possible mechanism of activation of platelets. [9]However, several studies indicate that platelet aggregation in stable heart failure patients is not significant to predict cardiovascular events and mortality, we want to determine in our project.[10]

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