

## A Case Report of Dextrocardia with Hypertensive Emergency and Congestive Heart Failure: A Rare Condition

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### ABSTRACT

**Introduction :** Dextrocardia is a rare congenital anomaly affecting the heart. The range of cardiac problems linked to dextrocardia is extensive. This case report seeks to enhance the current knowledge by providing a clinical description of dextrocardia accompanied by hypertensive emergency and congestive heart failure. **Case Presentation :** A 39-year-old woman came to the Emergency Department of RSI Sultan Hadirin Jepara, having vomitted more than ten times since last night. Vomiting was preceded by nausea. The patient has been experiencing shortness of breath for three years, particularly when sleeping with only one pillow, necessitating the use of multiple pillows. Through additional physical and supporting examinations, the patient was diagnosed with dextrocardia with hypertensive emergency and congestive heart failure. **Conclusion :** Dextrocardia is a rare birth defect characterized by a variety of complicated structural abnormalities in the heart. There is no difference in symptoms and signs in patients with congestive heart failure in dextrocardia or levocardia. So that the approach to managing congestive heart failure in our patient is identical. Gaining a comprehensive understanding of this uncommon ailment is essential for delivering suitable medical treatment and effectively managing any problems in affected persons.

**Keyword:** dextrocardia; CHF, hypertension

## **INTRODUCTION**

Dextrocardia is the term used to describe the displacement of the heart towards the right side of the thorax. This includes mirror-image dextrocardia, heart dextroversion, and heart dextroposition.<sup>1</sup> Dextrocardia is a rare congenital heart defect that is believed to occur in approximately 1 in 8000 to 25,000 live births.<sup>2</sup> No specific ethnic or gender-related preference has been reported for dextrocardia.<sup>3,4</sup> Dextrocardia is more prevalent in individuals with situs solitus, situs ambiguous, and situs inversus totalis.<sup>4</sup> Structural cardiac abnormalities were observed in all cases of situs ambiguous, in 80% of cases with situs solitus, and in 25% of cases with situs inversus. The spectrum of cardiac anomalies associated with dextrocardia is wide. The broad range of complicated cardiac abnormalities are linked to embryonic dextrocardia. The precise cause of dextrocardia is currently unknown. However, it is believed to be a result of the aberrant positioning of the organs during embryonic

development.<sup>3,4</sup> Isolated dextrocardia is asymptomatic; however, dextrocardia with situs inversus and Kartagener syndrome may exhibit symptoms that vary based on other abnormalities.<sup>3</sup> Often, the diagnosis is fortuitous. The scope of management encompasses medical treatment, cardiac surgery, other surgical procedures, and the management of post-operative problems.<sup>5</sup>

The complications associated with dextrocardia include poor growth and development, inability to breathe properly, serious infections, male infertility in Kartagener syndrome, recurring sinus and lung infections, intestine blockage caused by abnormal rotation, and congestive heart failure (CHF).<sup>6</sup> Approximately 64.3 million individuals globally are now experiencing CHF. The estimated prevalence of diagnosed heart failure in developed countries typically ranges from 1% to 2% of the overall adult population.<sup>7</sup> CHF remains the primary cause of hospitalization in the United States for individuals aged 65 years and

beyond, with over 1 million patients admitted annually. CHF still causes almost 300,000 deaths annually, despite advancements in diagnosis and therapy.<sup>8</sup> Heart failure is a medical condition marked by specific symptoms (such as difficulty breathing, swelling in the ankles, and fatigue) that may be accompanied by observable signs (such as increased pressure in the jugular veins, crackling sounds in the lungs, and swelling in the extremities).<sup>9,10</sup> The four primary causes that account for approximately two-thirds of cases of congestive heart failure (CHF) are ischemic heart disease, chronic obstructive pulmonary disease (COPD), rheumatic heart disease, and hypertensive heart disease.<sup>7</sup>

Elevated blood pressure induces mechanical strain through heightened afterload and neurohormonal alterations that lead to an augmentation in ventricular mass. Hypertension is closely linked to other comorbidities that contribute to the development of CHF.<sup>7</sup> Hypertensive urgency is described as a condition without any damage to the organs, while hypertensive

emergency is classified as a condition with a risk of death, as shown by damage to the organs.<sup>11</sup> Hypertensive emergency is a critical condition that poses a risk to life and has been associated with higher rates of hospital admission. The number of emergency department (ED) visits for hypertensive emergency has doubled per million adults from 2006 to 2013, according to estimates.<sup>12</sup> This case report aims to contribute to the existing knowledge base by presenting a clinical description of dextrocardia with hypertensive emergency and congestive heart failure, emphasizing the rarity of this condition and its management.

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### **CASE PRESENTATION**

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A 39-year-old woman came to the Emergency Department of RSI Sultan Hadirin Jepara, having vomitted more than ten times since last night. Vomiting was preceded by nausea. After vomiting, the patient felt abdominal pain around the navel. The patient admitted that he had previously eaten normally. The patient has been experiencing shortness of breath for three years,

particularly when sleeping with only one pillow, necessitating the use of multiple pillows. Additionally, the patient experiences shortness of breath during activities, such as sweeping or mopping. The patient frequently woke up in the middle of the night due to sudden shortness of breath, which made it difficult for her to sleep. The patient denied experiencing chest pain, but occasionally experienced cold sweats. Three years ago, the patient received treatment for shortness of breath but did not take regular medication. The patient also underwent echocardiography three years ago, but the results were still good. A history of hypertension following the birth of the second child was found. The patient prefers consuming savory/salty foods. Eight hours prior to hospital admission, the patient urinated in a small volume.

The patient was weak, compositis, tachycardic with a regular pulse rate of 107 beats/min, high blood pressure 235/150 mmHg, and tachypnea with respiratory rate of 24 cycles/min. On physical examination, ronchi were found in both

hemithorax, ictus cordis (+) dilatation (+), the left border of the heart is at ICS V, linea midclavícula dextra, approximately 4 cm laterally (sign of enlargement), on auscultation, pansystolic murmur was heard on tricuspid valve and S3 and murmur were heard on mitral valve. On abdominal examination, umbilical and epigastric pain were found. The laboratory tests revealed leukocytosis (13,400/ $\mu$ L) and hyperuricemia (8.2 mg/dL)(Table 2). Renal function and CKMB, blood glucose were normal (Tables 2). The electrocardiogram (ECG) showed negative P wave in leads aVL and I but positive P wave in lead aVR, right axis deviation (leads II, III and aVF are positive; leads I and aVL are negative), and dominant S waves throughout on precordial leads (Figure 1). Chest X-ray showed dextrocardia (Figure 2). Our patient was diagnosed with Hypertensive emergency + Dextrocardia + Congestive heart failure + Hyperuricaemia + Dyslipidemia.

In the emergency department, the patient was started promptly on treatment with Asering 20

drops/minute, Ranitidine 50 mg injection every 8 hours, Ondancetron 4 mg injection every 12 hours, Candesartan 16 mg every 24 hours, Amlodipin 10 mg every 24 hours, and a soft low-sodium diet. The treatment was continued according to the advice of internist. Asering fluid 20 drops/minute, a soft low-sodium diet, Ranitidine 50 mg injection every 8 hours, Ondancetron 4 mg injection every 12 hours, Clonidin 0.15 mg (Extra), Nicardipine 2.5 mg/hour if systolic blood pressure >200 mmHg, if blood pressure drops Lisinopril 10 mg/24 hours and Amlodipin 10 mg every 24 hours were given.

Monitoring on the second days of treatment, the patient still complained of nausea and vomiting, but her condition had improved. The blood pressure was still high, 160/100 mmHg, respiratory rate was 22 cycles/min. Asering fluid was changed to RL, 10 drops/min. We continued the management from the previous day and added a few medications, including Furosemid 20 mg every 12 hours, Nicardipine 2.5-10 mg/hour tapering down to 2.5 mg/hour via syringe pump,

Cefotaxime 1 gr every 12 hours, Candesartan 16mg/24 hours, ISDN 5 mg every 8 hours, Sucralfat 10 cc every 8 hours, Betahistin 6 mg every 8 hours, Flunarizin 10 mg every 12 hours, and Dimenhydrinat 50 mg every 8 hours.

Monitoring on the third day of treatment, the patient still complained of dizziness. Blood pressure was 160/90 mmHg and heart rate was 112 beats/min. Alprazolam 0.5 mg/24 hours was given if patient had insomnia. Check lipid profile was ordered. Monitoring on the fourth day of treatment, the patient was still dizzy. However, the blood pressure was decreased (130/70 mmHg). Atorvastatin 20 mg/12 hours was given. The patient improved on the fifth day of treatment, allowing for outpatient care. The patient received Amlodipine 10 mg (1x1), Betahistine 6 mg (1x1), Candesartan 16 mg (1x1), Dimenhidrinat (3x1), Levofloxacin 500 mg (1x1), Nitrokaf retard cap (2x1), Paracetamol 500 mg (1x1), Spironolactone 25 mg (1x1), Vertilon (3x1), and Tranexamat acid (3x1). Patients were given education to return to have regular follow-up.

**Table 1. Laboratory tests.**

| No | Test          | Results | Reference Value |
|----|---------------|---------|-----------------|
| 1  | CKMB          | 14      | <24             |
| 2  | Haemoglobin   | 14      | 12-16           |
| 3  | Blood glucose | 114     | 80-160          |
| 4  | Creatinine    | 1.1     | 0.5-1.0         |
| 5  | Ureum         | 23      | 10-50           |
| 6  | Thrombocyte   | 264,000 | 150,000-400,000 |
| 7  | Leucocytes    | 13,400  | 4,000-10,000    |
| 8  | Hematocrit    | 42.7    | 37-43           |
| 9  | Ureum         | 23      | 10-50           |
| 10 | Uric acid     | 8.2     | 2.4-5.7         |
| 11 | Basophil      | 0       | 0-1             |
| 12 | Lymphocytes   | 8       | 20-40           |
| 13 | Monocytes     | 2       | 2-6             |
| 14 | N. Segment    | 90      | 50-70           |

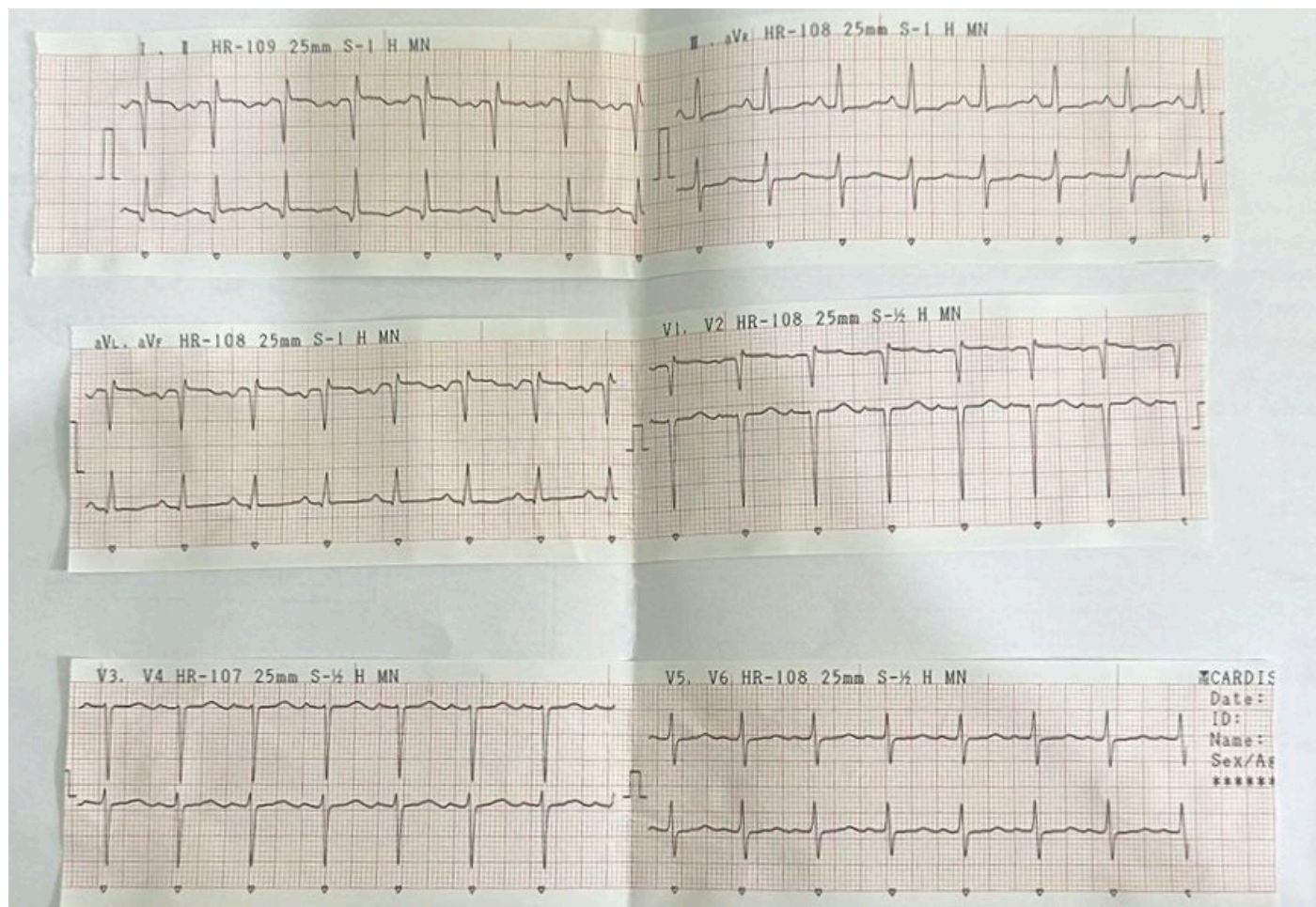


Figure 1. ECG

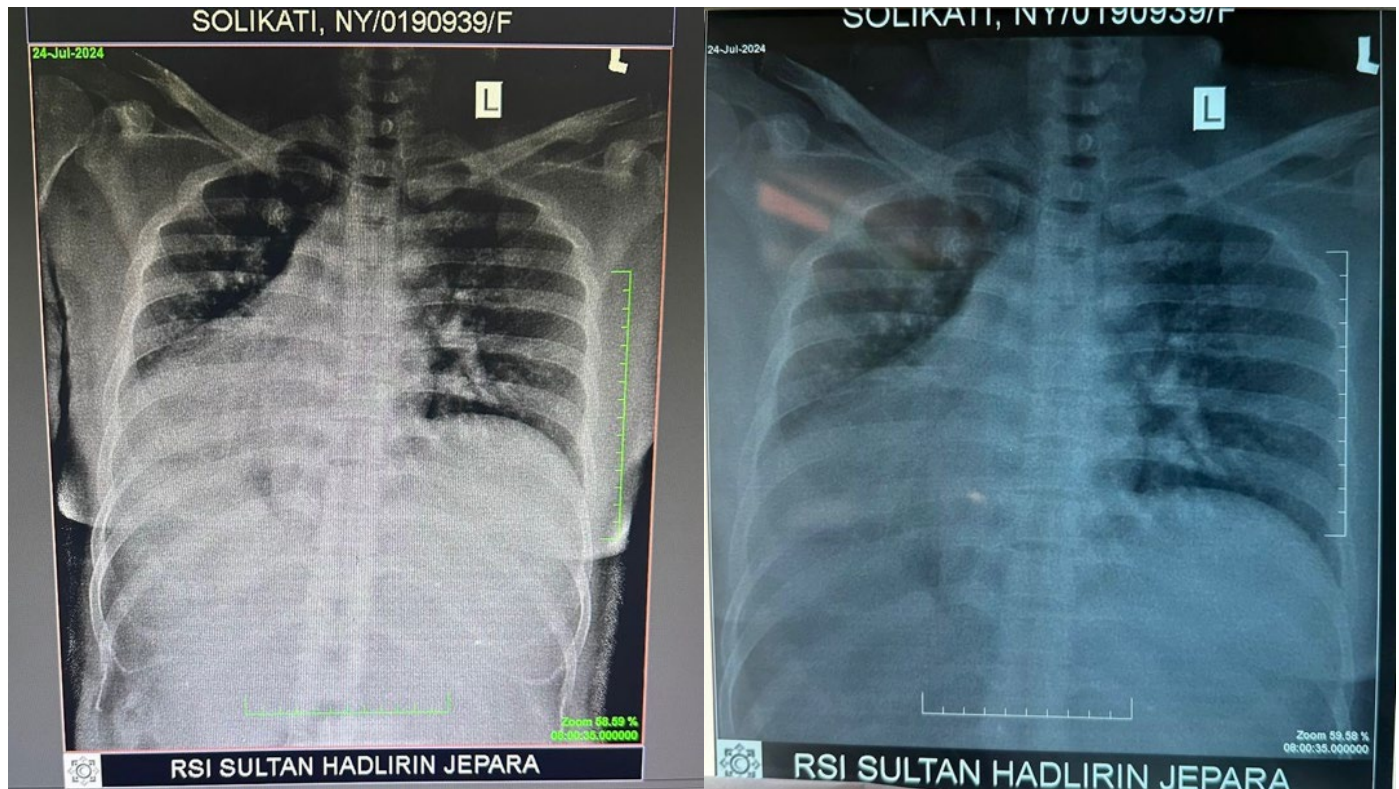


Figure 2. Chest X-ray.

## DISCUSSION

Dextrocardia is a congenital anomaly characterized by the positioning of the heart in the right half of the chest cavity.<sup>4</sup> Dextrocardia is an exceptionally uncommon medical conditions, with studies indicating an incidence rate of approximately 1 in 12,000 pregnancies.<sup>3</sup> In order to categorize the diagnosis, it is necessary to ascertain the situs type. Situs refers to the spatial arrangement of structures within the human body.<sup>4</sup> The

prevalence of primary dextrocardia is highest in individuals with situs solitus (45.4%), followed by situs ambiguous (36.3%), and finally situs inversus totalis (18.1%).<sup>3</sup> Previous analysis found that 56.4% of patients were male, which aligns with the findings of Squarcia et al. (70.0%), Garg et al. (65.6%), Tripathi et al. (56.7%), and Evans et al. (57.4%). This indicates a consistent gender distribution across multiple studies.<sup>13</sup>

Dextrocardia is frequently linked with situs inversus rather than situs solitus or situs ambiguus, as indicated in numerous studies. Nevertheless, heart abnormalities are frequently linked to situs solitus. Situs inversus with dextrocardia is more commonly found in individuals with a structurally normal heart and a normal life expectancy, which might make it difficult to identify.<sup>14</sup> Individuals with dextrocardia with situs inversus typically do not require targeted therapy unless they experience symptoms or have a treatable congenital cardiac abnormality.<sup>15</sup>

The cause of dextrocardia varies depending on whether it is primary or secondary in nature. Primary dextrocardia is exclusively present from birth, whereas secondary dextrocardia can occur either from birth or later in life. The precise cause for the heart's orientation towards the right side of the chest, rather than the left side, remains unknown. However, it is believed to be a result of the anomalous positioning of other organs during the developmental process.<sup>16</sup> In cases where dextrocardia coexists with situs inversus, there is a

3-5% probability of encountering other congenital cardiac abnormalities, usually characterized by the transposition of the major blood arteries. Kartagener syndrome, a condition characterized by situs inversus totalis, sinusitis, and bronchiectasis, is observed in individuals with primary ciliary dyskinesia.<sup>16,17</sup>

In a different form of dextrocardia, the atypical positioning of the heart is accompanied by the atypical arrangement of other organs in the thoracic and abdominal area, a condition known as heterotaxy. The condition is referred to as heterotaxy syndrome and is a consequence of a disturbed orientation of the left-right axis. This condition is characterized by a significant number of abdominal and cardiac defects, such as asplenia, faulty gallbladder system, lung problems, and severe heart and blood vessel defects, including anomalous pulmonary venous connections and systemic venous abnormalities. It is a highly serious disorder.<sup>16,18</sup>

Isolated dextrocardia typically does not show any symptoms. However, dextrocardia with situs

inversus and Kartagener syndrome may exhibit symptoms depending on associated abnormalities. These symptoms can include cyanosis, dyspnea, failure to thrive, fatigue, jaundice, pallor, decreased exercise tolerance, recurrent sinus or lung infections, hydrocephalus, arrhythmias (particularly atrioventricular blocks), and intestinal obstruction.<sup>3,6,19</sup> Previous study reported the most prevalent symptom observed was respiratory distress, reported in 87.5% of cases. Cyanosis was the second most common symptom, occurring in 25% of cases. Passing of clay-colored feces was reported in 12.5% of cases. The first two symptoms were evenly distributed among both boys and girls, but the last symptom was observed exclusively in girls.<sup>5</sup> Upon physical examination, a patient with dextrocardia may exhibit clubbing, cyanosis, a pronounced apical impulse on the right side of the chest, and easily audible heart sounds on the right side of the chest.<sup>3,6,19</sup> Our patient did not show these symptoms. However, on physical examination,

our patient pronounced apical impulse on the right side of the chest.

It is frequently diagnosed as an accidental finding during standard radiological examinations, which reveal an aberrant positioning of the heart. Dextrocardia can be detected by an ECG by seeing right axis deviation, inversion of all complexes in lead I, an upright p wave in AVL, and the absence of R wave progression in anterior leads.<sup>3,15</sup> The ECG of our patient revealed negative P wave in leads aVL and I but positive P wave in lead aVR, right axis deviation (leads II, III and aVF are positive; leads I and aVL are negative), and dominant S waves throughout on precordial leads.

The Centers for Disease Control and Prevention reports that around 70 million American adults, which is around 29% of the population, suffer from hypertension. However, only about half of them, specifically 52%, manage to keep their blood pressure under control. If left unmanaged over an extended period of time, hypertension can result in harm to critical organs, specifically the cardiovascular,

neurological, and renal systems.<sup>20</sup> Severe hypertension can cause kidney damage, which can manifest as acute oliguria or any of the common symptoms of renal failure, such as nausea/vomiting, loss of appetite, changes in mental state, uremic pericarditis, or swelling.<sup>21</sup> Our patient came with vomiting and nausea. Our patient also had uncontrolled hypertension. This can lead to heart failure.

Heart failure is becoming an increasingly significant health and economic challenge for the United States, mostly due to the aging population. From 2012 onwards, the age-adjusted death rate per person with HF experienced its first increase in the United States. A review conducted in the United States revealed that the overall number of deaths attributed to heart failure (HF) has risen from 275,000 in 2009 to 310,000 in 2014.<sup>7,22,23</sup> Most patients with dextrocardia are asymptomatic and lead a normal life. The management of dextrocardia varies depending on the presence of other congenital anomalies or conditions. Our patient had congestive heart

failure, according to AHA guideline, guideline-directed medical therapy (GDMT) can be given including renin-angiotensin system inhibition (ACE-I, ARB, ARNi), beta-blocker, mineralocorticoid receptor antagonists (MRAs) and sodium-glucose cotransporter 2 inhibitors (SGLT-2 Inhibitors).<sup>22</sup>

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### CONCLUSION

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Dextrocardia is a rare birth defect characterized by a variety of complicated structural abnormalities in the heart. There is no difference in symptoms and signs in patients with congestive heart failure in dextrocardia or levocardia. So that the approach to managing congestive heart failure in our patient is identical. Gaining a comprehensive understanding of this uncommon ailment is essential for delivering suitable medical treatment and effectively managing any problems in affected persons.

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