CARDIO ROUNDS NEWSLETTER

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IN THIS ISSUE

Dear Doctor,

Greetings from Micro Labs Limited. We are happy to bring you "Cardio rounds" which contains latest and interesting news in the field of Cardiology

CORRELATION BETWEEN LOW AND HIGH SERUM CHLORIDE LEVELS AND IN-HOSPITAL MORTALITY IN HEART FAILURE PATIENTS

INTRODUCTION-Congestive heart failure is a serious clinical illness characterized by defective cardiac anatomy and/or function. Common causes of CHF include inadequate ventricular pumping or filling capacity, inability of cardiac blood output to satisfy the body's metabolic demands, tissue and organ hypoperfusion, and simultaneous pulmonary and systemic congestion.[1] In recent years, electrolyte abnormalities have received a lot of attention as a prevalent pathophysiological aberration in heart failure. An important electrolyte found in extracellular fluids and plasma was serum chloride (Cl^-). Reduced blood chloride levels have been linked to a number of adverse health consequences, including cancer, metabolic syndrome, kidney disease, cardiovascular disease (CVD), and hypertension, according to numerous epidemiological studies. The relationship between serum chloride levels and mortality within the hospital in patients with heart failure was not well understood. This study's goal was to investigate the relationship between serum chloride levels—both high and low—and in-hospital mortality in patients with congestive heart failure who were admitted to the intensive care unit.[2]

CARDIOVASCULAR DISEASE

METHODS-This retrospective study analysed clinical data from the Medical Information Mart for Intensive Care database. There were 15,983 participants in this study. Age, race, sex, severity score (Sequential Organ Failure Assessment, or SOFA) and APSIII scores], comorbidities (such as diabetes, hepatic failure (HepF), acute myocardial infarction (AMI), and chronic obstructive pulmonary disease (COPD), drug information (e.g., norepinephrine, dopamine, epinephrine, phenylephrine, and vasopressin), mechanical ventilation and intubation, and vital signs initially recorded on ICU admission (heart rate, respiratory rate, systolic blood pressure, and temperature) were among the demographic characteristics. Anion gap (AG), chloride, blood urea nitrogen (BUN), calcium, creatinine, potassium, sodium, mean corpuscular haemoglobin (MCH), mean corpuscular haemoglobin concentration (MCHC), mean corpuscular volume (MCV), platelet count, haemoglobin (Hb), red blood cell distribution width (RDW), red blood cell (RBC) count, and white blood cell (WBC) count were among the laboratory test indexes that were also gathered upon admission. Vital signs and demographic data were noted within the first twenty-four hours following admission, and the outcomes of the initial assessment upon admission were chosen as markers for the laboratory examination.

The primary endpoint was in-hospital mortality, and the study group was made up of patients who were classified according to their serum chloride levels.

RESULTS- It was shown that the lowest serum chloride level (less than 94 mmol/L) was linked to a higher risk of death in the unadjusted model. When comparing serum chloride levels of less than 94 mmol/L to those of 103-105 mmol/L, the corresponding OR for death was 1.77 (95% CI: 1.47-2.13). There was also a significant correlation found between the highest blood chloride level (109 mmol/L) and a higher risk of mortality [OR= 1.31 (95% CI: 1.09-1.57)]. Adjustments were made to a multivariable model for blood biochemical indicators (AG, BUN, creatinine, Hb, MCH, MCHC, MCV, platelet count, RBC, RDW, and WBC), basic vital signs (temperature, respiratory rate, heart rate, and SBP), complicating diseases (COPD, AMI, MC, diabetes, and HepF), body mass index, and medication usage (norepinephrine, dopamine, epinephrine, phenylephrine, and vasopressin). Lower serum chloride levels (less than 94 mmol/L) were linked to a higher mortality risk. When comparing serum chloride levels of less than 94 mmol/L to those of 103–105 mmol/L, the associated OR for death was 1.36 (95% CI: 1.08–1.71). Additionally, there was a significant correlation found between the highest blood chloride level (109 mmol/L) and a higher risk of mortality [OR = 1.25 (95% CI: 1-1.56)]. A U-shaped relationship, controlling for potential confounding variables, has been identified between serum chloride levels and in-hospital mortality in patients with CHF (Figure 1). Serum chloride concentrations and the probability of in-hospital mortality were specifically correlated negatively up to a threshold of 105.017 mmol/L [OR = 0.969 (95% CI: 0.957-0.982)]. Nevertheless, there was a significant increase in the probability of in-hospital death [OR = 1.039 (95% CI: 1.002-1.076)] above this threshold, which was exceeded by 8 hours.

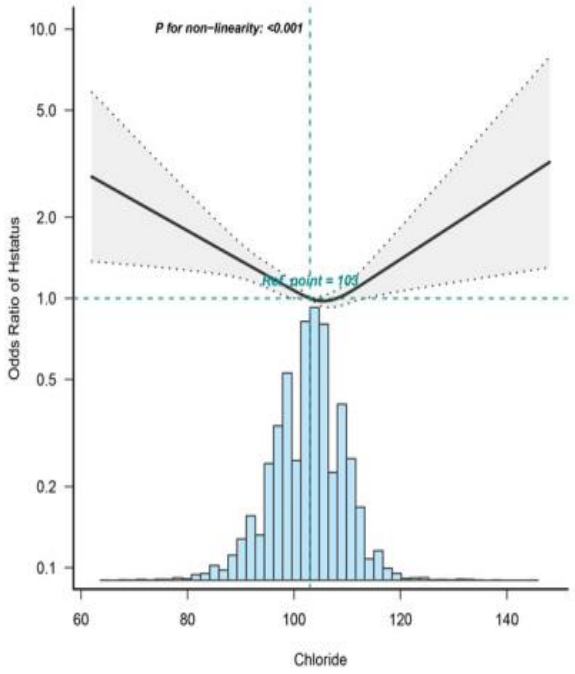

Figure 1. A U-shaped relationship, controlling for potential confounding variables, has been identified between serum chloride levels and in-hospital mortality in patients with CHF

DISCUSSION-The current study showed that serum chloride levels, both high and low, were substantially linked to higher mortality rates among CHF patients in the intensive care unit.[2] Similar to this, Cuthbert JJ et al. showed that even after a thorough adjustment for a variety of covariates, there was a substantial correlation between low admission serum chloride levels and mortality in heart failure patients.[3]

CONCLUSION-The current study concluded that a strong correlation was found between increased mortality risk and serum chloride levels, both high and low, in patients with CHF

admitted to the intensive care unit. More specifically, in this patient cohort, the study found a U-shaped connection between serum chloride levels and in-hospital mortality.

Serum chloride levels and in-hospital mortality in heart failure patients had a U-shaped association, with an inflection point around 105.017 mmol/L.

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RELATIONSHIP BETWEEN MORTALITY IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION AND 24-HOUR BLOOD PRESSURE VARIABILITY

INTRODUCTION-Acute myocardial infarction (AMI) is a severe cardiovascular condition that causes significant morbidity and death. In AMI, ischemia and necrosis impact certain cardiomyocytes, resulting in a reduction in myocardial contractility, which was followed by an immediate proinflammatory response and elevated sympathetic tone. Meanwhile, excessive blood pressure variability (BPV) increases left ventricular workload, heart rate, myocardial oxygen demand, and promotes inflammation and endothelial dysfunction. As a result, a high BPV and its accompanying pathological consequences were likely to exacerbate the physiological function of the heart and influence the formation of acute cardiac problems in AMI patients.[1] The mortality rate among AMI patients has not decreased considerably in recent years. The correlation between blood pressure variability (BPV) and acute myocardial infarction (AMI) remains unclear. The aim of the study was to investigate the correlation between 24-hour BPV and death in AMI patients.[2]

METHODS-This was a retrospective cohort study and enrolled 1291 patients with AMI, including 475 females. The MIMIC-IV 2.0 database provided the patient's data. The following three factors were required for inclusion: (1) an AMI diagnosis; (2) age more than 18; and (3) a first ICU hospitalisation. Age, sex, race, length of stay in the intensive care unit, comorbidities, clinical conditions (hypertension, diabetes, acute heart failure, cardiogenic shock, cardiac arrest), first 24-hour blood pressure recordings after admission, and survival outcomes (all-cause mortality at 30 days, 1 year, and 3 years) were among the patient data extracted for this study from the MIMIC-IV 2.0 database. All disease diagnoses were categorised using the International Classification of Diseases, Ninth and Tenth Editions (ICD-

9 and ICD-10). In this investigation, average real variability (ARV) was used to evaluate BPV. The key outcome measures were 30-day, 1-year, and 3-year all-cause mortality rates.

RESULTS-Four groups of patients were created based on the diastolic blood pressure (DBP)-ARV characteristics. The 30-day, 1-year, and 3-year death rates across the four groups showed significant differences ($p = 0.02$, $p < 0.001$, and $p < 0.001$, respectively). Systolic blood pressure (SBP)-ARV could not predict AMI patient mortality after confounding factor adjustment ($p > 0.05$). However, the highest DBP-ARV was significantly linked to higher 30-day mortality (HR: 2.291, 95% CI 1.260-4.168), 1–year mortality (HR: 1.933, 95% CI 1.316-2.840), and 3–year mortality (HR: 1.743, 95% CI 1.235-2.461). The Kaplan-Meier graphs showed that patients in the highest ARV group had considerably poorer long-term survival probability than patients in other groups, irrespective of SBP or DBP. When DBP-ARV <8.04, RCS curves demonstrated that the mortality risk of patients with AMI initially declined and subsequently rose with an increase in ARV. When DBP-ARV >8.04, the 30-day mortality risk elevated and then reduced, while the 1- and 3-year death rates climbed and then stabilized with ARV rise (Figure 1).

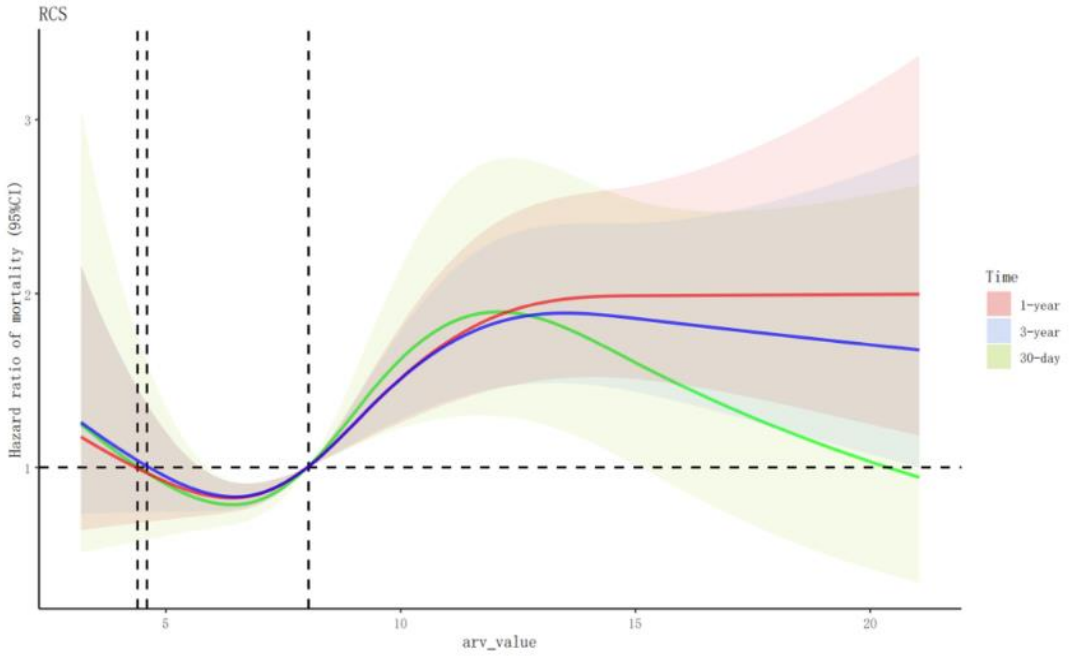

Figure 1. Restricted cubic spline (RCS) curves for mortality risk based on diastolic blood pressure average real variability (ARV) values. CI-confidence interval.

DISCUSSION-The current study showed that DBP-ARV was a significant risk factor for increased mortality in patients with AMI.[2] Similar to this study, Hassan AKM et al. showed that systolic and diastolic BPV duration of the daytime and night-time interval (SD_{dn}) were the only independent predictors of in-hospital major adverse cardiovascular events (MACEs) in patients with ACS, including those with or without ST-segment elevation myocardial infarction and hypertension. A receiver operating characteristic curve analysis revealed that SBP-SDdn has the highest specificity (98.4%) for predicting MACEs in hospitals, with a sensitivity of 47.3% at a threshold of 12.6 mmHg.[3]

CONCLUSION-The current study concluded that this investigation shed light on the connection between BPV and mortality risks in AMI patients. To further understand the underlying processes of BPV and to assess the predictive usefulness of short- and long-term BPV for cardiovascular events and mortality risk, more clinical research was required. Additionally, the association between long-term BPV and prognosis in patients with AMI has to be explored.

If a patient's DBP or ARV was higher or lower within the first 24 hours in the intensive care unit, they might be at a higher risk of both short- and long-term mortality.

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EFFECT OF ATHEROSCLEROTIC VASCULAR DISEASE BURDEN ON THE RISK OF VASCULAR EVENTS IN PATIENTS WITH RECENT ISCHEMIC STROKE AND ATRIAL FIBRILLATION

INTRODUCTION-Atrial fibrillation (AF) is the most prevalent cardiac arrhythmia, with a 5-fold higher risk of thromboembolic stroke and a 2-fold risk of all-cause death. AF is commonly associated with atherosclerotic vascular disease (ASVD), and the two disorders share a number of vascular risk factors such as hypertension, diabetes, obesity, and heart failure. Patients with AF were more likely to suffer coronary artery disease (CAD) than healthy individuals with normal sinus rhythm. Individuals with AF were more likely to develop peripheral artery disease (PAD), which increases their risk of stroke, cardiovascular events, and death.[1] Recurrent vascular events were more common in patients with atrial fibrillation (AF) and ischemic stroke (IS). In individuals who have had an ischemic stroke and AF during the last 90 days, this study assessed the effect of the burden of atherosclerotic vascular disease in various vascular areas on the risk of vascular events.[2]

METHODS-This was a prospective observational study and included 2148 patients with IS and AF from the International RAF network in a prospective 90-day follow-up. Atherosclerotic vascular disease was identified by at least one of the following: Symptomatic ischemic heart disease, symptomatic peripheral artery disease, internal carotid stenosis ≥50%, or the presence of plaques in the aorta. The primary outcome was a composite of stroke, transient ischemic attack, systemic embolism, cerebral bleeding, and major extra cranial bleeding within 90 days post-acute stroke. Patients were categorized into 5 groups based on the number of affected

atherosclerotic vascular territories, with those with no atherosclerotic vascular disease as the reference.

RESULTS-The current study results showed that atherosclerosis affected 744 people (34.6%). A higher baseline NIHSS-score at admission, diabetes mellitus, hypertension, prior stroke/TIA, congestive heart failure, current smoking, and leukoaraiosis were all more common in older patients with AVD. Following the index stroke, patients without atherosclerosis were more likely to be prescribed oral anticoagulant medication. Patients with AVD were treated as follows: 441 (20.5%) patients received acute revascularization (rt-PA or mechanical thrombectomy, or both) less frequently than patients who received oral anticoagulation therapy (76.6%, P <0.001), and statins were more frequently given as pretreatment at admission (35.2%, P <0.001) and aspirin after the index event (16.1%, P <0.001). Following an acute stroke, NOACs were used to treat 344 patients with AVD (46.2%): 126 (36.6%) received rivaroxaban, 119 (34.5%) received Apixaban, and 99 (28.7%) received Dabigatran. The findings of the univariate analysis for the AVD-related variables. The following factors were substantially linked to atherosclerosis: history of stroke/TIA 237, hypertension 652, hyperlipidemia 342, and male sex 407. Variables that were independently correlated with AVD on multivariate analysis. The combined result was noted in 97 (6.9%) no-AVD patients and 77 (10.3%) AVD patients throughout a 90-day follow-up period (HR 1.22, 95% Cl: 0.87-1.92). 33 (11.3%), 26 (14.7%), 26 (15.8%) and 35 (9.7%) patients with AVD had a history of ischemic heart disease with symptoms, PAD, aortic plaques, and carotid stenosis. For all groups, there was overlap in the Kaplan Meier Curve for the combined outcomes in patients with and without AVD. Compared to patients without AVD, those with AVD affecting three or four vascular areas showed a significantly higher risk of composite vascular outcomes in the multivariable analysis. In particular, the hazard ratio (HR) was 2.80 (95% CI: 1.20–6.53) for three territories involved and 6.81 (95% CI: 1.02–36.24) for four territories (Figure 1). Furthermore, when three territories were involved (HR 2.88, 95% CI: 1.04–7.95), and much more so when four territories were involved (HR 7.76, 95% CI: 1.22–49.23), the risk for ischemic outcomes increased dramatically.

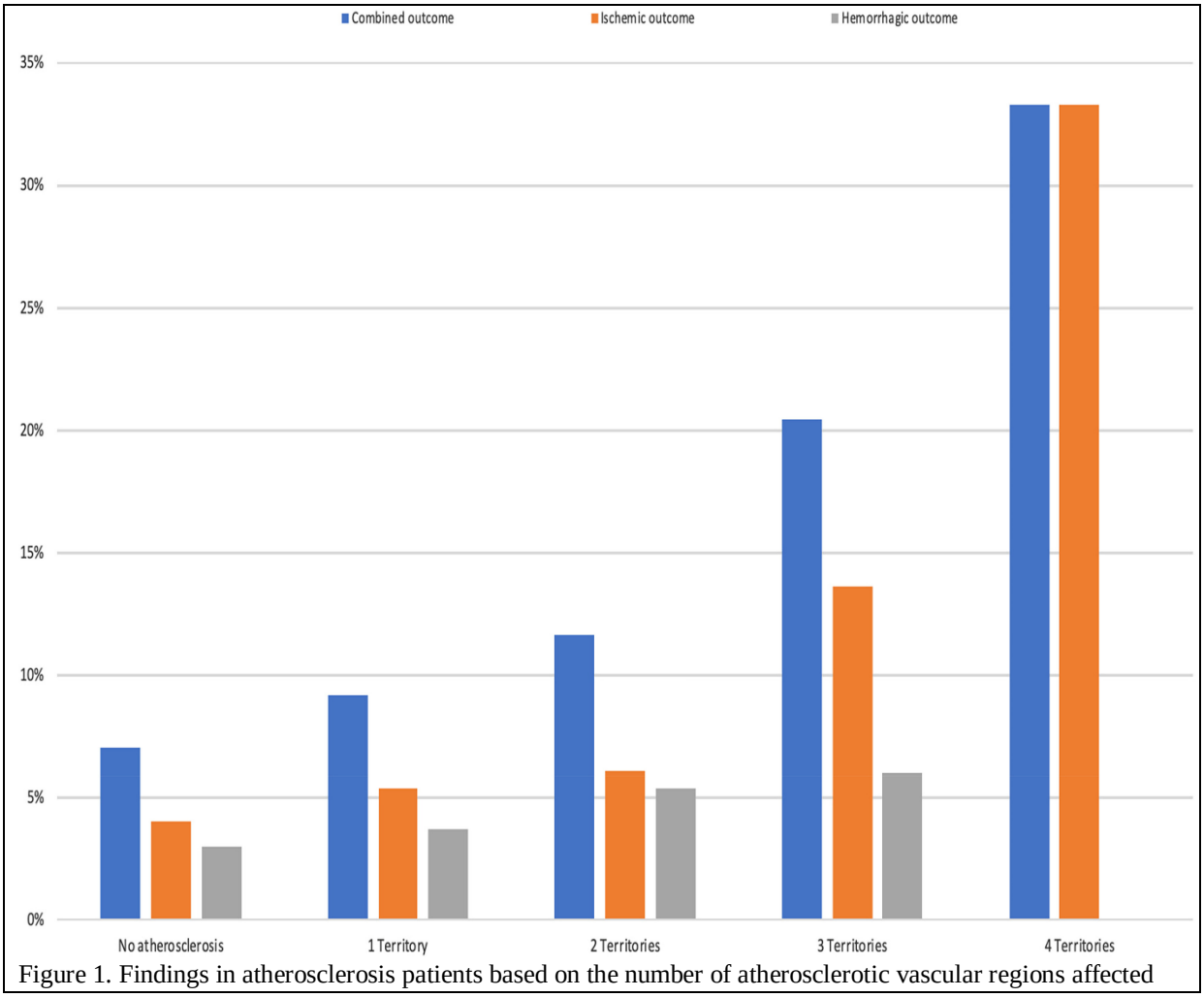

Figure 1. Findings in atherosclerosis patients based on the number of atherosclerotic vascular regions affected

DISCUSSION-The current study showed that there was a considerable elevated risk of combined vascular and ischemic events in patients with AVD covering three or four territory. On the other hand, there does not seem to be a relationship between the level of atherosclerotic involvement and bleeding consequences. This suggested that ischemic rather than hemorrhagic events were predominantly responsible for the higher risk in the combined patient group.[2] The current study results were in coincides with Park et al. that found a dose-dependent increase in the incidence of major adverse vascular events (MACE) and all-cause death in stroke patients with concurrent AVD. The study found that extracranial stenosis was highly prevalent.[1]

CONCLUSION-The current study concluded that in patients who have had a recent stroke and AF, AVD was a prognostic indication, especially if three or four territory were involved. An increased risk of combined and ischemic events was strongly correlated with the degree of vascular territory involvement.

An increased risk of combined and ischemic events was strongly correlated with the degree of vascular territory involvement.

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A RARE CASE ON CARDIAC TAMPONADE IN DOWN'S SYNDROME ASSOCIATED WITH HYPOTHYROIDISM

INTRODUCTION

Down syndrome is among the most common chromosomal defect. Many congenital abnormalities, such as heart problems, duodenal atresia, anorectal malformations, hypospadias, and cataracts, were linked to down syndrome.[1] Furthermore, down syndrome was frequently associated with hypothyroidism, which can cause pericardial effusion (PE). However, cardiac tamponade is a rare consequence and is an emergency condition requiring prompt diagnosis and treatment. The aim of this case report was to present a case on cardiac tamponade in down's syndrome associated with hypothyroidism.[2]

CASE PRESENTATION

A 52-year-old male with down's syndrome and levothyroxine-treated hypothyroidism arrived to the emergency department with shortness of breath and chest pain. He had previously been tested for similar reasons and was diagnosed with asymptomatic PE. The patient's physical examination revealed normal heart rate and blood pressure, reduced heart sounds on auscultation, and jugular venous distention. He did not have any murmurs or frictional rubs. The remainder of the physical test was ordinary. The initial EKG revealed normal sinus rhythm and a normal heart rate of 72 beats/min. The blood pressure was 125/86 mm Hg upon arrival. The patient was given supplementary oxygen due to shortness of breath, but there was no improvement. CXR revealed an expanded cardiac profile (Figure 1). Arterial blood gas results indicated a pH of 7.47, pCO2 of 40, pO2 of 80, bicarbonate of 29, and oxygen saturation of 97%. The TSH level was 10. The patient did not take his thyroid pills as prescribed. When the patient's symptoms worsened, a bedside ultrasound detected cardiac tamponade. An official

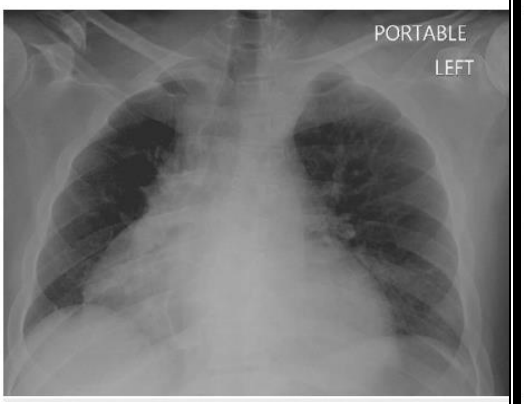

Figure 1. Initial chest X-ray showing the enlarged heart

echocardiography confirmed the findings. Cardiologists consultation was done, and they advised moving the patient for emergency care. The patient was taken to a different hospital and had pericardiocentesis. A pericardial window was implanted to prevent recurring symptoms. A repeat CT chest scan revealed reduced pleural effusion and no evidence of cardiac tamponade. His shortness of breath improved, and he was discharged with frequent check-ups.

DISCUSSION

This case report showed consider cardiac tamponade while diagnosing down's syndrome patients who have symptoms of cardiovascular impairment.[2] Similar to this, Perttunen H et al., showed a pericardial effusion linked to hypothyroidism in an adult girl with down's syndrome.[3]

CONCLUSION

This case study showed that a medical emergency frequently arises from cardiac tamponade. Patients with down syndrome may have hypothyroidism, albeit this was uncommon. Patients may have unfavorable results from therapy delays. Also, it was demonstrated how early detection of PE with ultrasonography enabled the timely start of suitable care through this instance. In order to efficiently assess patients with Down syndrome and dyspnea, the case report expect that medical professionals will also be aware of this.

Identifying possible differential diagnoses in Down's syndrome individuals can reduce therapy delays and missed diagnoses. This example highlights the need of using ultrasonography to detect PE early and initiate proper therapy.

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