Comprehensive Nursing Management of a School-Aged Child with Subpulmonary Ventricular Septal Defect and Moderate–Severe Aortic Regurgitation Complicated by Congestive Heart Failure: A Case Study

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Abstract

Congenital heart disease remains a major cause of morbidity in children worldwide. Ventricular septal defect (VSD) is among the most common congenital cardiac anomalies and may be complicated by aortic valve prolapse and aortic regurgitation (AR), leading to progressive heart failure. This case study describes the comprehensive nursing management of a 9-year-old boy with subpulmonary VSD complicated by right coronary cusp prolapse and moderate-to-severe AR, status post VSD closure and aortic valve repair, who was readmitted with congestive heart failure (CHF) and jaundice. Clinical findings, laboratory investigations, imaging results, pharmacological management, and nursing interventions are analyzed. Emphasis is placed on pathophysiology, hemodynamic monitoring, medication management, prevention of complications, and family-centered care. The case highlights the importance of early detection of residual valvular dysfunction, vigilant cardiac monitoring, and multidisciplinary management in pediatric cardiac patients.

Keywords: Ventricular septal defect; aortic regurgitation; congestive heart failure; pediatric nursing; congenital heart disease

Introduction

Congenital heart disease affects approximately 8–10 per 1,000 live births globally and represents a significant cause of pediatric hospitalization (Kliegman et al., 2023). Ventricular septal defect (VSD) accounts for approximately 20–30% of all CHDs (Park, 2021).

Subpulmonary (supracristal) VSD is particularly associated with prolapse of the right coronary cusp of the aortic valve due to the Venturi effect, which may lead to progressive aortic regurgitation (Anderson et al., 2020). Chronic AR results in left ventricular volume overload, compensatory hypertrophy, and eventual heart failure if untreated (Kliegman et al., 2023).

Despite surgical closure, some patients continue to experience residual valvular dysfunction and progressive cardiac remodeling, requiring long-term surveillance and multidisciplinary management (Park, 2021).

Comprehensive nursing management plays a crucial role in preventing complications and promoting hemodynamic stability in pediatric patients with complex CHD. Nursing care must include continuous assessment of cardiac function, monitoring for signs of heart failure and congestive hepatopathy, fluid balance management, medication adherence support, and family education. An individualized and evidence-based nursing care plan is essential to minimize risk, enhance early detection of complications, and ensure patient safety throughout hospitalization and follow-up care (Thidaratana, 2023).

This case study aims to present a detailed clinical and nursing analysis of a school-aged child with complex post-operative CHD who developed CHF and congestive hepatopathy.

2. Case Presentation

2.1 Patient Information

A 9-year-old Thai male was admitted for the third hospitalization with the chief complaint of bilateral leg edema and abdominal distension for one week prior to admission.

Medical History:

- Diagnosed with VSD at birth
- Referred to a tertiary cardiac center
- Underwent emergency subpulmonary VSD closure with aortic valve repair on July 19, 2025
- Regular follow-up post-surgery

Current Diagnosis on Admission:

1. Congestive heart failure
2. Jaundice
3. Status post subpulmonary VSD closure with moderate-to-severe aortic regurgitation

2.2 Socioeconomic and Environmental Context

The patient lives in a rural agricultural area with exposure to farm environments and domestic poultry. Family income is limited (approximately 11,000 THB/month combined parental income). Although healthcare access is geographically convenient, financial constraints may influence long-term follow-up adherence.

Family history revealed maternal hypertension during pregnancy. No confirmed genetic transmission of VSD was identified.

3. Clinical Findings

3.1 Physical Examination

- Weight: 31 kg
- Height: 136 cm
- BMI: 16.76 kg/m² (normal for age)
- Temperature: 37.1°C
- PR: 114 bpm (tachycardia)
- RR: 28/min
- BP: 110/58 mmHg
- O_2 Saturation: 98%

3.2 Cardiac Findings:

- Diastolic murmur grade 2/6 at aortic area
- PMI at 5th intercostal space, left midclavicular line
- No pulmonary hypertension

3.3 Abdominal Findings:

- Hepatomegaly (liver palpable 1 cm below costal margin)
- Moderate ascites

3.4 Skin:

- Mild pallor
- Scleral icterus

4. Diagnostic Assessment

4.1 Echocardiography

- Status post VSD closure (no residual shunt)
- Right coronary cuspid prolapses
- Moderate-to-severe aortic regurgitation
- Good biventricular systolic function
- No pulmonary hypertension

4.2 Electrocardiogram

- Sinus tachycardia
- Left ventricular hypertrophy
- Left atrial enlargement
- Nonspecific ST-T abnormalities

4.3 Laboratory Findings

Cardiac Biomarker:

- NT-proBNP: 5806 pg/mL (markedly elevated, consistent with CHF)

Liver Function:

- Total bilirubin: 4.97 mg/dL
- Direct bilirubin: 4.46 mg/dL
- AST mildly elevated ⟶ Consistent with congestive hepatopathy

Hematology:

- Microcytic indices (low MCV, low MCH)
- Anisocytosis ⟶ Suggestive of possible iron deficiency anemia

Electrolytes:

- Mild hypokalemia (3.4 mMol/L)
- Mild hypocalcemia

Imaging (Ultrasound):

- Hepatomegaly with congestive hepatopathy
- Moderate ascites
- Bilateral pleural effusion

5. Pathophysiological Discussion

Subpulmonary VSD predisposes to prolapse of the right coronary cusp due to Venturi effect, leading to progressive aortic regurgitation. Chronic AR results in left ventricular volume overload, causing LVH and LAE.

Despite surgical closure of the VSD, residual moderate-to-severe AR persisted, maintaining chronic volume overload. Elevated NT-proBNP confirms ventricular wall stress.

Right-sided congestion resulted from advanced heart failure, causing:

- Hepatic venous congestion ⟶ hepatomegaly ⟶ direct hyperbilirubinemia
- Ascites
- Pleural effusion

This case demonstrates the continuum from structural congenital defect to chronic heart failure and systemic complications.

The structural abnormality is illustrated in Figure 1, which was generated using artificial intelligence. As shown in Figure 1, subpulmonary VSD leads to progressive aortic regurgitation and congestive heart failure.

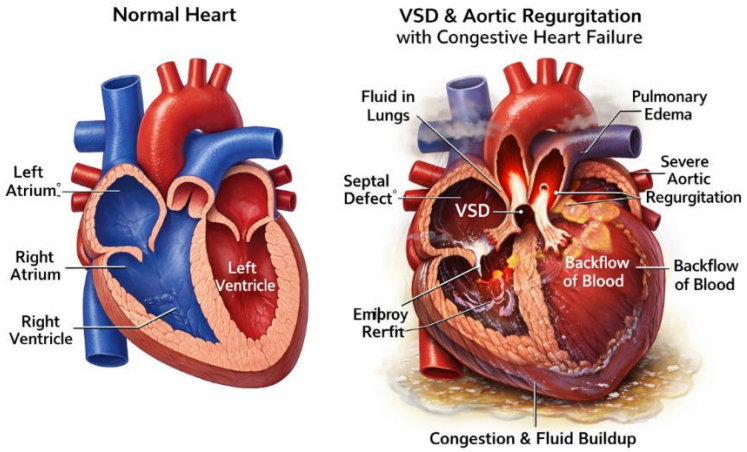

Figure 1 *Comparison between a normal heart and subpulmonary ventricular septal defect with moderate–severe aortic regurgitation complicated by congestive heart failure.*

Note. Image generated using ChatGPT (OpenAI), 2026, for academic illustration purposes.

6. Medical Management

6.1 Inotropic Support

Dobutamine IV infusion

- Positive β1-adrenergic agonist
- Increases myocardial contractility
- Improves cardiac output

6.2 Diuretics

Furosemide (oral)

- Loop diuretic
- Reduces preload
- Controls edema and ascites

6.3 ACE Inhibitor

Enalapril

- Reduces afterload
- Prevents cardiac remodeling
- Improves long-term heart failure outcomes

6.4 Cardiac Glycoside

Digoxin (Lanoxin)

- Enhances myocardial contractility
- Controls heart rate

6.5 Antibiotic Prophylaxis

Benzathine Penicillin

- Prevents infective endocarditis

7. Nursing Diagnoses and Nursing Care Plan

Nursing care for pediatric congestive heart failure requires systematic clinical reasoning guided by standardized nursing diagnoses. The following diagnoses are based on NANDA-I (2021–2023) classification and supported by pediatric cardiac nursing literature (Hockenberry & Wilson, 2021; Herdman & Kamitsuru, 2021 ;Thidaratana, 2023).

7.1 Nursing Diagnosis 1: Decreased Cardiac Output

Related to:

- Altered myocardial contractility
- Volume overload secondary to moderate–severe aortic regurgitation

As evidenced by:

- Tachycardia (PR 114 bpm)
- Elevated NT-proBNP (5806 pg/mL)
- Hepatomegaly and ascites
- Fatigue and peripheral edema

Expected Outcomes

Short-term outcomes:

- Heart rate within age-appropriate range
- Stable blood pressure
- Improved urine output (>1 mL/kg/hr)

Long-term outcomes:

- Reduced NT-proBNP levels
- Absence of worsening heart failure symptoms

Nursing Interventions and Rationales

1. Monitor vital signs every 2–4 hours.

   Rationale: Tachycardia is an early compensatory response to decreased cardiac output (Hockenberry & Wilson, 2021).

2. Assess for signs of worsening heart failure (increased edema, dyspnea, hepatomegaly).

   Rationale: Early detection prevents decompensation (Kliegman et al., 2023).

3. Administer prescribed inotropic agents (e.g., dobutamine) as ordered.

   Rationale: Dobutamine enhances myocardial contractility and increases cardiac output (Brunner & Suddarth, 2022).

4. Promote rest and cluster nursing activities.

   Rationale: Reduces myocardial oxygen demand and cardiac workload (Park, 2021).

5. Monitor ECG for arrhythmias.

   Rationale: Structural heart disease increases risk of rhythm disturbances.

7.2 Nursing Diagnosis 2 : Excess Fluid Volume

Related to:

- Compromised regulatory mechanisms
- Increased venous pressure secondary to heart failure

As evidenced by:

- Bilateral lower extremity edema
- Ascites
- Pleural effusion
- Weight gain

Expected Outcomes

- Reduction in edema
- Stable body weight
- Balanced intake and output

Nursing Interventions and Rationales

1. Strict intake and output monitoring.

   Rationale: Accurate fluid balance assessment guides diuretic therapy (Brunner & Suddarth, 2022).

2. Daily weight measurement at the same time each day.

   Rationale: Rapid weight gain indicates fluid retention (Hockenberry & Wilson, 2021).

3. Administer loop diuretics (furosemide) as prescribed.

   Rationale: Reduces preload and relieves congestion (Park, 2021).

4. Monitor serum electrolytes, especially potassium.

   Rationale: Loop diuretics increase potassium loss, risking arrhythmias (Kliegman et al., 2023).

5. Elevate lower extremities when appropriate.

   Rationale: Promotes venous return and reduces peripheral edema.

7.3 Nursing Diagnosis 3: Impaired Gas Exchange (Risk for)

Related to:

- Pulmonary congestion
- Pleural effusion

Expected Outcomes

- Oxygen saturation ≥ 95%
- Absence of respiratory distress

Nursing Interventions

1. Monitor respiratory rate and oxygen saturation continuously.
2. Position patient in semi-Fowler's position.
3. Administer oxygen therapy if prescribed.

Rationale: Pulmonary congestion secondary to heart failure impairs oxygen diffusion (Kliegman et al., 2023).

7.4 Nursing Diagnosis 4: Imbalanced Nutrition: Less Than Body Requirements

Related to:

- Increased metabolic demand
- Early satiety due to ascites

Expected Outcomes

- Adequate caloric intake
- Stable growth percentile

Nursing Interventions

1. Provide small, frequent meals.
2. Collaborate with dietitian for high-calorie, low-sodium diet.
3. Monitor growth parameters.

Rationale: Children with CHF have increased metabolic demands and fatigue during feeding (Hockenberry & Wilson, 2021).

7.5 Nursing Diagnosis 5: Risk for Electrolyte Imbalance

Related to:

- Diuretic therapy
- ACE inhibitor therapy

Nursing Interventions

1. Monitor serum potassium and sodium regularly.
2. Assess for signs of hypokalemia (muscle weakness, arrhythmias).
3. Educate caregivers regarding medication adherence.

Rationale: Electrolyte imbalance increases risk of life-threatening dysrhythmias (Park, 2021).

7.6 Nursing Diagnosis 6: Anxiety (Child and Family)

Related to:

- Hospitalization
- Chronic cardiac condition

Expected Outcomes

- Verbalization of reduced anxiety
- Active participation in care

Nursing Interventions

1. Provide age-appropriate explanations.
2. Encourage parental presence.
3. Offer psychological support.

Rationale: Family-centered care improves coping and adherence in pediatric chronic illness (Hockenberry & Wilson, 2021).

Summary of Nursing Care Plan

The nursing management of this case focused on:

- Hemodynamic stabilization
- Fluid volume regulation
- Prevention of complications
- Medication safety
- Growth and nutrition support
- Family-centered education

Comprehensive nursing assessment and early intervention were essential in preventing further cardiac decompensation and promoting recovery.

8. Outcomes

After medical stabilization:

- Edema improved
- Vital signs stabilized
- Renal function preserved
- Congestive symptoms reduced

However, moderate-to-severe AR persists, requiring long-term cardiology follow-up and potential future valve intervention.

9. Discussion

This case emphasizes that:

1. Surgical closure of VSD does not eliminate risk of progressive AR.
2. Early detection of valvular complications is critical.
3. CHF in pediatric patients may present with hepatomegaly and jaundice.
4. NT-proBNP is a valuable biomarker in pediatric heart failure monitoring.

5. Multidisciplinary and nursing-driven monitoring significantly improves outcomes.

Long-term prognosis depends on progression of aortic regurgitation and ventricular remodeling.

10. Conclusion

This case illustrates the complex trajectory of congenital heart disease from structural defect to chronic heart failure despite surgical correction. Comprehensive pediatric cardiac nursing care—focusing on hemodynamic stability, medication management, complication prevention, and family education—is fundamental in improving clinical outcomes and quality of life.

Ongoing surveillance of aortic valve function remains essential in patients with subpulmonary VSD and RCC prolapse.

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12. Ethical Considerations

This case study was conducted in accordance with ethical principles for research involving human participants. Written informed consent was obtained from the patient's parent/legal guardian for the use of anonymized clinical data for academic publication purposes. All identifying information has been removed to ensure confidentiality and privacy protection.

The study adhered to the principles outlined in the Declaration of Helsinki and institutional ethical guidelines for case reporting. As this manuscript presents a single case without experimental intervention, formal institutional review board approval was not required according to institutional policy.

13. Author Contributions

All authors contributed substantially to the intellectual content of this manuscript.

- **Conceptualization and Case Analysis:** Primary author
- **Clinical Data Collection:** Clinical nursing team
- **Literature Review and Theoretical Integration:** Primary author
- **Manuscript Drafting:** Primary author
- **Critical Revision and Final Approval:** All authors

All authors reviewed and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

14. Conflict of Interest Statement

The authors declare that there are no conflicts of interest regarding the publication of this paper.

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