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CASE REPORT

Hypoparathyroidism with situs inversus totalis: A case report

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Abstract

BACKGROUND

Hypoparathyroidism (HP) is a rare endocrine disorder, while situs inversus totalis (SIT) is a rare condition in which the internal organs are positioned in a mirrored pattern compared to their usual positions. This case illustrates some potential shared mechanisms between HP and SIT, highlighting the importance of accurate identification and prompt first emergency, offering insights for future research.

CASE SUMMARY

This report discusses a case of a middle-aged patient with adolescent-onset HP with concurrent SIT. The patient experienced recurrent episodes of increased neuromuscular excitability (manifesting as spasms in the hands and feet and laryngospasms) and even periods of unconsciousness. Initially, these symptoms led to a misdiagnosis of epilepsy. Nevertheless, upon thorough examination and treatment in the general medicine ward, the correct diagnosis was established. Corresponding treatment resulted in improved management of the patient's symptoms.

CONCLUSION

Co-occurrence of HP and SIT may be associated with genetic mutations, chromosomal anomalies, or hereditary factors, as may other similar conditions.

Key Words: Hypoparathyroidism; Situs inversus totalis; Consciousness disorder; Epilepsy; Genetic inheritance; Case report

Core Tip: We report a case of patient misdiagnosed with "epilepsy", who responded well to calcium gluconate. Upon admission, the patient exhibited altered consciousness, which was initially attributed to epilepsy. However, subsequent tests identified situs inversus totalis (SIT). Despite achieving stabilization, recurring hand muscle spasms persisted. The diagnosis was revised to hypoparathyroidism with SIT and was managed *via* supplementation with calcium and vitamin D. Effective symptom control was noted at the nine-month follow-up.

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INTRODUCTION

Hypoparathyroidism (HP) is a clinical syndrome characterized by neuropsychiatric symptoms, hypocalcemia, and hyperphosphatemia. HP, which is caused by insufficient secretion or reduced activity of the parathyroid hormone, can lead to complications such as secondary epilepsy, cognitive impairment, intracranial calcifications, and cataracts[1-3]. HP is a rare disorder[4], and limited data are available on its prevalence in most countries and regions[5]. The etiology of HP can be classified into five categories: Neck surgery, autoimmune disorders, genetic factors, idiopathic reasons, and other rare causes[6]. Most cases involving genetic, autoimmune, or magnesium-metabolism abnormalities have well-defined genetic defects and chromosomal locations. Situs inversus (SI) is a condition where the internal organs are positioned in a "mirrored" layout of their normal positions. SI can be categorized as totalis (SIT) or SI partialis[7,8]. Most individuals with SI have normal physiological functions despite organ displacement, exerting minimal impact on daily life. In the absence of symptoms, no treatment is warranted. Consequently, the condition is often detected incidentally during examinations for other reasons[9-11]. Nevertheless, a small subset of patients may have additional organ anomalies or genetic diseases, leading to various symptoms.

It is speculated that both conditions may be linked to chromosomal abnormalities, genetic mutations, or hereditary factors. Prenatal screening and genetic counseling can potentially facilitate early identification of these risks, ensuring timely interventions such as the option to terminate the pregnancy.

Herein, we present a case of a 55-year-old man who experienced adolescent-onset HP with concurrent SIT.

CASE PRESENTATION

Chief complaints

A 55-year-old man presented to our hospital with a complaint of persistent cough and sputum production with intermittent shortness of breath since four years. The patient experienced a recent exacerbation in respiratory distress, which lasted over two hours.

History of present illness

Over the past four years, the patient experienced recurrent cough and sputum production after catching a cold, with a small amount of white, foamy sputum, primarily in the mornings. These episodes were more frequent in the winter and spring, lasting for approximately 2-3 months each year. The patient also experienced shortness of breath while climbing small inclines, which was alleviated with antibiotic treatment. However, due to ongoing breathlessness, he began walking slower on flat surfaces compared to his peers. Irregular oral antibiotic treatment provided temporary relief. Two hours prior to seeking medical attention, the patient's shortness of breath became significantly aggravated, accompanied by a state of reduced consciousness, but without limb convulsions or loss of bladder or bowel control. He was subsequently transferred to the emergency department of our hospital for further treatment, where he was admitted to the Coronary Care Unit for further evaluation due to the unclear cause of his consciousness disturbance. The patient's general mental state, appetite, and sleep patterns were average. Additionally, his bladder and bowel functions were normal, with no recent significant weight changes.

History of past illness

The patient recalled being hospitalized more than 30 years ago with a diagnosis of epilepsy. He had not taken medication regularly, but occasionally experienced localized muscle spasms in his lower limbs. When severe, these spasms could cause a feeling of tightness in the throat and difficulty in breathing. These symptoms were effectively relieved by intravenous calcium gluconate.

Personal and family history

The patient had a smoking history of over 40 years at approximately two packs *per* day, with no attempts to quit. There was no notable family medical history.

Physical examination

The patient's body temperature was 36.4 °C, blood pressure was 115/80 mmHg, pulse was 87 beats per minute, respiratory rate was 23 breaths per minute, and pulse oxygen saturation was 88% (without supplemental oxygen). He was comatose, with pupils that appeared round, equal in size (approximately 0.4 cm in diameter), and responsive to light. There was no jugular vein distention, and the hepatojugular reflux sign was negative. His chest was barrel-shaped with reduced tactile fremitus on both sides. Percussion revealed hyperresonance, and breath sounds were coarse with distinct moist rales, although without noticeable wheezing. The apex beat was located at the fifth intercostal space along the right midclavicular line, with a heart rate of 87 beats *per* minute and regular rhythm. Heart sounds were strong, with no murmurs detected across the valve auscultation areas. Neurological examination results were negative.

Laboratory examinations

Upon admission, laboratory examinations revealed a slight abnormality in white blood cells. The levels of creatine kinase (CK) and CK-MB were elevated, while the troponin level was within the normal range. The blood lactate concentration was 7.32 mmol/L, suggesting a substantial elevation. It was noted that the serum calcium ion level was markedly decreased, and the serum magnesium level was mildly diminished. The levels of serum phosphorus ion, sodium ion, potassium ion, chloride ion, liver function, coagulation function and creatinine were all normal. Subsequently, parathyroid hormone and 25-hydroxyvitamin D were measured and found to be significantly decreased (Table 1 and Table 2).

Imaging examinations

Cranial (Figure 1) and chest computed tomography (Figure 2, Figure 3A, and Figure 3B) demonstrated: (1) Multiple calcifications in the cerebral hemispheres, cerebellum, and brainstem; (2) Complete situs inversus with right-sided cardiac pulsation, cardiac enlargement mainly characterized by left ventricular hypertrophy and a small amount of pericardial effusion; and (3) Patchy high-density regions in both lungs, indicating inflammation. Parathyroid gland ultrasound reveals no visible parathyroid glands on either side. Doppler echocardiography shows right - heart and left-atrial enlargement, reduced left-ventricular systolic function, and an ejection fraction of 40% (Table 3).

FINAL DIAGNOSIS

The final diagnosis was HP with SIT, chronic obstructive pulmonary disease with acute lower respiratory tract infection, and hyperlactatemia.

TREATMENT

The patient, after being placed on a non-invasive ventilator, slowly regained consciousness and was transferred to the Department of General Medicine after his condition stabilized. During hospitalization, he experienced repeated muscle spasms on the radial side of his right hand and occasional wheezing. Treatment included intravenous calcium gluconate (10–20 mL *per* dose), oral calcitriol (0.5 μg once daily), and calcium carbonate with vitamin D3 granules (3 g twice daily). He also received magnesium supplementation, anti-infection therapy, cough suppressants, sputum reducers, anti-inflammatory drugs, and bronchodilators. After this treatment, the patient did not experience muscle spasms or throat tightness.

OUTCOME AND FOLLOW-UP

After discharge, the patient stopped taking his medication. Two weeks later, he experienced a recurrence of muscle spasms in his hand, prompting his family to return to the hospital to refill his oral prescriptions. With regular medication, the patient did not experience any further airway spasms or convulsions during the nine-month follow-up.

DISCUSSION

This patient experienced recurrent episodes of increased neuromuscular excitability (spasms in hands, feet, and laryngospasms) during his teenage years. Laboratory and radiological examinations revealed decreased parathyroid hormone levels, hypocalcemia, hypomagnesemia, no detectable parathyroid gland on ultrasound, and multiple intracranial calcifications on cranial computed tomography (CT). Electroencephalography did not find epileptic waves. Magnesium sulfate treatment alone was ineffective, while calcium gluconate and vitamin D resolved the symptoms, suggesting a diagnosis of HP[1]. The patient had no history of surgery, radiotherapy, or chronic medication use, ruling out a surgical etiology. No invasive parathyroid diseases were observed. Since the symptoms commenced after childhood and there were no signs of other multi-system issues such as thyroid disorders, diabetes, anemia, or candidiasis, an autoimmune cause seemed improbable despite lack of autoimmune antibody screening and no relevant family history

Table 1 Laboratory examinations

Item	WBC (× 10^9 /L)	α-HBD (U/L)	LDH (U/L)	CK-MB (U/L)	CK (U/L)	CREA (μmol/L)	LAC (mmol/L)	Ca (mmol/L)	Mg (mmol/L)	IP (mmol/L)
Result	12.4	290.4	449.4	45	774.8	60	7.32	1.28	0.72	1.31
Reference range	4–10	90–182	109–245	0–24	18–198	44–131	0.5–1.5	2.25–2.58	0.8–1.2	0.97–1.61

WBC: White blood cells; α-HBD: α-hydroxybutyrate dehydrogenase; LDH: Lactate dehydrogenase; CK-MB: Creatine kinase-MB; CREA: Creatinine; LAC: Lactate; Ca: Serum calcium; Mg: Serum magnesium; IP: Inorganic phosphate.

Table 2 Laboratory examination

Item	Na (mmol/L)	K (mmol/L)	Cl (mmol/L)	cTNT (μg/L)	PTH (pg/mL)	25(OH)D (nmol/L)	AST (U/L)	ALT (U/L)	TBIL (μmol/L)
Result	Normal	Normal	Normal	Normal	4.5	25	Normal	Normal	Normal
Reference range	135-145	3.5-5.5	96-108	< 0.1	12–88	75–250	0-40	5-40	3.4-17.1

Na: Serum sodium; K: Serum kalium; Cl: Serum chloride; cTNT: Troponin T; PTH: Parathyroid hormone; 25(OH)D: 25-hydroxyvitamin D; AST: Aspartate aminotransferase; ALT: Alanine transaminase; TBIL: Total bilirubin.

Table 3 Imaging examinations

Item	Result
Cranial computed tomography (Figure 1)	Multiple calcifications are found in bilateral cerebral hemispheres, cerebellar hemispheres and brainstem
Chest computed tomography (Figure 2, Figure 3, and Figure 4)	Total situs inversus, mirror - image dextrocardia, cardiac enlargement (mainly left ventricular enlargement) and a small amount of pericardial effusion are noted; Multiple patchy high - density shadows are seen in both lungs, indicating bilateral pulmonary inflammation; A small amount of pleural effusion is present on both sides
Doppler echocardiography	Dextrocardia, enlarged left atrium, decreased left ventricular systolic function, with an ejection fraction of 40%
Parathyroid ultrasound	No parathyroid glands visible on either side

[12]. Although the patient had concurrent hypomagnesemia, considering the long history and recurring symptoms along with additional abnormalities such as intracranial calcifications, SIT, and enzyme anomalies, the symptoms were unlikely to be solely caused by magnesium metabolism disorders, especially since the symptoms did not start in infancy. When the patient experienced respiratory distress and loss of consciousness, there were no signs of developmental malformations, growth retardation, or intellectual impairment. Despite no apparent family clustering, the combination of SI, enzyme abnormalities, and HP suggested a more complex syndrome. A decrease in parathyroid hormone levels is the opposite of Fahr syndrome[13]. However, genetic testing was not conducted in this study, leaving the presence of hereditary factors uncertain. In this case, the patient was misdiagnosed with epilepsy for many years[14,15], but was subsequently correctly diagnosed with HP combined with SIT (They are confirmed by chest computed tomography and electrocardiogram) (Figure 4), resulting in effective symptom management. This case highlights the importance of routine testing of calcium, phosphorus, magnesium, and parathyroid hormone levels, and if necessary, parathyroid ultrasound when encountering patients with epilepsy-like symptoms or brain CT findings of bilateral symmetric calcifications[16,17].

In this patient, SIT did not present with organ dysfunction, so the focus was on managing HP, with the main treatments being calcium and vitamin D supplementation. Parathyroid hormone or receptor agonists[18,19] were also considered, with the final option being parathyroid transplantation. Surgical intervention may be necessary for an exceedingly small number of SIT patients with organ dysfunction.

However, due to the patient's financial constraints, he did not complete some important diagnostic tests, such as autoimmune antibody screening, bone density scanning, slit lamp examination, and parathyroid scintigraphy. Additionally, the patient is at risk of long-term complications affecting the kidneys and cardiovascular system due to HP, arising from structural changes on Doppler echocardiography and abnormal levels of CK and creatinine. However, further evaluation of the coronary arteries or kidneys was not pursued. Moreover, the lack of genetic testing and family screening hindered the determination of genetic factors, and the patient's inconsistent follow-up and refusal to undergo further examinations pose barriers to comprehensive treatment and monitoring. Therefore, to confirm the diagnosis of primary isolated HP, further evaluation is needed to rule out other possible causes.

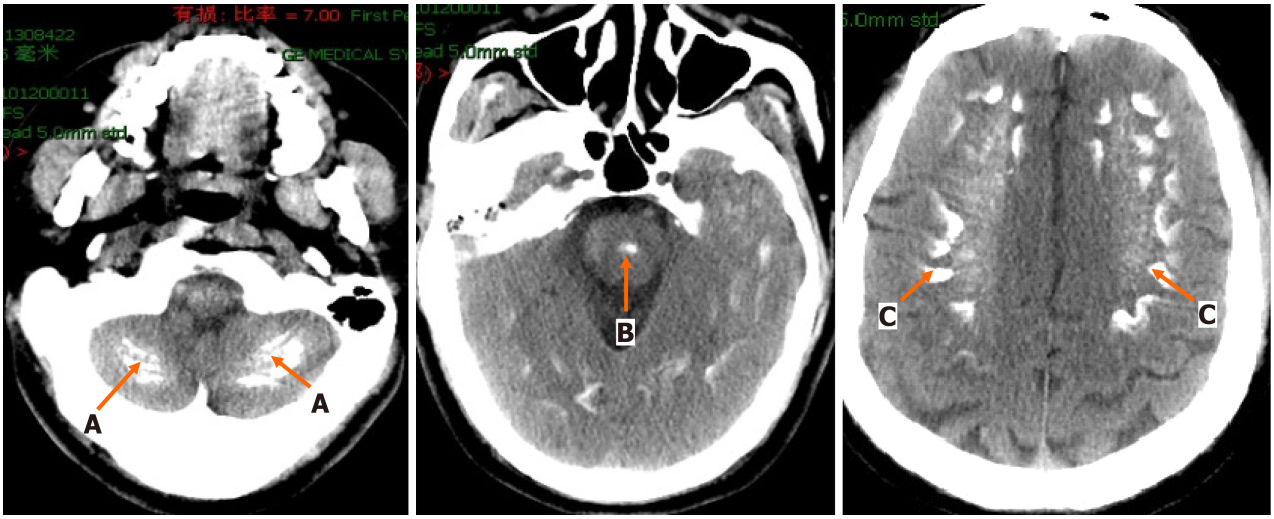

Figure 1 Cranial computed tomography scan. A: Bilateral cerebellar calcifications; B: Brainstem; C: Bilateral cerebral calcifications.

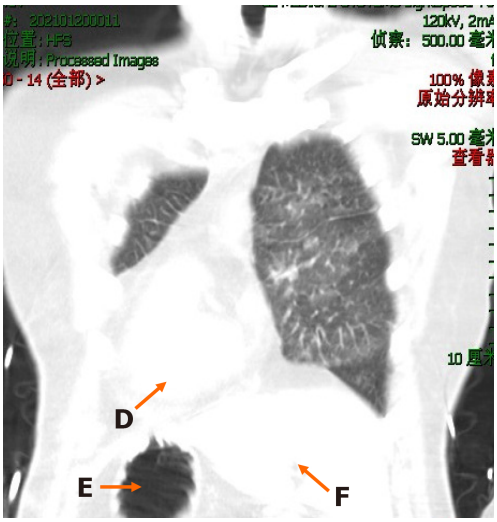

Figure 2 Chest computed tomography scan. (Coronal view: D: Heart; E: Stomach; F: Liver)

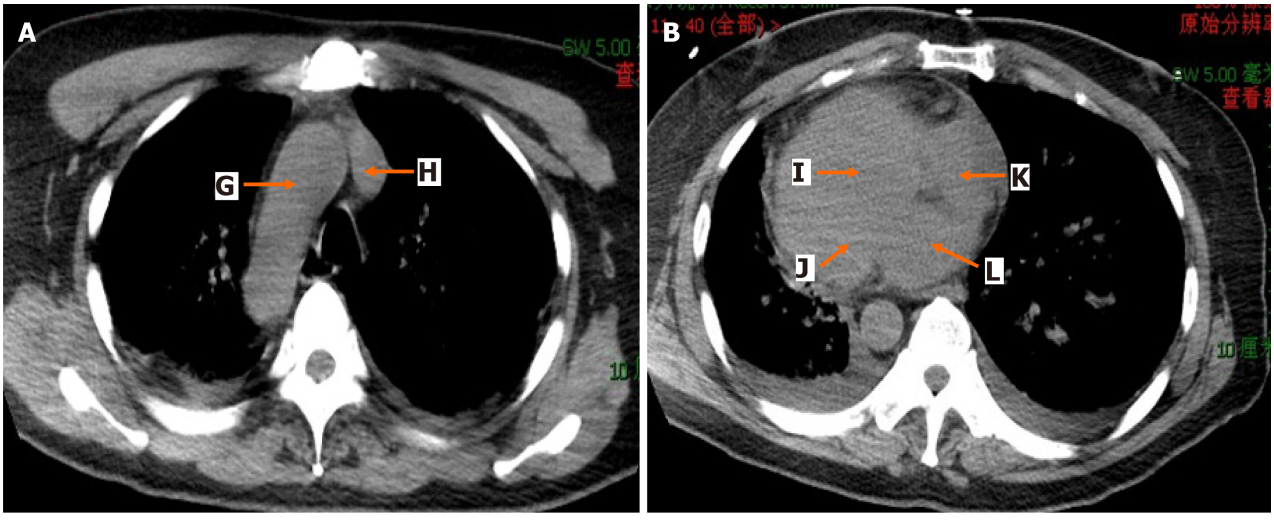

Figure 3 Chest computed tomography scan, A: Aortic arch level (G: Aortic arch; H: Superior vena cava); B: Four-chamber heart level (I: Right ventricle; J: Left ventricle; K: Right atrium; L: Left atrium)

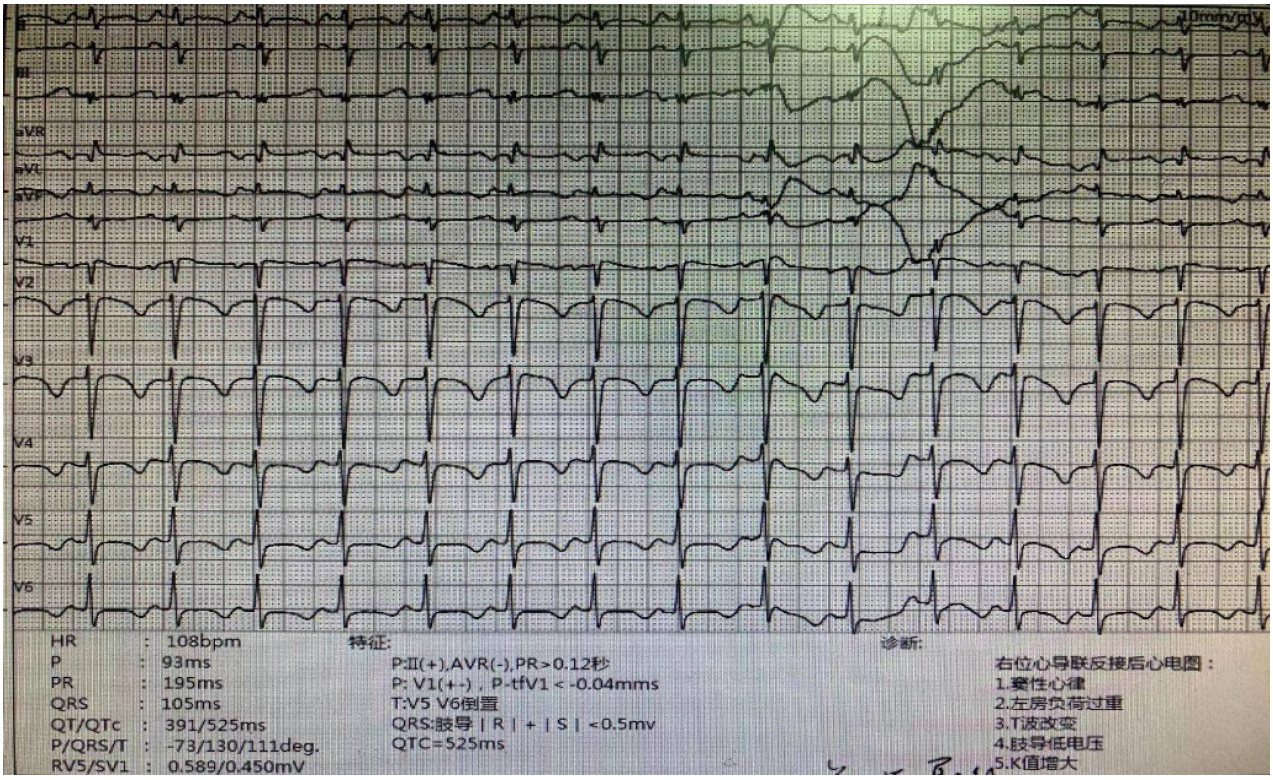

Figure 4 Electrocardiogram. (1) Sinus tachycardia; (2) Electrocardiogram changes indicating dextrocardia.

Currently, although there are significant differences between HP and SIT, there are no reports of their coexistence or common overlapping genetic genes. However, given that both involve gene mutations, it is reasonable to speculate that they may share common underlying genetic mechanisms. The two etiologies of HP are autoimmune disease and genetics, which can manifest as Fahr syndrome, Barakat syndrome[20,21], DiGeorge syndrome[22], *etc*., involving over 40 mutations in the *AIRE*, *TBX1*, *GATA3*, *TBCE*, *CHD7*, and other genes. HP associated with 22q11.2 microdeletion, namely DiGeorge syndrome, is characterized by multiple organ developmental abnormalities including the heart, which may be the breakthrough point connecting HP and SIT. SIT is associated with primary ciliary dyskinesia [23], which leads to atypical fluid flow and incorrect heart tube rotation[24]. Quan *et al*[25] have systemically reviewed the genetic basis of the pathogenesis of HP. Gene abnormalities in *AIRE*, *TBXI*, *GATA3*, *TBCE*, *CHD7, CASR*, and *GCMB* have been clearly identified in hereditary HP. It has been reported that 40 related genes have been identified[23,26]. Additionally, some of these mutations can be inherited through autosomal dominant, autosomal recessive, or X-linked recessive patterns[27, 28]. The coexistence of HP and SIT may be a new syndrome awaiting further research to elucidate the relationship and explore its impact on diagnosis, treatment, and patient management.

HP combined with SIT changes the anatomical landmarks for diagnosis, increases the diagnostic difficulty, and may mask clinical manifestations, thus affecting accuracy. Therefore, individualized treatment is needed, and medication should be adjusted as the situation demands. Moreover, SIT may affect drug absorption, make surgical treatment challenging and increase the associated risks, while the anatomical changes render the operation more difficult, and postoperative rehabilitation management needs to be more detailed. During emergencies, atypical symptoms increase the difficulty faced by clinicians in judging the condition, treatment decisions are complex and need to account for drug efficacy and safety; emergency medical equipment technology also needs to be adapted to cardiac inversion. During surgery, anatomical variations make positioning and operation difficult, since protecting surrounding important vessels is essential, and postoperative monitoring of complications and timely adjustment of treatment plans is required. Clinicians should fully understand the characteristics and mutual influence of these two diseases, improve the level of diagnosis and treatment, to provide patients with safe and effective medical care.

CONCLUSION

Further clinical and epidemiological research is needed to explore the relationship between HP and SIT, as well as to determine if they share common underlying causes. It is speculated that their co-occurrence may be associated with genetic mutations, chromosomal abnormalities, or hereditary factors. Prenatal genetic screening and genetic counseling might help reduce the risk of this syndrome from a genetic perspective.

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