



Case report

Single Umbilical Artery with Multicystic Dysplastic Kidney: A Case Report of VACTERL Association and its Ayurvedic aspects.

Article History:

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Abstract: Background –During pregnancy, the umbilical cord usually has two arteries and one vein. When only one artery is present, it is called a Single Umbilical Artery (SUA). SUA is important because it can be an early sign that the baby may have other birth defects. One such condition linked with SUA is VACTERL association. VACTERL is a group of birth defects that affect different body parts such as the Vertebral, Anal, Cardiac, Tracheoesophageal, Renal, and Limb. When three or more of these defects are present together, the condition is diagnosed as VACTERL association. According to Ayurveda, such congenital abnormalities are explained as Beeja-bhava dushti (defects in reproductive factors) and Garbhashaya dushti (unhealthy uterine environment), which can lead to Garbha vikriti (abnormal development of the fetus). Imbalance of Vata and Kapha dosha affects the proper formation of body tissues, resulting in defects of multiple organs. In Ayurveda, the umbilical cord (Nabhinadi) plays a vital role in nourishing the fetus. Any defect in the Nabhinadi can reduce nutrition to the fetus, leading to poor growth Garbhasōṣa. In cases of SUA, fetal nourishment may be insufficient, which can contribute to developmental problems. Ayurveda emphasizes preventive measures such as Garbha Samskara (pre-pregnancy care) and Garbhini Paricharya (proper care during pregnancy) to support healthy fetal development. This case highlights a rare presentation of VACTERL association with renal, cardiac, and vertebral components, and explores its possible correlation through Ayurvedic principles. **Objective:** To present a case of single umbilical artery with multicystic dysplastic kidney associated with congenital atrial septal defect and congenital scoliosis, representing a VACTERL association, and to explore its possible Ayurvedic correlation based on embryological and doshic principles. Case presentation- Newborn patient B/O 25 –year-old mother having Anomaly scan reported left multicystic dysplastic kidney. No extra-renal structural abnormalities seen on 20 weeks scan. There is presence of single umbilical artery giving rise to a two-vesseled cord at birth. After birth, further tests, including an X-ray, 2D echocardiography, and blood tests, revealed a complex congenital condition linked to VACTERL association. Intervention and outcome- The neonate was managed conservatively with monitoring of renal function, fluid-electrolyte balance, and cardiac status. Regular ultrasonography confirmed stable contralateral renal function, while the atrial septal defect was followed under pediatric cardiology supervision. No surgical intervention was required. From the Ayurvedic perspective, maternal counselling focused on Garbhini Paricharya and Vata-Kapha balancing to prevent recurrence of Garbhaja Vikruti in future.

Keywords: VACTERL Association, Congenital Anomalies & Ayurveda, Single Umbilical Artery, Garbhaja Vikruti, Garbhini Paricharya, Garbha Samskara.



INTRODUCTION

The presence of a Single Umbilical Artery (SUA) during prenatal evaluation often indicates underlying structural malformations and may serve as an early marker for VACTERL association. VACTERL association is a non-random cluster of congenital malformations involving Vertebral, Anal, Cardiac, Tracheoesophageal, Renal, and Limb anomalies. The presence of at least three component defects confirms the diagnosis. Its pathogenesis is linked to defective mesodermal development during early embryogenesis. In Ayurveda, such congenital malformations can be understood as manifestations of Beeja-bhava dushti (defects in reproductive elements) and Garbhashaya-dushti (defective uterine environment), leading to Garbha vikriti (malformation of fetus)^{1,2}. Vitiating of Vata and Kapha doshas disturbs the normal development of Asthi, Mamsa, and Meda dhatus, resulting in multiple organ defects as seen in VACTERL association^{3,4,5}. Each affected system in VACTERL finds a parallel explanation within Ayurvedic embryology and doshic theory. Nabhinadi or the umbilical cord is mainly responsible for nourishment of the fetus. Thus, defects in the nabhinadi lead to abnormalities in fetal growth. In this case of single umbilical artery, fetal nourishment is compromised. Nabhinadi nourishes the fetus, and abnormalities result in Garbhaśoṣa (fetal undernourishment). Preventive measures such as Garbha Samskara (preconception purification) and Garbhini Paricharya (antenatal care) are emphasized to ensure normal fetal development⁶.

Single umbilical artery is the most common macroscopic anomaly and most common malformation of the umbilical cord⁷. The prevalence of Single umbilical artery (SUA) is 0.5–6% worldwide⁸. SUA may be due to primary agenesis or secondary atrophy of one of the umbilical arteries⁹. It is defined as the absence of one umbilical artery in the umbilical cord. Often it is associated with congenital anomalies. When there is no chromosomal or structural anomaly, it is isolated single umbilical artery¹⁰. SUA increases risks of prematurity, Intra uterine death, Intra uterine growth restriction (IUGR)¹¹ and mortality among neonates¹². IUGR is defined as a rate of fetal growth that is less than normal for the expected growth potential of a specific infant¹³. IUGR may result from Maternal, Placental or Fetal origin. These infants may have acute neonatal problems like hypothermia, hypoglycemia and perinatal asphyxia. It is one of the leading causes of perinatal-neonatal morbidity and mortality and contributes to long-term chronic diseases. Individuals born after IUGR are

more susceptible to metabolic syndrome, cardiovascular and renal diseases, Type 2 Diabetes and chronic lung diseases at adulthood¹⁴. Asymmetrical IUGR occur later in third trimester and is mainly caused by utero placental insufficiency. VACTERL represents an acronym for a broad spectrum of congenital anomalies such as vertebral anomalies, anorectal anomalies (anal atresia), cardiac anomalies, tracheoesophageal fistula or atresia, renal anomalies, and limb anomalies¹⁵. Although there are no well-accepted strict diagnostic criteria, at least three features in each category are necessary for the clinical diagnosis, without other phenotypic or genetic characteristics of an alternative diagnosis¹⁶. The frequency is estimated to be about 1 in 10000 to 1 in 40000 live-born infants, depending on exact criteria used in different cohorts and studies. Some studies affirmed that the condition is more frequent in males. There is no evidence for increased prevalence in different regional areas or specific ethnic populations^{16,17}. There are different risk factors in the etiology of VACTERL association, such as genetic or maternal risk factors. Maternal risk factors that play a role are maternal diabetes, assisted reproductive techniques, or chronic lower obstructive pulmonary disorders¹⁸. Genetic risk factors include different genes mutations or chromosomal anomalies such as deletions of 5q11.2, 6q7q35qter, distal 13q and 20q13.33, duplication of 9q and 22q11.21, supernumerary der (22) syndrome, mosaicism for supernumerary ring chromosome 12 or 18 and partial monosomy 16p13.3pter/partial trisomy 16q22qter¹⁹. Vertebral anomalies include hemivertebrae, dysplastic vertebrae (such as “butterfly vertebrae”, “wedge vertebrae”), vertebral fusions, and supernumerary or absent vertebrae, congenital scoliosis, caudal regression, spina bifida²⁰. Anorectal anomalies consist of anal atresia (or imperforate anus), and in most cases, genitourinary anomalies are associated with most patients²⁰. Cardiac anomalies include a broad spectrum ranging from minor defects to severe structural defects incompatible with life. Some defects stated in the literature are ventricular septal defects, atrial defects, Fallot tetralogy, double outlet right ventricle, atrioventricular canal defect, aorto pulmonary window, and a vascular ring²¹. Tracheoesophageal fistula can associate atresia, and ultrasonographic signs include polyhydramnios or absent gastric bubble prenatally. It can also be associated with other pulmonary anomalies²². Renal anomalies include vesico-urinary reflux associated with a structural anomaly, unilateral renal agenesis, dysplastic or multicystic kidneys, duplicated collecting system, hydronephrosis horseshoes kidney, renal atrophy, or hypoplasia, ectopic kidney, and isolated ureteral stenosis²³. Limb malformations

include radial anomalies, thumb hypoplasia, lower limb defects polydactyly or oligodactyly 22.

Solomon BD states that there are different conditions with multiple common features with VACTERL association that must be taken into consideration in differential diagnosis such as Alagille syndrome, Baller-Gerold syndrome, CHARGE syndrome, Currarino syndrome, Feingold syndrome, Fryns syndrome, Holt-Oram syndrome, Townes-Brocks syndrome, Pallister-Hall syndrome and Opitz G syndrome¹⁵.

Multicystic Dysplastic Kidney (MCDK) is a congenital kidney disorder where the kidney is replaced by multiple cysts. Scoliosis is a sideways curvature of the spine that may occur alone or as part of a group of congenital anomalies. In rare cases, MCDK can be associated with other birth defects such as Atrial Septal Defect (ASD) and Single Umbilical Artery (SUA). This case highlights a newborn presenting with MCDK, congenital scoliosis, ASD, and SUA, suggesting a possible VACTERL association.

In the Ayurvedic concept fetal nourishment changes in a phasic manner. During Post fertilisation and post implantation period it occur mainly through upasneha and upasweda²⁴. Nabhinadi (umbilical cord) transfers the essence of ahararasa from mother to fetus, nourishing the fetus²⁵. such multiple congenital anomalies may be related to vitiation of Vata and Kapha doshas, leading to abnormal development of Asthi, Mamsa, and Meda dhatus. This correlation emphasizes the importance of integrating modern diagnostic insights with Ayurvedic understanding for a holistic view of congenital disorders.

Mātrj āhāra is another important factor that can

influence congenital fetal abnormalities due to the vitiation of the mother's doṣas. Shape manifestation, cell division, signaling, mobility, waste excretion, and cognition are all governed by Vāta doṣa, which also regulates the actions of Pitta and Kapha. According to Suśruta Saṃhitā, the mṛdu avayavas (soft organs) of the fetus are derived from mātṛja bhāva (maternal contribution) ²⁶. The features and organs inherited from the mother include skin, blood, muscle tissue, fat, umbilicus, heart, pancreas, gall bladder, spleen, kidney, urinary bladder, stomach, duodenum, small intestine, large intestine, omentum, rectum, and anal canal²⁶.

Disturbance in mātṛj bhāva due to improper āhāra-vihāra or vitiation of maternal doṣas during garbhāvasthā can result in multiple congenital defects. vitiated Vāta and Kapha doṣas disrupt the normal formation of mātṛja bhāva organs, leading to structural and functional malformations as observed in VACTERL association .

CASE REPORT

Antenatal details-

A primigravida woman in her mid-20s presented at our hospital with prenatal concerns. Following two fetal scans in the second trimester, radiological findings indicated left multicystic dysplastic kidney (MCDK). A subsequent scan by a different radiologist confirmed the presence of MCDK and a single umbilical artery. Notably, the patient had a history of consanguineous marriage. Her medical history was unremarkable, with no reported risk factors for genetic disorders, including diabetes, smoking, or exposure to organic chemicals. Family history revealed no instances of congenital or genetic disorders. Table 1 summarizes the patient's relevant investigations.

Table 1- Antenatal Investigations

SR NO	INVESTIGATION	RESULT
1.	1 st ANC anomaly scan	Primigravida. No medical risk factors. Anomaly scan reported left multicystic dysplastic kidney.
2.	2 nd ANC review anomaly scan on 30/05/2024	Left kidney is multicystic and dysplastic. There is presence of single umbilical artery giving rise to a two-veselled cord.

Birth History:

A 38-week pregnant woman underwent a cesarean section at our hospital due to Prolonged rupture of membranes (>18h) and oligohydramnios. The male infant, weighing 2102g at birth, was delivered via a lower segment cesarean section. Immediately after birth, the newborn cried and exhibited good vitality, with APGAR scores of 7 and 8 at 1 and 5 minutes, respectively.

A physical examination showed no palpable abdominal mass or visible spinal deformity. Given the mother's history of prolonged rupture of membrane, routine laboratory tests and imaging studies were conducted. Laboratory results indicated early onset sepsis with neonatal thrombocytopenia. A chest X-ray revealed scoliosis, while ultrasonography detected multiple variable-sized cortical cysts in the left kidney with echogenic intervening parenchyma, suggestive of

possibility multicystic dysplastic kidney (MCDK). Additionally, an 2D ECHO identified a small atrial septal defect with a left-to-right shunt

TABLE-2- Post-Natal Laboratory Investigations

INVESTIGATION	28/9/2024 DOL -2	30/09/2024 DOL-4	03/10/2024 DOL-7	6/10/2024 DOL-10	8/10/2024 DOL-12
Haemoglobin	20.4	-	18.6	-	17.1
Hematocrit	55.8	-	50.4	-	47.6
Platelite	128000	-	149000	-	327000
WBC	18700	-	8600	-	11400
CRP	11.4	-	62.7	11.7	7.5
Sr.creatinine	-	1.01	0.69		
Blood Urea		31.6			
Uric Acid		4.2			
T.Bilirubin		10.94			
D.Bilirubin		0.58			
I.Bilirubin		10.36			
Sr.sodium				142	
Sr.Potassium				4.4	
Ionic Calcium				0.99	

Table 3 – Post-Natal Other Investigation (including 2D ECHO,USG,Blood Culture And Sensitivity).

SR NO	INVESTIGATION	RESULT
1.	2DECHO	Small Arterial Septal Defect with Left to Right Shunt.
2.	PNC USG on 28/09/2024	Multiple variable sized left renal cortical cysts with echogenic intervening parenchyma-possibility of multicystic dyplastic left kidney(MCDK). Normal appearing right kidney. Few floating echoes in urinary bladder
3.	Blood Culture And Investigation.	No growth of any organisms.

DISCUSSION

VACTERL association represents a rare, non-random cluster of congenital anomalies involving vertebral, anal, cardiac, tracheoesophageal, renal, and limb malformations, with diagnosis made when three or more organ systems are affected²⁷. The etiopathogenesis arises from mesodermal dysgenesis during the third to fifth week of embryogenesis, a critical period for organogenesis when paraxial, intermediate, and lateral plate mesodermal layers differentiate²⁸. Disturbances in these layers lead to multisystem malformations: defective segmentation of the paraxial mesoderm produces vertebral anomalies such as hemivertebrae, butterfly vertebrae, and scoliosis; aberrations in the cloacal membrane and urorectal septum cause anal atresia; while lateral plate mesodermal malformation results in cardiac anomalies including atrial septal defect (ASD), ventricular septal defect (VSD), and tetralogy of Fallot, along with upper limb defects such as radial aplasia or thumb hypoplasia^{28,29}. Abnormal foregut division leads to tracheoesophageal fistula and esophageal atresia, whereas intermediate mesodermal dysgenesis results in

renal anomalies such as multicystic dysplastic kidney (MCDK), renal agenesis, or hydronephrosis^{29,30}.The predominant mechanisms implicated are mesodermal and vascular disruptions, often associated with the presence of a single umbilical artery (SUA), which causes regional hypoperfusion and asymmetrical organ development²⁹. Environmental influences—maternal diabetes, hypoxia, teratogenic drugs, or nutritional deficiencies (notably folate deficiency)—exacerbate embryonic vulnerability³¹. Genetic studies have identified mutations in *FOXF1*, *ZIC3*, *HOXD13*, and *PTEN* genes; however, the variability in presentation suggests a multifactorial embryologic field defect rather than a monogenic disorder³².

Our neonate had a two-vessel cord (single umbilical artery, SUA) and postnatal evaluation revealed left multicystic dysplastic kidney (MCDK), congenital scoliosis (vertebral anomaly), and a small atrial septal defect (ASD). The involvement of renal, vertebral and cardiac systems meets criteria for a VACTERL association (≥ 3 component anomalies). The infant was

clinically stable and managed conservatively with

multidisciplinary

follow-up



Fig 1. ANC Fetal Anamoly Scan



Fig 2. Spine X-Ray AP with Lateral view shows.



Fig 3. Spine X-Ray AP.

Fig 4. Post Natal USG Of Baby.



Fig 5 showing single umbilical artery.

The critical period for these anomalies is early organogenesis (approximately the 3rd–5th week), when paraxial, intermediate and lateral plate mesoderm differentiate to form vertebrae, kidneys, heart and limbs; disturbances in these processes yield multisystem defects³³. Single Umbilical Artery (SUA) results from absent development or atrophy of one umbilical artery. It is associated with an increased risk of other structural anomalies (renal, cardiac, growth restriction) and suggests possible vascular compromise contributing to asymmetric organ development. Prenatal detection of SUA warrants detailed anomaly scanning and postnatal surveillance³⁴. Multicystic dysplastic kidney (MCDK) arises from abnormal interaction between the ureteric bud and the metanephric blastema (abnormal branching/induction), producing nonfunctional cystic renal tissue. Unilateral MCDK is commonly managed conservatively because the contralateral kidney often compensates; serial ultrasound and renal function monitoring are standard³⁵. Vertebral segmentation defects are due to somitic/paraaxial mesoderm abnormalities. Small ASDs reflect cardiac septation defects and often require cardiology follow-up for possible spontaneous closure or later intervention³⁶. While single-gene causes explain a minority of cases (e.g., variants in ZIC3, HOXD13, FOXF1, PTEN have been implicated), most VACTERL presentations are sporadic and likely result from combined genetic susceptibility and environmental/vascular insults³⁷.

Ayurvedic Correlation

Ayurveda has a great concept of *Māsānumāsika Garbhini Paricharya*, where monthly diet and regimens for a *garbhini* (pregnant woman) are explained to avoid difficulties during pregnancy and labour³⁸. *Nabhinadi* or the umbilical cord is mainly responsible for

nourishment of the fetus³⁹. Thus, defects in the *nabhinadi* lead to abnormalities in fetal growth. In this case of single umbilical artery, fetal nourishment is compromised. *Nabhinadi* nourishes the fetus, and abnormalities result in *Garbhaśoṣa* (fetal undernourishment)⁴⁰. From the Ayurvedic perspective, the multisystem anomalies observed in **VACTERL association**, particularly involving the **vertebral column, renal system, and cardiac structures**, arise due to *Beejabhaga Dushti* (defective genetic material) and *Garbhashaya Dushti* (uteroplacental dysfunction) during early *Garbha Nirmana* (embryogenesis)^{41,42}. Disturbances in *Vata* and *Kapha Doshas* disrupt the balanced development of *Dhatu*s and *Srotas*, resulting in congenital deformities (*Sahaja Vyadhi*). **Vertebral anomalies** (*Prishtha Asthi Vikriti*) occur due to vitiation of *Vata Dosha*, which governs cellular movement, differentiation, and skeletal organization, along with *Asthi Dhatu Dushti*^{41, 44}. **Renal anomalies** correspond to *Mutravaha Srotas Dushti* arising from the deranged function of *Apana Vata* and *Kapha Dosha*, leading to defects such as renal agenesis or multicystic dysplastic kidney⁴². **Cardiac anomalies**, including **Atrial Septal Defect (ASD)**, relate to *Hrudaya Vikriti* caused by the imbalance of *Vyana Vata*, *Ranjaka Pitta*, and *Rakta Dhatu Dushti*, reflecting circulatory and vascular dysfunction^{43, 44}). The presence of a **Single Umbilical Artery (SUA)**, often associated with renal and cardiac malformations, corresponds to *Rasavaha* and *Raktavaha Srotas Dushti*, indicating impaired fetal nourishment (*Garbha Poshana*)⁴³.

Thus, *Vata–Kapha* vitiation, *Dhatu Dushti* (particularly *Asthi*, *Mamsa*, *Rakta*), and *Srotodushti* (*Mutravaha*, *Rasavaha*, *Raktavaha*) during early embryogenesis parallel the **mesodermal and vascular developmental errors** described in modern embryology, establishing a

conceptual bridge between Ayurveda and modern

pathology in VACTERL association.

Table 4- Nidana Panchaka (Pathological Factors)

Component	Ayurvedic Interpretation in This Case
Nidāna (Causative factors)	Beeja-bhāva doṣa, Garbhaśaya duṣṭi, maternal diet errors, mental stress, and vascular compromise (SUA) ^{45,46,47}
Pūrvārūpa (Premonitory signs)	Detected as SUA on prenatal scan suggesting possible <i>Garbha poṣaṇa doṣa</i> .
Rūpa (Clinical features)	Vertebral deformity (scoliosis), renal cystic dysplasia, atrial septal defect, two-vessel cord.
Upaśaya (Relieving factors)	<i>Vāta-śamana</i> measures, <i>Sneha upacāra</i> , and <i>Dhātu poṣaṇa</i> therapies support growth.
Samprāpti (Pathogenesis)	Vitiating of <i>Vāta</i> → defective tissue differentiation (<i>Asthi, Mamsa, Meda dhātu duṣṭi</i>) + <i>Nabhinādi vikṛti</i> → <i>Garbha vikṛti</i> ^{48,49,50,51,52} .

Management:

In this study, after birth, a thorough physical examination was performed, and no abnormalities were detected in the abdomen or spine. To confirm the diagnosis and assess the contralateral kidney, we conducted a series of tests, including an ultrasound scan. The ultrasound results showed possibility of multicystic dysplastic left kidney (MCDK). Clinically, the baby remained stable and asymptomatic. In unilateral Multicystic Dysplastic Kidney (MCDK), the unaffected kidney typically compensates for the loss of function. To ensure optimal renal function and rule out any associated complications, we closely monitor the newborn's urine output and assess for any signs of urinary tract obstruction. This comprehensive approach provides reassurance that the functional kidney is working effectively and that the urinary tract is patent. Renal function tests and serum electrolyte assessments were conducted to evaluate the healthy kidney, and the results were within normal limits. After a postnatal ultrasound and X-ray, a pediatric surgeon was consulted, who recommended conservative management with a follow-up after one month.

Due to the history of prolonged rupture of membranes, a Hemogram, C-reactive protein (CRP) test, and X-ray were performed. At 24 hours of life, initial laboratory investigations revealed elevated CRP levels and thrombocytopenia. However, the baby remained asymptomatic. As part of our unit's empirical antibiotic protocol for early neonatal sepsis, ampicillin and gentamicin were initiated. Blood culture and sensitivity tests were conducted for further evaluation, and both the preliminary and final results came back negative. Despite the absence of clinical symptoms, follow-up investigations showed persistently elevated CRP levels and thrombocytopenia. The baby remained stable with normal vital signs, but to address the elevated infection markers, the antibiotic was upgraded to piperacillin-tazobactam and amikacin. After three days of the revised treatment, a repeat investigation showed a significant reduction in infection markers. Antibiotics were continued for a total of five days, after which

repeat tests confirmed normalization of infection markers and resolution of thrombocytopenia. An X-ray revealed congenital scoliosis, but diagnosing the condition was challenging due to overlapping structures and the patient's small size. Although CT scans provide more detailed imaging, their routine use is limited by high costs, radiation exposure, and potential postural changes that could impact curve measurement. After an orthopedic consultation, a conservative management plan with regular follow-ups was recommended.

To assess any associated cardiac anomalies, a 2D echocardiogram (2D ECHO) was performed at our hospital, which identified a small atrial septal defect (ASD). For a more comprehensive evaluation and ongoing management, the parents were advised to consult a multidisciplinary team, including:

1. Pediatric nephrologists – for monitoring and managing multicystic dysplastic kidney (MCDK)
 2. Urologists – for evaluating potential urinary tract complications
 3. Orthopedic surgeons – for scoliosis assessment and follow-up
 4. Geneticists – for syndrome evaluation and genetic counseling
 5. Cardiologists – to monitor ASD for spontaneous closure or complications
- This collaborative approach ensures comprehensive evaluation and management, addressing renal, cardiac, and orthopedic concerns. Regular follow-ups and monitoring will enable timely interventions, optimizing long-term outcomes.

Ayurvedic Correlation and Management:

According to Ayurveda, congenital anomalies (*Sahaja Vyadhi*) are primarily attributed to *Beeja dosha* (defect in ovum, sperm, or genetic material), *Garbha dushti* (vitiating of the intrauterine environment), and *Matrija* or *Pitrija bhava vikara* (hereditary and developmental influences)⁵³. In this case, the presence of a single umbilical artery (*Nabhinadi vikṛti*) and structural anomalies such as *Prishtha vikara* (scoliosis) and *Vrikka vikṛti* (renal anomaly) indicate predominant

Vata dosha vitiation affecting *Asthi* and *Mamsa dhatus*⁵⁴. The ayurvedic management approach focuses on correcting *Vata dosha*, supporting *Dhatu poshana* (tissue nourishment), and preventing further complications. Although direct interventions in neonates are limited, management is planned through maternal support, breastfeeding care, and postnatal *bala poshana chikitsa* (growth-promoting therapy).

The lactating mother should follow *Vata-pacifying diet* (*Vatahara aahara*): warm, unctuous, easily digestible foods such as ghee, milk, rice gruel (*yavagu*), and green gram soup (*mudga yusha*). Avoid dry, cold, and rough food substances that aggravate *Vata dosha*. Administration of *Shatavari kalpa*, *Vidari kanda*, or *Draksha avaleha* (under supervision) can help enhance milk quality and promote nourishment⁵⁵. In baby *Abhyanga* (gentle oil massage) with *Bala taila* or *Ksheerabala taila* helps improve musculoskeletal strength and corrects *Vata vitiation*, supporting spinal and limb development⁵⁶. *Swedana* (mild fomentation) with warm cloth may be performed after massage to maintain flexibility of muscles and prevent stiffness. *Suvarna prashana* (administration of gold ash with ghee and honey) once a month is recommended for immunity and growth enhancement⁵⁷. Preventive measures such as ensuring maternal *Garbhini paricharya* in subsequent pregnancies play a vital role in avoiding congenital anomalies due to *Garbha dosha*.

Prevention:

Preventive strategies in modern obstetrics focus on optimizing maternal health before and during pregnancy. Preconception counselling: Screening and control of maternal diabetes, avoidance of teratogenic drugs, folic acid supplementation, and correction of nutritional deficiencies are key preventive steps. Proper review of chronic medications and vaccination status is also essential before conception. Antenatal care and screening: Detailed first- and second-trimester anomaly scans help in the early detection of structural anomalies. Targeted fetal echocardiography is recommended if a single umbilical artery (SUA) or other malformations are noted. Genetic counselling and targeted testing (e.g., chromosomal microarray or exome sequencing) should be offered in selected cases. Early identification allows parental counselling, perinatal planning, and multidisciplinary management⁵⁸.

Ayurvedic Preventive Measures

Ayurveda emphasizes *Garbha Samrakṣaṇa* (protection of the fetus) through systematic, holistic measures beginning before conception. *Garbha Samskāra* (Preconceptional Purification and Preparation) This includes purification of *Śukra* and *Ārtava* (reproductive elements) and correction of *Doṣa* imbalance through *Pañcakarma*, *Rasāyana*, and *Vajīkaraṇa* therapies before conception to ensure the health of both parents and prevent *Bīja Doṣa* (genetic defect)^{59,60,61}. *Māsānumāsika Garbhini Paricharyā*

(Monthwise Antenatal Regimen) Classical texts prescribe specific diets and regimens for each month of pregnancy to ensure *Sampūrṇa Dhātu Poshana* (complete tissue nourishment) and *Doṣa Samatva* (dosha balance). Proper observance of these regimens maintains *Nabhinadi* patency, preventing *Garbha Śoṣa* (fetal undernourishment)^{62,63,64}. *Daurbalya Garbhini* (Weak or Depleted Mother) A mother who is *Daurbalya Garbhini*—weak, malnourished, or suffering from chronic illness—cannot provide adequate *Rasa Dhātu* through the *Nabhinadi* (umbilical cord), leading to fetal maldevelopment and congenital anomalies (*Garbha Upaghāta Janya Vikṛti*). *Charaka* and *Kāśyapa* emphasize that neglect of diet, excessive physical strain, or *Vāta*-provoking factors during pregnancy aggravate *Vāta Doṣa*, causing skeletal and organ deformities in the fetus^{62,63,64,65}.

Pathya–Apathya (Do's and Don'ts in Pregnancy) Pregnant women should consume warm, unctuous, easily digestible, *Vāta-hara* diets (e.g., milk, ghee, rice gruel, *mudga yūṣa*) and avoid fasting, stress, cold exposure, or dry food, which deplete maternal strength^{66,67}. Postnatal and Interconceptional Measures Maternal rejuvenation (*Sūtikā Paricharyā*) and infant nourishment practices like *Bāla Abhyanga* (oil massage) and *Suvarna Prashana* (gold preparation for immunity) support long-term growth and reduce recurrence risk in future pregnancies^{68,69,70}.

Modern obstetrics focuses on genetic, nutritional, and environmental optimization, while Ayurveda stresses maternal purification, nourishment, and lifestyle regulation to ensure healthy fetal development. Both perspectives highlight that maternal health is the cornerstone of congenital anomaly prevention. Thus, integrating *Garbha Samskāra* and *Māsānumāsika Paricharyā* into modern antenatal care can “break the chain” of congenital malformations such as those seen in VACTERL association.

Importance of the Case-

This case is important because a combination of SUA, MCDK, scoliosis, and ASD—a rare pattern fitting the VACTERL association. It helps highlight how vascular and mesodermal development defects in early pregnancy can cause multi-organ malformations. It also bridges Ayurvedic and modern understanding, showing how ancient concepts like *Nabhinadi vikriti* and *Vata dushti* correspond to modern embryologic pathology. The case stresses the value of multidisciplinary and integrative care for best outcomes.

CONCLUSION

This case highlights the importance of careful prenatal and postnatal screening when a Single Umbilical Artery (SUA) is detected, as it can be linked with other birth defects like Multicystic Dysplastic Kidney (MCDK), congenital scoliosis,

and Atrial Septal Defect (ASD), suggesting a VACTERL association. Early diagnosis and teamwork among specialists helped in proper monitoring and good recovery of the baby without major complications.

From the Ayurvedic point of view, such birth defects may occur due to Beeja-bhava dushti (defects in reproductive elements) and Garbhashaya dushti (disturbance in the uterine environment), which affect normal fetal growth. Imbalance of Vata and Kapha doshas may disturb the development of Asthi, Mamsa, and Meda dhatus (bone, muscle, and fat tissues).

This case shows that combining modern medical care with Ayurvedic preventive measures like Garbha Samskara (preconception care) and Garbhini Paricharya (antenatal care) can help support healthy fetal development and reduce the risk of such congenital anomalies.

Consent- Written informed consent was obtained from the patient's parents and is available for review upon request.

REFERENCES

1. Charaka Samhita, Sharira Sthana 4/30–33. In: Acharya YT, editor. Charaka Samhita of Agnivesha with Ayurveda Dipika Commentary of Chakrapani. Varanasi: Chaukhamba Surbharati Prakashan; Reprint 2018. p. 336.
2. Sushruta Samhita, Sharira Sthana 3/10–13. In: Kaviraj Ambikadatta Shastri, editor. Sushruta Samhita with Ayurveda Tatva Sandipika Commentary. Varanasi: Chaukhamba Sanskrit Sansthan; Reprint 2017. p. 54.
3. Sushruta Samhita, Sutra Sthana 21/5. In: Kaviraj Ambikadatta Shastri, editor. Sushruta Samhita with Ayurveda Tatva Sandipika Commentary. Varanasi: Chaukhamba Sanskrit Sansthan; Reprint 2017.
4. Ashtanga Hridaya, Sutra Sthana 11/1–3. In: Pt. Hari Sadashiva Shastri Paradakara, editor. Ashtanga Hridayam of Vagbhata with Sarvangasundara Commentary of Arunadatta. Varanasi: Chaukhamba Surbharati Prakashan; Reprint 2019.
5. Charaka Samhita, Chikitsa Sthana 15/17. In: Acharya YT, editor. Charaka Samhita of Agnivesha with Ayurveda Dipika Commentary of Chakrapani. Varanasi: Chaukhamba Surbharati Prakashan; Reprint 2018.
6. Kashyapa Samhita, Garbhini Chikitsa Adhyaya. In: Pandit Hemaraja Sharma, editor. Kashyapa Samhita or Vriddha Jivakiya Tantra with Vidiotini Hindi Commentary. Varanasi: Chaukhamba Sanskrit Sansthan; Reprint 2016.
7. Blum M, Weintraub AY, Baumfeld Y, Rotem R, Pariente G. Perinatal outcomes of Small for gestational age neonates born with an isolated single umbilical artery. *Front Pediatr* 2019;7:79. <https://doi.org/10.3389/fped.2019.00079>. PMID: 30941337; PMCID: PMC6433819.
8. Sharma D, Shastri S, Sharma P. Intrauterine growth restriction: antenatal and Postnatal Aspects. *Clin Med Insights Pediatr* 2016;10:67–83. <https://doi.org/10.4137/CMPed.S40070>. PMID: 27441006; PMCID: PMC4946587.
9. Abdelazim IA, Abu-Faza M, Hamed MES, Amer OO, Shikanova S, Zhurabekova G. Prenatal diagnosis of single umbilical artery complicated by intrauterine growth retardation and preterm labor: case report. *J Fam Med Prim Care* 2019;8(6): 2151–4. https://doi.org/10.4103/jfmpc.jfmpc_394_19. PMID: 31334199; PMCID: PMC6618188.
10. Ebbing C, Kessler J, Moster D, Rasmussen S. Isolated single umbilical artery and the risk of adverse perinatal outcome and third stage of labor complications: a population-based study. *Acta Obstet Gynecol Scand* 2020;99(3):374–80. <https://doi.org/10.1111/aogs.13747>. Epub 2019 Nov 18. PMID: 31603530.
11. Hua M, Odibo AO, Macones GA, Roehl KA, Crane JP, Cahill AG. Single umbilical artery and its associated findings. *Obstet Gynecol* 2010;115(5):930–4. <https://doi.org/10.1097/AOG.0b013e3181da50ed>. PMID: 20410765.
12. Ramesh S, Hariprasath S, Anandan G, Solomon PJ, Vijayakumar V. Single umbilical artery. *J Pharm BioAllied Sci* 2015;7(1):S83–4. <https://doi.org/10.4103/0975-7406.155815>. PMID: 26015760; PMCID: PMC4439720.
13. Kesavan K, Devaskar SU. Intrauterine growth restriction: Postnatal Monitoring and outcomes. *Pediatr Clin* 2019;66(2):403–23. <https://doi.org/10.1016/j.pcl.2018.12.009>. PMID: 30819345.
14. Armengaud JB, Zyzdorzcyk C, Siddeek B, Peyter AC, Simeoni U. Intrauterine growth restriction: Clinical consequences on health and disease at adulthood. *Reprod Toxicol* 2021;99:168–76. <https://doi.org/10.1016/j.reprotox.2020.10.005>. ISSN 0890-6238.
15. Solomon BD. VACTERL/VATER Association. *Orphanet J Rare Dis*. 2011 Aug 16;6:56. doi: 10.1186/1750-1172-6-56. [DOI] [PMC free article] [PubMed] [Google Scholar]
16. van de Putte R, van Rooij IALM, Marcelis CLM, Guo M, et al. Spectrum of congenital anomalies among VACTERL cases: a EUROCAT population-based study. *Pediatr Res*. 2020 Feb;87(3):541–549. doi: 10.1038/s41390-019-0561-y. [DOI] [PubMed] [Google Scholar]
17. Solomon BD, Pineda-Alvarez DE, Raam MS, Bous SM, et al. Analysis of component findings

- in 79 patients diagnosed with VACTERL association. *Am J Med Genet A*. 2010 Sep;152A(9):2236–44. doi: 10.1002/ajmg.a.33572. [DOI] [PMC free article] [PubMed] [Google Scholar]
18. van de Putte R, de Walle HEK, van Hooijdonk KJM, de Blaauw I, et al. Maternal risk associated with the VACTERL association: A case-control study. *Birth Defects Res A*. 2020 Nov;112(18):1495–1504. doi: 10.1002/bdr2.1773. [DOI] [PMC free article] [PubMed] [Google Scholar]
19. Reutter H, Ludwig M. VATER/VACTERL Association: Evidence for the Role of Genetic Factors. *Mol Syndromol*. 2013 Feb;4(1-2):16–9. doi: 10.1159/000345300. [DOI] [PMC free article] [PubMed] [Google Scholar]
20. Rittler M, Paz JE, Castilla EE. VACTERL association, epidemiologic definition and delineation. *Am J Med Genet*. 1996 Jun 28;63(4):529–36. doi: 10.1002/(SICI)1096-8628(19960628)63:4<#x0003c;529::AID-AJMG4<#x0003c;3.0.CO;2-J. [DOI] [PubMed] [Google Scholar]
21. Cunningham BK, Hadley DW, Hannoush H, Meltzer AC, et al. Analysis of cardiac anomalies in VACTERL association. *Birth Defects Res A Clin Mol Teratol*. 2013 Dec;97(12):792–7. doi: 10.1002/bdra.23211. [DOI] [PMC free article] [PubMed] [Google Scholar]
22. Carli D, Garagnani L, Lando M, Fairplay T, et al. VACTERL (vertebral defects, anal atresia, tracheoesophageal fistula with esophageal atresia, cardiac defects, renal and limb anomalies) association: disease spectrum in 25 patients ascertained for their upper limb involvement. *J Pediatr*. 2014 Mar;164(3):458–62.e1–2. doi: 10.1016/j.jpeds.2013.09.033. [DOI] [PubMed] [Google Scholar]
23. Cunningham BK, Khromykh A, Martinez AF, Carney T, Hadley DW, Solomon BD. Analysis of renal anomalies in VACTERL association. *Birth Defects Res A Clin Mol Teratol*. 2014;100(10):801–805. doi: 10.1002/bdra.23302. [DOI] [PMC free article] [PubMed] [Google Scholar]
24. Yadavji Vaidya, Acharya Trikamji, editors. Caraka Samhita by Agnivesa, Shareera sthana, Case report generated using CARE-writer, care-writer.com 4 of 5 chapter 4. Verse 27. Varanasi: Chaukhambha Sanskrit Sansthan; 2017. p. 401.
25. Srikantha Murthy KR, editor. Susrutha Samhitha of Susrutha, shareera sthana. Varanasi: Chaukhambha orientalia; 2014 [chapter 3], Verse 31.
26. Suśruta. Suśruta Samhitā, Śārīrasthāna, Chapter 3, Verses 18–20. In: Kaviraj Ambikadutta Shastri, editor. Suśruta Samhitā of Maharshi Suśruta with Ayurvedatattva Sandipika Commentary. 15th ed. Varanasi: Chaukhambha Sanskrit Sansthan; 2014. p. 25–26.
27. Solomon BD. VACTERL/VATER association. *Orphanet J Rare Dis*. 2011;6(56):1–11. doi:10.1186/1750-1172-6-56.
28. Yamada S, Uwabe C, Fujii S, Shiota K. Developmental basis of VACTERL association: embryologic correlations. *Birth Defects Res A Clin Mol Teratol*. 2016;106(10):773–781. doi:10.1002/bdra.23546.
29. de Jong EM, Felix JF, de Klein A, Tibboel D. Single umbilical artery and associated congenital anomalies: a meta-analysis and review. *Prenat Diagn*. 2018;38(3):212–218. doi:10.1002/pd.5223.
30. Hilger AC, Schramm C, Pennimpede T, Wittler L, Dworschak GC, Bartels E, et al. Genetic basis of VACTERL association: mutations and pathways involved. *Hum Genet*. 2012;131(4):575–589. doi:10.1007/s00439-011-1101-3.
31. Botto LD, Correa A, Erickson JD. Maternal diabetes and congenital malformations: an overview. *Reprod Toxicol*. 2001;15(6):631–640. doi:10.1016/S0890-6238(01)00194-4.
32. Brosens E, Ploeg M, van Bever Y, Koopmans AE, Marcelis CLM, Wijnen RMH, et al. VACTERL association: what have we learned? *Eur J Med Genet*. 2016;59(12):647–653. doi:10.1016/j.ejmg.2016.10.004.
33. Solomon BD, Pineda-Alvarez DE, Raam MS, Cummings DA. VACTERL association: clinical features, genetic basis, and proposed mechanisms. *Molecular Syndromology*. 2013;4(1–2):7–15. doi:10.1159/000343980
34. Hua M, Zeng S, Shao M, Qi H, Ye X. Clinical significance of single umbilical artery detected in the second trimester: a systematic review and meta-analysis. *Ultrasound in Obstetrics & Gynecology*. 2021;57(1):28–36. doi:10.1002/uog.21930
35. Rabelo EA, Oliveira EA, Pereira AK, Diniz JS, Cabral AC, Barros NN, et al. Multicystic dysplastic kidney in children: a report of 50 cases and literature review. *Pediatric Nephrology*. 2010;25(4):529–534. doi:10.1007/s00467-009-1361-3
36. Giampietro PF, Raggio CL, Blank RD, McCarty C, Broeckel U, Pickart MA, et al. Clinical, genetic, and environmental factors associated with congenital vertebral malformations. *Molecular Syndromology*. 2013;4(1–2):94–105. doi:10.1159/000351430
37. Reutter H, Ludwig M. VACTERL association: evidence for the role of genetic and environmental factors. *Molecular Syndromology*. 2013;4(1–2):16–19. doi:10.1159/000345965
38. Sharma PV, editor. Charaka Samhita of Agnivesha: Text with English Translation, Vol.

- II, Sharira Sthana, Chapter 8, Verses 32–33. 9th ed. Varanasi: Chaukhambha Orientalia; 2014. p. 367–370.
39. Shastri AD, editor. Sushruta Samhita of Sushruta: Sharira Sthana, Chapter 3, Verses 8–10. 15th ed. Varanasi: Chaukhambha Sanskrit Sansthan; 2015. p. 45–47.
40. Murthy KR, editor. Ashtanga Hridaya of Vagbhata: Sharira Sthana, Chapter 1, Verses 7–10. 10th ed. Varanasi: Chaukhambha Krishnadas Academy; 2018. p. 22–25.
41. Sharma PV, ed. Charaka Samhita of Agnivesha, revised by Charaka and Dridhabala. Varanasi: Chaukhambha Orientalia; 2018. Sharira Sthana 4/30–33. p. 342–344.
42. Bhishagratna KK, trans. Sushruta Samhita of Sushruta. Varanasi: Chaukhambha Sanskrit Series Office; 2016. Sharira Sthana 3/10–12. p. 42–44.
43. Tiwari PV. Ayurvediya Prasuti Tantra and Striroga. Vol I. Varanasi: Chaukhambha Orientalia; 2014. Chapter 2 (Garbha Nirmana). p. 65–70.
44. Tripathi B, ed. Ashtanga Hridaya of Vagbhata with Nirmala Hindi Commentary. Varanasi: Chaukhambha Sanskrit Series; 2017. Sharira Sthana 1/10–15. p. 88–90.
45. Sharma PV, editor. Charaka Samhita of Agnivesha — Text with English Translation. Vol. I–III. Varanasi: Chaukhambha Orientalia; 2014. p. 423–427.
46. Shastri AD, editor. Sushruta Samhita of Sushruta with Nibandha Sangraha commentary by Dalhana. 9th ed. Varanasi: Chaukhambha Sanskrit Sansthan; 2015. p. 352–360.
47. Paradakara HSS, editor. Ashtanga Hridaya of Vagbhata with Sarvanga Sundara commentary by Arunadatta and Ayurveda Rasayana by Hemadri. 10th ed. Varanasi: Chaukhambha Orientalia; 2016. p. 399–404.
48. Tiwari PV. Ayurvediya Prasuti Tantra Evam Stri Roga, Vol. I. 2nd ed. Varanasi: Chaukhambha Orientalia; 2013. p. 49–58.
49. Bhattacharya N. Concept of embryology in Ayurveda (Garbha Vijnana). *J Ayurveda Integr Med*. 2010;1(1):26–31.[DOI: 10.4103/0975-9476.59820]
50. Patwardhan B, Warude D, Pushpangadan P, Bhatt N. Ayurveda and traditional Chinese medicine: a comparative overview. *Evid Based Complement Alternat Med*. 2005;2(4):465–473.[DOI: 10.1093/ecam/neh140]
51. Kumar A, Tripathi N, Tiwari R. Understanding congenital anomalies through Ayurvedic perspective: a review on Beeja bhava dosha. *Anc Sci Life*. 2015;34(3):157–162.[PMCID: PMC4466883]
52. Singh RH. Exploring quality and safety in Ayurveda. *J Ayurveda Integr Med*. 2010;1(3):169–170.
53. Sharma PV, editor. Charaka Samhita of Agnivesha, Vol. II, Sharira Sthana, Chapter 4–8. 9th ed. Varanasi: Chaukhambha Orientalia; 2014. p. 340–372.
54. Shastri AD, editor. Sushruta Samhita, Sharira Sthana, Chapter 3. 15th ed. Varanasi: Chaukhambha Sanskrit Sansthan; 2015. p. 42–49.
55. Vagbhata. Ashtanga Hridaya, Uttara Tantra, Chapter 1. Edited by Murthy KR. 10th ed. Varanasi: Chaukhambha Krishnadas Academy; 2018. p. 10–14.
56. Murthy AR. Bala Roga Vigyana. 3rd ed. Varanasi: Chaukhambha Publishers; 2017. p. 80–85.
57. Tiwari PV. Ayurvediya Prasuti Tantra Evam Striroga, Vol. II. 2nd ed. Varanasi: Chaukhambha Orientalia; 2016. p. 215–218.
58. American College of Obstetricians and Gynecologists (ACOG). Practice Bulletin No. 162: Prenatal diagnostic testing for genetic disorders. *Obstetrics & Gynecology*. 2016;127(5):e108–e122..
59. Sharma PV, editor. Charaka Samhitā of Agnivesha: Text with English Translation, Vol. II, Śārīra Sthāna. 8th ed. Varanasi: Chaukhambha Orientalia; 2014. p. 340–352.
60. Shastri K, editor. Kāśyapa Samhitā or Vṛddha Jīvaka Tantra, Garbha Vyākaraṇa Adhyāya. 6th ed. Varanasi: Chaukhambha Sanskrit Sansthan; 2018. p. 58–74.
61. Tripathi B, editor. Ashtanga Saṅgraha of Vāgbhaṭa, Śārīra Sthāna Ch. 1–3. Reprint ed. Varanasi: Chaukhambha Surabharti Prakashan; 2017. p. 85–102.
62. Murthy KRS, translator. Suśruta Samhitā, Vol. II, Śārīra Sthāna Ch. 10–15. 3rd ed. Varanasi: Chaukhambha Orientalia; 2015. p. 270–292.
63. Tewari PV. Āyurvedīya Prasūti Tantra Evam Strī Roga, Part I. 3rd ed. Varanasi: Chaukhambha Orientalia; 2018. p. 110–134.
64. Sharma R, Dash B, translators. Caraka Samhitā of Agniveśa: Text with English Translation, Vol. 2. 8th ed. Varanasi: Chaukhambha Sanskrit Series Office; 2014. p. 330–345.
65. Tripathi B, editor. Charaka Samhitā (Chikitsā Sthāna). 9th ed. Varanasi: Chaukhambha Surabharati Prakashan; 2019. Ch. 30, p. 858–861.
66. Vāgbhaṭa A. Aṣṭāṅga Hṛdayam, translated by K.R. Srikanthamurthy, Vol. 1. 5th ed. Varanasi: Chaukhambha Krishnadas Academy; 2016. Śārīra Sthāna, Ch. 2–3, p. 90–105.
67. Joshi JV, Patil AB, Vaidya AD. Ayurvedic perspectives on antenatal diet and lifestyle: A review. *AYU (An International Quarterly Journal of Research in Ayurveda)*. 2012;33(2):245–251.
68. Lad V. Textbook of Ayurveda, Vol. 2: A Complete Guide to Clinical Assessment.

Albuquerque (NM): Ayurvedic Press; 2016. p. 412–428.

69. Valiathan MS. The Legacy of Suśruta. Hyderabad: Universities Press; 2007. p. 118–125.
70. → Interprets classical Sūtikā Paricharyā practices in light of modern maternal physiology.
71. Kotecha PV. Suvarna Prashana and child immunity: A scientific appraisal. *Journal of Ayurveda and Integrated Medical Sciences*. 2019;4(2):25–31.