CASE REPORT

Congenital talipes equinovarus and congenital vertical talus secondary to sacral agenesis

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SUMMARY

Sacral agenesis is a rare congenital defect which is associated with foot deformities such as congenital talipes equinovarus (CTEV) and less commonly congenital vertical talus (CVT). We report a 3-year-old Caucasian girl who was born with right CTEV and left CVT secondary to sacral agenesis. Her right foot was managed with a Ponseti casting method at 2 weeks, followed by an Achilles tenotomy at 4 months. The left foot was initially managed with a nocturnal dorsi-flexion splint. Both feet remained resistant and received open foot surgery at 10 months producing plantigrade feet with neutral hindfeet. At 19 months, she failed to achieve developmental milestones and examinations revealed abnormal lower limb reflexes. A full body MRI was performed which identified the sacral agenesis. We advocate early MRI of the spine to screen for spinal defects when presented with resistant foot deformities, especially when bilateral.

BACKGROUND

Sacral agenesis is a rare radiological diagnosis consisting of variable osteological defects of the sacrum and associated spine.¹ Otherwise known as the clinical entity caudal regression syndrome, this condition is more common with gestational diabetes and is associated with lower limb deformities.^{2–5} Congenital talipes equinovarus (CTEV) is a common foot deformity occurring in 1.2 of 1000 births in Europe⁶, whereas congenital vertical talus (CVT) is more uncommon with an incidence estimated at 1 in 10 000.⁷ If left uncorrected, both deformities lead to substantial disability, pain and functional impairment.

CTEV presents as clubfoot and consists of plantar-medial subluxation of the calcaneus and navicular bone relative to an abnormally small talus.⁸ Similarly, CVT also has talonavicular joint subluxation, although in the dorso-lateral direction producing a rocker bottom foot appearance.⁹ The deformities can be resistant to realignment and are often held in malposition by the tendons and ligaments of the foot and ankle. The varus deformity of CTEV is held in an inverted and internally rotated position by tibialis posterior, flexor digitorum longus and flexor hallucis longus and in hindfoot equinus by Achilles tendon contracture (figure 1A),⁸ whereas the valgus deformity of CVT is held in an everted and externally rotated position by the tibialis anterior and peroneal tendons (figure 1B).⁹ In CTEV, the Pirani score is used to grade severity and monitor treatment outcomes. A cumulative score is

created based on constituent features of deformity from the midfoot and hindfoot.¹⁰

The majority of the cases of CTEV are idiopathic; however, non-idiopathic CTEV can arise as a result of a variety of aetiologies. The most common is secondary to genetic abnormalities (41%), closely followed by neuropathic disorders (37%), but it can also be due to muscular disorders (18%) and various other conditions.^{11 12} The aetiology of CVT is also unclear, previous studies have suggested that around half of cases are non-idiopathic and associated with genetic syndromes^{13 14} and neurological abnormalities.¹⁵ Regardless of aetiology, the mainstay of modern treatment for CTEV starts with the Ponseti method of casting, involving serial plaster of paris casting followed by limited surgery if required.¹⁶

CASE PRESENTATION

A 2-week-old Caucasian girl was referred to paediatric foot and ankle clinic for bilateral foot deformities which had not improved since birth. No deformity had been noted following antenatal ultrasound screening. She was born at full term following an emergency caesarean section for foetal distress. There was no family history of congenital deformities. The mother had a history of type 1 diabetes mellitus which was monitored throughout pregnancy in antenatal clinics. The child was neither a twin birth nor found to be in a breech position at birth.

On examination, she had a normal spine, stable hips with negative Barlow and Ortolani tests and no leg length discrepancy. She had a stiff right CTEV with a Pirani score of 5.5 and on the left side a calcaneovalgus foot which was only partially correctable. There was no bowing of the left tibia. On peripheral neurological examination, it proved difficult finding Achilles and plantar reflexes, but no other neurology was detected. No MRI was requested at this stage, and reflex development was monitored closely in clinic by the paediatrician and paediatric orthopaedic surgeon.

INVESTIGATIONS

Because of the slow response to initial treatment, at 4 months of age, bilateral radiographs were produced of the foot and ankle. They showed a vertical talus in the left foot (figure 2A), explaining the calcaneovalgus position. The right foot radiographs were less clear, but revealed that the right calcaneum was parallel to the talus and in equinus (figure 2B).

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Figure 1 (A) Right congenital talipes equinovarus and (B) left congenital vertical talus with a schematic diagram showing net muscular force direction following muscular imbalance (red) and direction of joint subluxation (blue).

Foot deformities secondary to additional conditions are often resistant to treatment. Therefore, as little progress had been made by 4 months, genetic screening was performed but found to be normal. Despite these results and with the addition of the child's inability to walk until 25 months, a full body MRI was performed which revealed sacral agenesis. Figure 3 is a lumbar MRI T2-weighted sagittal midline frame showing a transitional appearance of L5 and almost complete lack of a sacrum apart from a small portion of S1. The thecal sac ends abnormally high around the lower border of L3, and the spinal cord ends bluntly at the upper border of T12.

DIFFERENTIAL DIAGNOSIS

At birth, CVT may be difficult to differentiate from the more benign positional calcaneovalgus, caused by restricted intra-uterine which tends to improve spontaneously within months.¹⁷ Furthermore, other sinister conditions must be ruled out:

1. Paralytic calcaneus foot deformity—A deformity of the calcaneus secondary to relatively weak plantar flexion which may result from paralysis or absence of the superficial posterior compartment of the leg. Hence, it could be differentiated radiologically and would present clinically as a motor deficit during the physical examination.¹⁸
2. Posteromedial bow of the tibia—Bowing of a shortened tibia which presents as a calcaneovalgus foot. This can be differentiated by detection of a leg-length discrepancy on examination and using radiology.¹⁹

There are several differential aetiologies for non-idiopathic CTEV other than sacral agenesis, many of which can be differentiated using MRI:

1. Tethered cord syndrome—This neurological condition can accompany sacral agenesis and often presents in children with back pain, deteriorating urological symptoms having achieved successful toilet training and progressive motor and

Figure 2 Foot and ankle radiographs at 4 months of age showing right congenital talipes equinovarus in an equino-serpentine position following Ponseti casting and left congenital vertical talus.

Figure 3 T2-weighted sagittal midline lumbosacral MRI frame showing sacral agenesis.

sensory dysfunction.²⁰ Clinical evaluation in conjunction with an MRI of the entire spine showing a low conus medullaris and thickened filum terminalis is the gold standard method of diagnosis.²¹

2. Myelomeningocele—Often evident at birth on examination, but can be confirmed using antenatal MRI and ultrasound.²²
3. Genetic abnormalities such as Edwards' syndrome (Trisomy 18)—Phenotypic features often include, for example, intrauterine growth restriction, hypertonia and micrognathia. Diagnosis can be confirmed using conventional karyotyping.²³
4. Amniotic band sequence—Prenatal ultrasound can reveal bands of amnion crossing the gestational sac resulting in often asymmetrical limb defects or amputations. If suspected, diagnosis can be confirmed using three-dimensional ultrasound and MRI.²⁴
5. Arthrogryposis multiplex or distal arthrogryposis—Postnatally, a diagnosis can be made by physical examination and can be confirmed using ultrasound, MRI or muscle biopsy.²⁵

TREATMENT

The Ponseti method of casting was initiated for the right CTEV within 2 weeks of birth, and after 4 weeks and five casts, the right foot's Pirani score improved from 5.5 to 3. Despite the initial improvement, it then remained resistant with an uncorrected equinus position. The left-sided CVT was in a calcaneovalgus position and was initially treated with plantar flexion stretches. Four weeks later, the left foot was not improving and found to be in 20° of dorsiflexion which passively corrected only to plantigrade. This was subsequently treated with a nocturnal dorsi-flexion splint aiming to hold the forefoot in alignment.

Following weekly casts, at 4 months of age, a right Achilles tenotomy was completed as the right-sided talipes remained in equinus. As the flexor tendons were markedly stiff, the forefoot remained plantarflexed, leaving a mid-foot break. An above knee cast was applied which eventually dislodged allowing the foot to return into a stiff equinus position.

The right CTEV continued to prove resistant to treatment and with the confirmation of CVT in the left foot, both feet underwent

open foot surgery at 10 months of age. The right-sided atypical talipes was addressed with Z lengthening of the tendo-Achilles and the hindfoot varus was subsequently corrected by capsular release of both the ankle and subtalar joints. This enabled passive manipulation into a plantigrade position with a neutral hindfoot and was held with K-wires from the posterior-lateral corner of the talus into the navicular.

The left-sided CVT was addressed with a Cincinnati incision from tibialis anterior to the tendo-Achilles, followed by division of the dorsal and inferior capsule of the talonavicular joint. The vertical talus was subsequently reduced by Z lengthening tibialis anterior and posterior, along with release of the lateral talonavicular joint and posterior capsule of both ankle and subtalar joints. A K-wire was inserted to maintain the reduced talonavicular joint. Furthermore, Z lengthening of the tendo-Achilles and release of the talofibula ligaments enabled alignment of the calcaneum. However, the peroneal tendons were not surgically adjusted.

Two months postoperatively, when the K-wires were removed, the right foot was stiffer but could be passively dorsiflexed to 10°, whereas the left foot could reach 20°. Nevertheless, both feet were in a neutral position. Following cast removal, boots and bars were worn bilaterally for 23 hours per day for 4 weeks, with the left in slight plantarflexion. After 4 weeks, the boots and bars were only worn at night.

During follow-up at 17 months of age, both feet were in a plantigrade position with neutral hindfoot. She did not have the power to stand until 24 months of age, at which point patella knee jerk reflexes were normal but Achilles and plantar reflexes were absent. Following diagnosis of sacral agenesis at 25 months of age, at 31 months an abnormal lumbosacral skin fold became apparent, along with atrophic calf muscles and no sensation on the soles of her feet. There was a risk that worsening foot deformity and impairment of predominantly L5 and S1 nerve roots may impede her long-term ability to walk. Therefore, the main aim neurologically was to maximise walking and minimise mechanical loading through preventing excessive weight gain which would add extra strain to her weak lower limbs.

OUTCOME AND FOLLOW-UP

From 2 years of age, she has started walking independently and even demonstrated on multiple occasions the ability to jump. At 3 years, the left-sided CVT continues to pose difficulties with increasing pain in her left ankle and foot as a result of worsening valgus deformity, thought to be due to a relative imbalance of the peroneus tendon relative to tibialis posterior.

Treatment will be individualised and may involve tendon transfer or tendon release surgery between the ages of 3 and 6 years. Bone corrective surgery may be required between the ages of 6 and 10 years, followed by joint fusion surgery after 10 years if required.

At the age of 3 years, she did not experience urinary incontinence, and ultrasonography showed normal sized kidneys, with no ureteric dilatation or hydronephrosis. The urinary bladder was also semi-full but normal in appearance. Bowel function tended towards faecal incontinence several weeks postnatally and was then followed by constipation at around 4 months of age. The constipation was treated with 2–3 hospital admissions for disimpaction and enema treatment.

DISCUSSION

Foot and ankle deformities are common in sacral agenesis, featuring in 63% of cases.²⁶ However, sacral agenesis is very

uncommon in infants with foot and ankle deformities.¹¹ The mean age at initial diagnosis of sacral agenesis in individuals without myelomeningocele is 15 months, which is well past the ideal time to treat associated foot deformities.²⁷ A bilateral foot deformity pattern also indicates a non-idiopathic cause, as well as the deformity being resistant to the Ponseti method of casting.¹¹ Indeed, cases of non-idiopathic CTEV have been shown to require more casts and are associated with a higher rate of recurrence than their idiopathic equivalents which have an overall recurrence rate of 4%.¹¹ In contrast to idiopathic CTEV, non-idiopathic CTEV is, therefore, often treated using extensive soft tissue release surgery.^{28–29} Myelomeningocele is the most common neuropathic cause of CTEV and is associated with a much higher recurrence rate of 68%³⁰ relative to other common causes such as amniotic band syndrome or distal arthrogryposis with recurrence rates of 33%³¹ and 27%,³² respectively. It is likely that similar to myelomeningocele, the pathophysiology by which sacral agenesis produces these foot deformities also involves muscular imbalances. Similar to non-idiopathic CTEV, virtually all cases of CVT are only fully corrected with major reconstructive surgery, usually preceded by serial manipulation and casting.^{33–34} Therefore, prompt identification and diagnosis of sacral agenesis is key in providing appropriate orthopaedic investigations and planning of future treatments.

Early identification of sacral agenesis is also important as it often presents as part of a caudal regression syndrome which includes a spectrum of associated problems, such as neuropathic bladder and bowel dysfunction. Urinary symptoms including incontinence in children ≥ 4 years of age and recurrent urinary tract infections are common in patients with sacral agenesis and are reported at 85% and 74%, respectively.³⁵ Moreover, other urinary symptoms including vesicoureteric reflux, substantial postvoiding residual urine and high maximal voiding pressures are also common.³⁵ These symptoms can have important implications for both the patient's quality of life and can contribute to renal impairment. Indeed, renal function is impaired in around 9% of patients with caudal regression syndrome and so cystourethograms are routine in these patients to identify vesicoureteric reflux and minimise infections.^{36–37} Timely and appropriate intervention has been shown to result in social continence in the vast majority of patients, with 95% remaining dry for at least 4 hours.³⁶

Bowel problems are also common in patients with sacral agenesis, with up to 73% experiencing constipation³⁸ and 29% experiencing faecal incontinence.³⁹ Intermittent constipation and faecal incontinence have also been reported.⁴⁰ Depending on the extent of the dysfunction, the importance of distinguishing 'overflow pseudoincontinence' from 'true faecal incontinence' has been stressed in devising management for symptoms.⁴¹ Rectal manometry has been used to suggest that problems may arise due to abnormal parasympathetic innervation,⁴² and in some cases, sacral agenesis is associated with anorectal abnormalities which may be the cause of symptoms.⁴³ Antegrade continence enemas have been used with a high rate of success particularly in individuals with a tendency towards constipation.⁴⁴ It is well established that the extent of sacral deformity does not correspond well to the severity of bowel and urological problems.^{37–45}

There is a lack of detailed reports of treatment for foot deformity specifically associated with sacral agenesis. A Polish study with five cases emphasised the need for a personalised approach to treatment and aimed to have patients walking by their first birthday.⁴⁶ The goal of treatment is to achieve a painless, plantigrade foot, using any active power to help gait. However, prognosis in children with foot deformities secondary to sacral agenesis is very variable. Two patients with resistant CTEV and rigid calcaneovalgus

remained unable to walk due to impairment of motor innervation descending from around T12/L1,⁴⁷ whereas another patient with motor impairment from around L4/S1 started walking at 4 years following successfully corrected bilateral CTEV and had not relapsed 2 years later.⁴⁷ Cases of bilateral CTEV have also been reported in patients with severe caudal regression syndrome, associated with similar degrees of variable morbidity.⁴

Learning points

- Consider spinal problems in foot deformities proving difficult to treat and investigate using an anterior–posterior pelvic radiograph and then an MRI.
- It is important to identify sacral agenesis early to protect renal function in the case of neuropathic bladder and to correctly manage bowel symptoms.
- Patients with congenital foot deformities secondary to sacral agenesis can be particularly resistant to treatment.
- The long-term outcome of children with foot deformities and an underlying spinal pathology will be determined by a combination of mechanical and neurological variables.

Contributors JJHB is responsible for writing the first manuscript, producing the schematic diagram and extensively reviewing the literature surrounding the case. SC is responsible for obtaining consent and radiology images. RB is the consultant orthopaedic surgeon who performed the surgery, conceptualised the case report and thoroughly reviewed the introductory and background literature. All authors edited and reviewed the manuscript and have approved the final version.

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