Case Report

Congenital ‘butterfly vertebra’ associated with low back pain: a case report

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The butterfly vertebral defect is a rare congenital anomaly of the spine, which is generally considered benign. In this report we present the case of an active young man who presented with recurrent low back pain (LBP), and was found to have a butterfly vertebral defect at the symptomatic L4 lumbar spinal level. We describe the genesis of the butterfly vertebral defect, in the context of normal embryological development of the human vertebra and intervertebral disk. We report the clinical examination findings and therapeutic interventions undertaken prior to the radiographic discovery of the vertebral defect, and discuss the impact that the presence of a butterfly vertebral defect presented to therapeutic decision-making.

Keywords: Vertebrae abnormalities, Lumbar spine, Butterfly vertebral defect, Physical therapy interventions, Manual therapy

Introduction

Low back pain (LBP) is a common complaint presenting to physical therapists and can have many causes. Accurate diagnosis of LBP can be difficult due to a lack of validated clinical examination procedures for assessing the integrity of the various anatomical structures of the region, heterogeneous patterns of symptom reproduction, and psychosocial variables.^{1,2}

This case report describes the assessment and treatment of a patient (Mr BV) who presented to physical therapy with left-sided LBP, as well as pain in the left buttock and proximal posterolateral thigh. These are common symptoms for patients who present with the chief complaint of LBP, but in this case an uncommon anomaly was found by radiography – a butterfly vertebra. The purpose of this case report is to: (i) review the normal growth and development of the human spine; (ii) describe the butterfly vertebral deformity and; (iii) consider the impact of vertebral anomalies on LBP and therapy choices.

Butterfly Vertebra

Butterfly vertebra is an uncommon congenital anomaly of the spine, which is generally benign. Butterfly vertebra was first described in 1844, and is known variously as: cleft vertebra, sagittal cleft vertebra,

anterior rachischisis, anterior somatoschisis, and anterior spina bifida.^{3,4} It typically appears in the lumbar spine, and may occur either as an independent anomaly or associated with a syndrome of anomalous deformities affecting other skeletal regions and/or other systems; particularly the gastro-intestinal, genito-urinary, and central nervous systems.³ It may be associated with kyphoscoliosis.^{4,5}

Butterfly vertebra develops *in utero*, during somatogenesis, between the third and sixth weeks of gestation.^{3,6} Normally the somites, which are of mesenchymal tissue, begin to become cartilaginous at two chondrification centers, which then fuse to give rise to a single vertebral body. Between the vertebral bodies, a region of mesenchyme where adjacent somites meet develops a higher density and becomes the intervertebral disk.⁶ As the two chondrification centers of the vertebral body fuse, the notochord is ‘squeezed out’ into the intervertebral disk, where it contributes to the development of the nucleus pulposus.⁶ Failure of this fusion of the two halves of the vertebral body, believed to be due to persistent remnants of the notochord, results in the butterfly vertebral deformity.^{3,7,8} Typically, the vertebrae and intervertebral disks above and below compensate for the defect by elongating about the midline.³ See Fig. 1 for an overview of the normal growth and development of the vertebrae, and the development of the butterfly vertebra defect.

The defect is often an incidental finding in medical imaging, although it may, as is proposed in this case, be associated with LBP.^{4,9,10} Owing to the rarity of the

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Figure 1 Growth and development of the human vertebrae (adapted from Ref. 46). N: notochord; A: somites form two blocks of mesenchymal tissue, separated by the notochord; B: chondrification centers form within the mesenchymal blocks; C: the chondrifying blocks 'squeeze' the notochord out from between them, and coalesce. The notochord expands in the center of the intervertebral disk, to contribute to the nucleus pulposus; D: the cartilaginous vertebral body starts to ossify at a primary ossification center in the centrum. Two other primary ossification centers appear in each half of the neural arch (these unite posteriorly in the first postnatal year, however bony fusion of the neural arch to the centrum does not occur until approximately 6 years of age). Vertebral endplates begin to unite with the developing intervertebral disk, by collagen fibers. These are well developed by the sixth gestational month; E: multiple ossification centers form at the margins of the vertebral endplates, to eventually form the ring apophysis. These appear at roughly the age of 6–9 years, by approximately age 12 they coalesce, and eventually fuse to the vertebral body at approximately the age of 14–21. Also, secondary ossification centers appear at the tips of the spinous, transverse, and mamillary processes around puberty, and fuse at approximately 25 years of age; F: although the ring apophysis does not contribute to growth, its fusion with the vertebral body at approximately age 14–21 years signals the end of longitudinal growth. This fusion gives bony attachment of the outer annulus fibrosis to the vertebral body. The inner annulus fibrosis remains attached to the vertebral endplate; G: in the butterfly vertebral deformity, the two chondrification centers remain separated, develop separate ossification centers, and hence form two separate anomalous hemivertebrae with a sagittal cleft; H: adjacent adult vertebrae elongate in the sagittal midline to compensate for the deficit in the butterfly vertebra. (Figure adapted from Ref. 46. Much information in the text is credited to Ref. 30, pp. 129–144.)

condition, there is no epidemiological data regarding the association of butterfly vertebra to LBP.

As a result of its appearance in various medical imaging modalities (CT, magnetic resonance image (MRI), or lateral x-ray views), it may initially be mistaken for metastatic disease or wedge compression fracture.^{3,7} See Fig. 2 for a MRI image depicting of a butterfly vertebra.

The butterfly vertebra is easily identified on an antero-posterior radiograph as two hemivertebrae separated by an empty space, with compensatory midline elongation of the adjacent vertebrae.⁷ See Figs. 3 and 4 for lateral lumbar and antero-posterior views of the butterfly vertebral defect described in this case.

Case description

Mr BV, a 21-year old male university student, presented to physical therapy a day after injuring

his low back by falling backwards and twisting to the left while playing touch rugby.

Mr BV's chief complaint was LBP. Combined forward-bending and left rotation was the most irritating and he reported restricted movement. The primary area of pain was located in a small area superomedially to the top of the left iliac crest (Fig. 5), rated 6 on a scale of 0–10. The secondary area of pain was in the left buttock and upper posterolateral thigh, to just below the left buttock crease.

Both areas of pain were described as deep, intermittent aches. At its worst, the primary pain area was significantly more intense than the secondary pain area. Symptoms were worst in the morning and would get better with movement during the day. No night symptoms were present and there was no disruption to his sleeping pattern. No other symptoms

Figure 2 Magnetic resonance image (MRI) of a butterfly vertebral defect. Reprinted from Sonel *et al.*, *Butterfly vertebra: a case report. Clinical Imaging*, 2001;25(3): 206–8, with permission from Elsevier.⁴⁷

were reported in the low back, buttocks, or legs. Mr BV had no paresthesia, anesthesia, cord, or cauda equina symptoms.

Mr BV had suffered from episodes of similar LBP prior to the current injury. The first episode resulted from a similar incident, playing touch rugby 1 year prior, and was resolved without health-professional intervention in approximately 24 hours. The second episode occurred 6 months later playing cricket (batting). A physical therapist advised Mr BV to perform hamstring stretching and abdominal muscle exercises following the second incident, which he reported had been helpful but had not totally resolved the symptoms. At the time of the current (third) episode, symptoms from the second incident had improved, but had not completely resolved.

Mr BV reported no systemic illnesses or surgeries in his medical history. He reported his general health to be very good, and a review of systems revealed no abnormalities. Up until this point he had undergone no medical imaging investigations. At consultation he agreed to participate in a diagnostic research study,¹¹ which was being conducted during the time he presented for therapy. As a participant in this research he was referred for flexion-extension lateral radiographs and side-bending antero-posterior radiographs. As these were non-clinical, non-urgent radiographs, the results of the imaging would not be available for approximately 4 weeks.

In a relaxed standing position, Mr BV had a decreased lumbar lordosis and left lateral trunk shift. From visual observation, there appeared to be a general loss of extensor muscle bulk on either side of the spinous processes throughout the lumbar spine, and the lower abdominal muscle region was inactive and protruding.

Figure 3 Antero-posterior radiograph of the lumbar spine (patient is side-bending maximally to the left). A butterfly vertebral defect involving L4 is present.

Mr BV described forward bending with left rotation as his most irritating and restricted movement, however, on examination he was reluctant to move his lumbar spine into forward bending, as he associated the pain and restriction experienced with this movement with further deterioration of his problem. With lumbar active range of motion testing, forward bending slightly increased his pain, however, throughout the maneuver, Mr BV maintained a lordotic lumbar spine, thus the majority of forward bending was occurring at the hips. On returning to the neutral position from forward bending, Mr BV maintained an extended lumbar position and felt pain throughout the movement. Active lumbar extension increased Mr BV's primary pain significantly, and was limited by approximately 50%. Lateral flexion to the left and right was limited by 25–50% but not associated with pain reproduction.

Figure 4 Lateral radiograph of the lumbar spine (patient is in full lumbar flexion). A butterfly vertebral defect involving L4 is present.

Neuromuscular control of the pelvic tilt movement in standing was visually judged to be poor, with an inability to differentiate the pelvis from gross trunk movement.¹² The single-leg standing extension test, an unvalidated test for isthmic spondylolysis,¹³ was positive bilaterally. Single-leg balance was mildly impaired bilaterally, with single limb stance on the right worse than the left (right balance impairment of 3/10 and left balance 2/10 on the Hauptstein scale^{†,14}).

[†] The Hauptstein scale is a 5-second assessment of the quality of single limb stance balance. High inter-rater reliability has been established (ICC_(2,1) 0.81) by physical therapists for visual judgements of steadiness on a 10 cm visual analog scale. The rating scale consists of the numbers between 0 and 10. Zero corresponds with the 'best' performance, and equates to excellent steadiness of the pelvis and trunk, with only minor adjustments of foot pronation/supination and leg rotation, no contact of the non-weight bearing limb with the support limb or the ground, and hands remaining on hips; 5 corresponds with moderate unsteadiness of the pelvis or trunk, with some contact of the non-weight-bearing limb to the support limb, or taking hands off the hips, but maintaining the ability to stay in single leg stance for the full 5 seconds; 10 corresponds with the 'poorest' performance. This equates to considerable unsteadiness of the pelvis or trunk, inability to maintain single leg stance for 3 seconds, and extensive arm waving.

Figure 5 Pain drawing completed by Mr BV. PA: primary pain area: ache, intermittent, and deep in quality; 6/10 at worst; PB: secondary pain area: dull, intermittent, and deep in quality. Only present concurrent with PA.

Palpatory pressure, as well as central, left, and right unilateral passive accessory motion (PAIVM) testing,^{15,16} found the L4 level the most painful.¹⁷ The pain produced was concordant with the presenting pain. Lumbar spinal levels L3 and L5 were also tender, but less so than L4. Passive physiological intervertebral motion (PPIVM) testing^{16,18} found hypomobility in forward bending, backward bending and left lateral flexion between levels L3 and S1. Right lateral flexion was considered hypomobile from levels L1–S1, in particular between L3 and S1.

The straight leg raise test¹⁸ was negative bilaterally, however, upon slump testing,¹⁵ both the primary and secondary areas of pain were reproduced with left knee extension to 45° below full extension. Right knee extension was full range and pain-free. Sensory examination was negative, and deep tendon reflexes undiminished.

Diagnosis

Acute LBP is typically episodic,¹⁹ and differential diagnosis for LBP without neurological signs includes mechanical or chemical insult to virtually any innervated structure in the region, principally including: the intervertebral disk; zygapophyseal joints; ligaments of the spine; muscles and their associated fascia; neural tissue and its associated sheaths; and the bony vertebral body, neural arch, and processes (transverse, spinous, and articular). Patho-anatomical diagnosis by clinical examination has proven extremely difficult.^{20,21} Stress fracture of the pars interarticularis must be considered, particularly in a young active person.^{22,23} Organic disease, such as

Table 1 Key impairments and interventions

	Interventions
Spinal movement impairments	
• Hypomobility of spinal segmental physiological motion, L3-S1	Non-thrust spinal manipulation, pelvic tilting, and 'slump' exercises
• Tenderness with accessory motion testing, primarily at L4	Potential neurophysiologic effects of non-thrust spinal manipulative
• Impaired mobility associated with the slump test	'Slump' exercises
Trunk movement impairments	
• Restricted lumbar forward-bending	Pelvic tilting and 'slump' exercises following non-thrust spinal manipulation
• Impaired neuromuscular control of trunk movement	Pelvic tilting exercises, progressing to core stability exercise utilizing a Swiss ball
• Impaired bulk and tone of lumbar paraspinal muscles	Therapeutic exercises targeting erector spinae and multifidus recruitment, and core stability exercise utilizing a Swiss ball
• Impaired tone and recruitment of abdominal muscles	Therapeutic exercises targeting abdominal muscle recruitment

disease of the gastrointestinal system, genitourinary system, and rheumatic or infectious disease of the musculoskeletal system, must also be included in the differential diagnosis as these may cause referral of pain mimicking mechanical low back disorders.²⁴⁻²⁶

As Mr BV's problem had been present for the previous 6-12 months and he demonstrated movement restriction and symptom reproduction with stretch and compressive movement patterns upon clinical testing, the evaluating therapist (RH) considered the patient's symptoms to be mechanical in origin. The PAIVM^{15,16} findings, indicating pain concordant with the presenting pain, and the passive PPIVM tests^{16,18} demonstrating hypomobility in forward bending, backward bending, and left lateral flexion between levels L3 and S1 supported this conclusion.^{17,27}

The primary hypothesis for this pattern of mechanical findings was soft tissue fibrotic changes and articular restriction at the L4/5 zygapophyseal joint. Lumbar segmental instability, typically characterized by episodic exacerbation and remission,^{16,28} was also hypothesized as a potential diagnosis for Mr BV. Poor postural body awareness and an inadequate recruitment of trunk muscles for trunk stability during normal functional tasks supported this diagnosis, but in the absence of hypermobility findings with PPIVM or PAIVM testing, we rejected that hypothesis in favor of a motor control impairment of the trunk musculature. The impaired motor control was considered a factor contributing to re-injury, although whether motor control impairment was resultant or causal in Mr BV's case is debatable. The key impairments are listed in Table 1.

A bulging or herniated disk was not considered, as the straight leg raise test is considered sensitive for the detection of lumbar disk integrity,²⁹ with a negative test assisting in ruling out this diagnosis. While it is possible that restriction of neural tissue mobility may have resulted from inflammation in the vicinity of the intervertebral canal or foramina over

the 6 months leading up to his presentation to the clinic, explaining the slump test findings, the slump test also involves tension of many types of tissue, and does not provide information that is specific to the neural tissue.^{30,31}

The clinical features of this case displayed clinical features of both a syndrome requiring mobilization, and a syndrome requiring stabilization in the form of motor control exercises.^{32,33} While there is ample expert opinion regarding the clinical features of these diagnoses, criterion-related validity has not been established for the clinical diagnostic features of either.^{11,12,16,20}

In summary, the list of differential diagnoses included (but was not limited to): stress fracture of the pars interarticularis; organic disease such as rheumatic diseases or space occupying lesion; bulging or herniated intervertebral disk; lumbar segmental instability; impaired motor control; and spinal joint hypomobility. Examination findings available at the time of the initial examination ruled out all but the latter two. Examination findings supported diagnosis of both spinal joint hypomobility and coexistent impaired motor control.

Treatment

Physical therapy goals for this patient were to achieve pain-free and unrestricted movement in the lumbar spine, which would enable him to partake in functional activities of daily life with minimal recurrences of back pain. To achieve these goals, the intervention plan was:

1. To improve mobility of implicated soft tissue structures (e.g., joint capsule, ligament, nerve, muscle, and tendon) with passive mobilization and slump mobilization exercises.
2. To improve lumbar active stability by increasing trunk strength, improving positional awareness, and improving neuromuscular control of lumbar motion, as well as static and dynamic postures.

The interventions utilized are outlined in Table 1. Therapeutic manipulation of the low lumbar spine consisted of a non-thrust lumbar rotation mobilization technique,¹⁵ which was performed in right side lying. Stretch into resistance was applied for approximately

5 minutes. While spinal manipulation is an effective intervention for LBP, as yet there is no clear evidence that any one specific technique is superior to another.^{34–37}

In the first visit, Mr BV was also instructed in ‘slump’ exercises^{30,31} with the aim of increasing mobility of the nerve roots and associated structures at the lumbar intervertebral foraminae and distally. Adhesions of the normally mobile dura have been reported,³⁸ and are believed to be commonly associated with LBP.

To conclude visit one, the patient was instructed in pelvic tilting exercises in an anterior and posterior direction, to optimize positional awareness and control of lumbo-pelvic motion. This preceded exercises intending to recruit multifidus, erector spinae, and the abdominal muscles,³⁹ added progressively in subsequent visits, which have been shown to be efficacious for low back disorders in a range of clinical circumstances,^{40,41} including reducing rate of recurrence.⁴⁰ While the transversus abdominis and multifidus have been championed as the key component to lumbo-pelvic exercise regimens, experimental evidence suggests that all muscles of the region contribute to lumbar stability under various loads.⁴² Prior to the patient education, provided by the lead author progressively from visit two, Mr BV had no awareness of the important role these muscles play in the stabilization of the spine, particularly during athletic activity.

Results of Treatment

Treatment consisted of eight therapy sessions. Mr BV’s signs and symptoms responded positively to treatment within one session. Immediately following mobilization to the lumbar spine, pain experienced with active lumbar flexion and extension was reduced. Upon slump testing, pain previously reproduced at -45° degrees knee extension was now reproduced at -10° knee extension.

With postural correction and education on movement control during lumbar flexion and extension, an immediate improvement in range was noted, which was maintained over subsequent treatments. This was particularly evident in lumbar forward bending, a movement the patient had previously been avoiding.

Mr BV continued to improve following each of the next three treatment sessions. At that point, he was having minimal symptoms ‘on the odd day’ and no pain on other days. It was at this point that we received the report from the radiologist, informing us of the presence of a butterfly vertebral defect at the L4 level and associated narrowing of the intervertebral disk spaces at the L3/4 and L4/5 levels.

As the treating therapist was unfamiliar with this rare vertebral anomaly, mobilization was discontinued. Treatment continued with the emphasis on

improving active lumbar stability and dynamic and static movement control utilizing core stability exercises with and without a Swiss ball, and via the reintroduction of dynamic exercise, such as running, into the Mr BV’s fitness regime.

The patient’s symptoms and signs changed little following this point in time, and the patient was discharged after a total of eight sessions over a 5-week period. At this point Mr BV was still experiencing twinges of LBP approximately once or twice every 2–4 days, but was happy to self-manage with his rehabilitation program. The patient was informed of the nature of his vertebral anomaly and the possibility that this may predispose him to further episodes of LBP in the future.

Discussion

It is an interesting observation that spinal mobilization appeared to be useful in this case. Had the treating therapist known of this patient’s vertebral anomaly prior to treatment, it is likely that she would not have chosen mobilization as a treatment, as a precaution against structural instability not identified during physical examination.

In hindsight, the information provided by the radiographs was inconsequential, and possibly disruptive, to the therapeutic intervention based upon impairments found during clinical examination, i.e. segmental hypomobility and deficient neuromuscular control. It is well known that radiographs are not recommended for the routine management of patients with LBP, in the absence of ‘red flags’ for serious disease, even if it has persisted beyond 6 weeks.⁴³ While the radiologist’s report concluded that the spine did not appear to be grossly unstable in the flexion–extension radiographs, the authors suspected that, due to the shape of the vertebrae involved in the defect, this region of the spine was likely to be inherently less stable,⁴⁴ therefore requiring greater neuro-muscular control to enhance stability.⁴⁵ The radiological findings and the physical therapist’s decision to change the treatment plan are likely to have influenced the patient’s beliefs about the cause, nature, and prognosis of his LBP, however the extent of this cannot be quantified. At discharge, the lead therapist’s advice to this young man was to maintain the strength and endurance of his trunk muscles to encourage active lumbar stability and postural control during functional tasks. Follow-up at 12 months revealed a zero (0) score on the modified somatic perceptions questionnaire, indicating no residual clinically significant psychological distress. No assessment of kinesiophobia was conducted. His score of 2 points on the 18-item modified Roland Morris disability scale was also very low, indicating almost no LBP related disability at 1-year follow-up.

On the basis of this case, we cannot infer that the treatment provided caused the outcomes reported, however we can report that the manual therapy provided did not result in any adverse effects. We cannot comment more specifically with regard to other forms of manual therapy, such as thrust manipulation. A review of the literature does not suggest that the presence of butterfly vertebrae should contraindicate judicious use of manual therapy. There has been one report of symptomatic disk herniation at a butterfly vertebra,⁹ therefore this should be kept in mind as a potential diagnosis or adverse event.

Conclusions

While it is prudent to consider congenital bony defect of the spine to be a relative contraindication to spinal manipulation, this case may support the use of spinal mobilization in the presence of a butterfly vertebral defect, with due caution. Owing to the rarity of this vertebral anomaly, research studying specific interventions is impractical. Further research to establish the efficacy of neuromuscular training, with or without spinal mobilization, in the subgroup patients with LBP featuring segmental hypomobility, impaired trunk movement, and radiographic disk space narrowing, may however be generalizable to patients with butterfly vertebrae.

Conflict of Interest

The authors declare they have no conflicts of interest.

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