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Uncomplicated mechanically induced pelvic pain and organic dysfunction in low back pain patients

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Mechanical disorders of the lumbar spine have been given much attention in the literature. Short of an acute cauda equina syndrome, few reports exist detailing the findings and clinical course of patients with pelvic pain and disorders of bladder, bowel and gynecologic/sexual function of spinal origin. Two uncomplicated representative cases of mechanically induced pelvic pain and organic dysfunction (PPOD) in patients presenting with low back pain are detailed. These patients typically reveal a wide range of individual symptoms and demonstrate clinical features characteristic of a mechanical disorder of the lumbar spine as the cause of their PPOD. The clinical features of the mechanically induced PPOD syndrome are reviewed and the response to distractive decompressive manipulation of the lumbar spine is presented.
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KEY WORDS: chiropractic, low back pain, pelvic pain, parasympathetic nervous system, cauda equina, bladder dysfunction, bowel dysfunction, gynecologic dysfunction, manipulation.

Les troubles mécaniques de la colonne lombaire ont fait l'objet de nombreux écrits. Mais à l'exception du syndrome aigu de la queue de cheval, il existe peu de rapports détaillant les résultats et cheminements cliniques de patients souffrant de douleurs pelviennes et de troubles de la vessie, de l'intestin et de la fonction gynécologique/sexuelle d'origine spinale. Deux cas représentatifs simples de douleurs pelviennes et de dysfonction organique (DPDO) provoquées mécaniquement chez des patients présentant des douleurs lombaires sont exposés en détail. De façon typique, ces patients présentent un assortiment varié de symptômes individuels et sont la preuve que les caractéristiques cliniques typiques d'un trouble mécanique de la colonne lombaire constituent la cause de leurs DPDO. Les caractéristiques cliniques du syndrome de DPDO provoqué mécaniquement sont examinées et la réponse à la manipulation distractive et décompressive de la colonne lombaire est présentée.
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MOTS-CLÉS : chiropratique, douleurs lombaires, douleurs pelviennes, système nerveux parasympathique, queue de cheval, dysfonction de la vessie, dysfonction de l'intestin, dysfonction gynécologique, manipulation.

Introduction

Since its inception chiropractic theory has supported the concept that mechanical disorders of spinal origin could give rise to organic dysfunction or disease.[1,2] Despite the fact that the Chiropractic profession was originally founded upon the resolution of a visceral disorder by manipulation there is little scientific evidence available supporting this pathomechanical relationship.[1,3] Most reports addressing mechanical disorders of the lumbar spine continue to focus on the signs and symptoms of lower lumbar or upper sacral nerve root irritation or compression resulting in the typical low back and leg pain syndromes.

While it is known that severe cauda equina compression can result in gross disturbances of bladder and bowel function little attention has been given to the signs and symptoms of lower sacral nerve root involvement in which neurological function is only partially disturbed.[4] Like the acute cauda equina syndrome, mechanically induced pelvic pain and organic dysfunction (PPOD) encompasses disorders of bladder, bowel, gynecologic and sexual function. However, unlike the acute cauda syndrome in which symptoms reflect a severe compressive radiculopathy with rather consistent functional loss, the mechanically induced PPOD syndrome is quite varied in its presentation. Symptoms which have been attributable to lower sacral nerve root irritation or compression include pelvic pain (inguinal or suprapubic); miscarriage; vaginal spotting; vaginal discharge (leukorrhea); menstrual migraine; urinary frequency, urgency, dribbling, incontinence, sluggishness, retention, nocturia, dysuria, infection, loss of ability to perceive vesical filling, prostatovesiculitis; impotence; decreased genital sensitivity; anorgasmy; dyspareunia; deficient pre-coital lubrication; pelvic pain during orgasm; constipation; diarrhea; excessive

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flatus; anal sphincter spasm; encopresis; spontaneous bowel discharge; proctalgia and loss of ability to perceive rectal filling.[5–16]

Although most reports of patients with mechanically induced PPOD reveal a history of low back and/or leg pain, cases of mechanically induced PPOD in the "asymptomatic" low back patient have also been reported[7,17] Women are frequently more affected than men.[5,6] Many of these patients had previously sought and underwent treatment for their pelvic symptoms even though no specific etiology could be identified. Symptomatic treatment ranged from various medical to surgical procedures without significant or lasting improvement. However, subsequent evaluation of the patient's lower back revealed clinical evidence of mechanical insult of one or more of the lower sacral nerve roots. Furthermore, chiropractic treatment directed at correcting the lumbar spine disorder, theorized to have a decompressive effect,[18] resolved not only the patient's low back and leg pain symptoms but also the various PPOD symptoms, many of which had been long standing and of a recalcitrant nature.

This report will outline the typical clinical features and treatment of mechanically induced PPOD. Two cases are presented to illustrate the clinical features and response to therapy.

Case reports

Case 1 – A 29-year-old female presented with low back and leg pain of about one year in duration resulting from a lifting injury. At that time pain and paresthesias had extended down the right leg into the foot and toes. Initial medical evaluation recommended she have spinal surgery. Desiring another option, she sought chiropractic treatment at another office. Spinal manipulation performed, as described by the patient, in a prone position provided some relief of her low back and leg symptoms however, her leg pain did not completely resolve. Over the next few months her low back and right leg pain increased in intensity. Approximately one month prior to presentation, she re-aggravated her low back and right leg pain while lifting at work. The right leg pain extended along the posterolateral thigh and calf to the foot. She also experienced the onset of intermittent left leg pain and paresthesias radiating down the posterior thigh to the knee. Within hours of this re-aggravation she experienced the onset of bilateral inguinal pain (dominant on the right); constant sharp rectal pain; and urinary disturbances consisting of frequency, urgency, dribbling and intermittent stress incontinence without pain. Upon more detailed questioning she admitted that during the month following her re-aggravation she became aware of diminished genital sensitivity so that orgasm occurred less frequently and was of a diminished intensity. Accompanying her loss of genital sensitivity was a loss of libido. Further, dyspareunia and vaginal discharge (leukorrhea) had also begun to occur during this time. She stated that there was no accompanying bowel or menstrual dysfunction. Approximately one week prior to being seen, she had consulted her gynecologist for her pelvic symptoms. A complete gynecologic and pelvic examination however, failed to reveal any abnormal findings accountable for her symptoms. As a result, she was asked to return if her symptoms should increase. There was no prior history of urologic or gynecologic disturbances.

Clinical examination of her low back revealed a left thoracolumbar antalgia with inability to assume an erect posture. Active thoracolumbar range of motion was limited in right lateral bending and extension with low back pain being intensified in flexion, extension and right lateral bending. Paraspinal palpation provoked significant muscular spasm and pain over the mid and lower lumbar regions bilaterally, being most intense at the lumbosacral junction. Kemp's manoeuver was positive bilaterally, producing sharp low back pain. Toe and heel walking were performed with no observable evidence of muscular paresis. Active bilateral straight leg raise (SLR) and lumbar spine flexion produced low back and sharp rectal pain. Inguinal and suprapubic regions were painful to palpation. Right SLR produced ipsilateral posterolateral thigh pain at 60° and was intensified by dorsiflexion of the foot. The enhanced SLR (SLR peformed while simultaneosuly applying pressure over the pubic and inguinal areas[4]) produced right inguinal pain at 85°. The left SLR produced ipsilateral posterolateral thigh pain at 80°. Sensory evaluation revealed areas of hypesthesia to pinwheel and cotton, especially along the entire S1 dermatome; while the lower sacral sensory loss was detected over the gluteal region with the order of dominance being S2 and S3. Resistive strength testing of the lower extremities was unremarkable. The lower extremity deep reflexes were 2+ and equal bilaterally, with no evidence of long tract involvement. The anal reflex was intact bilaterally and sphincter tone was good.

Routine weight bearing lumbar spine x-rays revealed a left sided spinal antalgia. There was no evidence of osseous fracture, pathology, dislocation or congenital malformation. A dipstick urinalysis was within normal limits.

Clinical impression was that of an L5 disc lesion with a right sciatic sensory radiculopathy involving the right S1 nerve root and the S2 and S3 nerve roots bilaterally with secondary PPOD. She was treated daily with distractive decompressive manipulation of the lower lumbar spine in accordance with the basic guidelines as set forth by Cox,[18] followed by cryotherapy for 15–20 minutes. She was fitted with a semi-rigid lumbosacral appliance initially worn 24 hours per day. Pelvic tilt and knee to chest lumbar spine exercises were prescribed three times per day, followed by the application of ice for 30 minutes and at three hour intervals. She was instructed to remain non-weight bearing. Treatment following this protocol provided progressive relief of her complaints.

Within the first few days of treatment however, she became aware of sharp genital pain and paresthesias radiating to the clitoris, and described these sensations as being aggravated by touch or contact of any type. There was no history of local trauma or infection involving the genital region. Following one week of care, bladder dysfunction (frequency, urgency, dribbling and stress incontinence) had completely resolved. Ingui-

nodynia, dyspareunia and proctalgia had significantly improved. Menstruation at this time was more painful than usual although no abnormalities in flow or duration occurred. Weight bearing could be tolerated for only short periods without provoking back, leg and pelvic pain symptoms. After one month of care all PPOD sympytoms (pelvic pain, proctalgia, leukorrhea, depressed libido and anorgasmy) had resolved, and she was returned to full weight bearing activity with limitations on sitting, bending, lifting and twisting. Bladder function remained stable and genital pain and paresthesias had normalized. Intermittent back, leg and pelvic symptoms could be provoked by excessive weight bearing activity and prolonged sitting. She was discharged from care, fully recovered after two and one-half months of treatment.

As a result of a subsequent fall, she had a recurrence of her low back complaints with a simultaneous return of some of her PPOD symptoms. Clinical evaluation revealed a recurrent discogenic low back disorder with lower sacral nerve root involvement with secondary PPOD. Treatment, following the same protocol as previously outlined resolved all of her complaints in a similar manner.

Case 2 – A 40-year-old female presented with a complaint of low back and leg pain of about three weeks duration resulting from a fall on the buttocks. At the time of her injury pain was immediately felt in the low back and left gluteal region. Over the next few hours she experienced radiating pain down the right and left antero-medial thigh and postero-medial leg. During this same period of time, she also noticed bilateral inguinal pain, being dominant on the right. Concurrently she became aware of the onset of bladder dysfunction, consisting of urinary frequency, urgency, difficulty initiating micturition and sluggish voiding requiring straining. There was no associated dysuria. She stated that there was no bowel dysfunction, genital pain or paresthesias. At this time, she consulted another chiropractor, who according to the patient, manipulated her spine in a prone position. She discontinued therapy after a few visits due to the lack of improvement in her back, leg and pelvic symptoms. She subsequently presented to this office. During this period of time no additional symptoms had developed, however, her pelvic pain and urological disturbances had worsened. A review of her past history revealed a hysterectomy years earlier for bleeding irregularities however, no other prior history of urologic, colorectal, gynecologic or sexual dysfunction could be obtained.

Clinical evaluation of her low back disorder revealed a left sided thoracolumbar antalgia. Deep digital palpation of the lower lumbar paraspinal musculature revealed local pain and muscular spasm bilaterally. Deep palpation of the lumbosacral junction produced pronounced local pain with intermittent radiation into the right inguinal region. Active thoracolumbar range of motion was significantly decreased in all directions with lumbosacral and right inguinal pain being provoked during attempts at flexion and extension. Kemp's manoeuvre produced lumbosacral, right gluteal and right inguinal pain bilaterally, however, when performed to the right, right leg pain and paresthesias was noted. Bechterrew's manoeuvre produced low back, right inguinal and bilateral medial thigh and calf pain and paresthesias which were increased in their intensity with the addition of cervical spine flexion and the performance of the Valsalva manoeuvre. Toe and heel walking were performed without obvious evidence of muscular paresis. Palpation in the inguinal region produced pain bilaterally, especially on the right. There was no suprapubic tenderness. Straight leg raise on the right produced low back and right inguinal pain at 30°. At 40°, right SLR produced right medial thigh and calf pain which was further intensified with the addition of foot dorsiflexion. Left SLR at 45° produced ipsilateral medial plantar foot and right inguinal pain. Resistive strength testing of the lower extremity musculature was unremarkable. The lower extremity deep reflexes were 2+ and equal bilaterally and no evidence of long tract involvement. Anal sphincter tone was good and the superficial anal reflex was intact bilaterally. Cutaneous sensory evaluation of the lower extremities with pinwheel and cotton revealed areas of hyperesthesia within the boundaries of the right L5 through S3 dermatomes. Lower sacral sensory alteration was detected over the gluteal musculature and the order of dominance of sacral nerve root involvement was S3, S2 and S1.

Routine weight bearing lumbar spine radiographs revealed no evidence of fracture, pathology, dislocation or osseous congenital malformation. A dipstick urinalysis revealed no evidence of abnormality.

A clinical impression of an acute right medial L5 annular protrusion with a right sciatic sensory radiculapathy, involving the L5–S3 nerve roots with secondary PPOD was made. She was treated with distractive decompressive manipulation of the lower lumbar spine on a daily basis following the protocol outlined in case 1. Cryotherapy was applied to the low back for 15–20 minutes following each application of distraction. She was fitted with a lumbosacral appliance to be worn initially 24 hours per day and asked to remain non-weight bearing until further notice. Pelvic tilt and knee to chin exercisess were prescribed three times per day followed by ice applications to the low back for 30 minutes every 2–3 hours. Early during the course of treatment right sided genital pain and paresthesias had their onset and were described by the patient as a painful hypersensitivity involving ther right labial region and extending into the clitoris making contact or touch painful. There was no history of injury, infection or inflammation in this region. Her response to treatment was progressive. Following three weeks of care her low back, leg and pelvic pain symptoms had significantly improved. Genital pain and paresthesias had resolved, and concurrently, urinary frequency, urgency, difficulty and sluggishness had normalized. At this time she was ambulated and instructed to avoid prolonged sitting, bending, lifting and twisting.

Prior to being discharged from care, she re-aggravated her low back and right medial thigh pain and paresthesias subsequent to lifting. During this same period of time, she also

experienced the return of bilateral inguinal pain, intermittent sharp clitoral pain and urinary dysfunction. Within a few days she experienced the onset of bowel dysfunction, consisting of constipation and a loss of the normal urge to defecate. There had been no dietary changes that could account for her bowel functional disturbances. In addition to her genital pain and paresthesias she became aware of diminished genital sensitivity, such that orgasm was of a decreased intensity and could only be achieved after extended periods of coitus. There was no associated deep dyspareunia. Clinical assessment revealed a recurrent L5 disc lesion with lower sacral nerve root involvement and secondary pelvic pain; bladder, bowel and sexual dysfunction as outlined above. Treatment following the above protocol resulted in progressive improvement of all her pain syndromes and pelvic organic dysfunction so that eight weeks following her re-aggravation, all her symptoms had resolved.

Discussion

The acute cauda equina syndrome is a well-known although rare phenomenon in clinical practice.[4,19] This syndrome, characterized by bilateral paresis or paralysis of the lower extremity musculature; wide spread and bilateral lower extremity, gluteal and perineal sensory loss; and loss of bladder and bowel function requires immediate decompressive surgery.[20] On the other hand, mechanically induced PPOD of spinal origin in which lower sacral nerve function is only partially disturbed has a completely different character.[4] This syndrome, often times mis-diagnosed as a local pelvic disorder,[4] is frequently non-responsive to symptomatic medical or surgical treatment.[6,21] Current cases and earlier reports[5,17,21,22] demonstrate that when manipulative decompressive treatments are directed at the lower lumbar spine in patients with symptoms of bladder, bowel, gynecologic and sexual dysfunction, in whom clinical evidence of lower sacral nerve root or compression can be identified, the associated symptoms usually resolve. The implication is that the reported sensory dysfunctions most likely involve spinal nerve roots serving as the origin of the pudendal and pelvic splanchnic nerves. These two nerves provide both a somatic and autonomic influence respectively, and are extensively distributed to structures located throughout the pelvis.[23] Their roots of origin (S2, S3, S4) however, are susceptible to mechanical insult consequent to degenerative changes of the lower lumbar spine.[24]

The cauda equina roots, however, lack significant epineurium and perinerium,[25] and the absence of these structural components, typical of peripheral nerves, may play a role in their susceptibility to various types of mechanical insult. In fact, it has been suggested that this anatomical difference between nerve roots and peripheral nerves may actually cause spinal nerve roots to react differently to compression than peripheral nerves.[26,27]

Various mechanisms of nerve root insult have been assessed. Compression for example, has been found to alter nerve root function by several means. Experimental studies on porcine cauda equina roots have demonstrated that alterations in intraneural perfusion may be induced at very low levels of compression (5–10 mm Hg).[28] Sustained vascular compromise leads to increased vessel permeability,[29] with resultant intraneural edema formation,[29] and possibly affecting impulse conduction.[25] Direct pressure of only 10 mm Hg against dorsal roots for as little as 30 minutes has been found to reduce the compound action potential of the conducting root by 50 percent.[27] The functional change brought on by compression was thought to be due to mechanical deformation of the involved root fibers.[27] A compressive pressure of 30 mm Hg, however, has been found to block axoplasmic transport,[28,29] while acute compression at levels of 50–75 mm Hg pressure causes significant alteration in impulse conduction.[30]

Although compression at different levels has been found to alter nerve root function in different ways, nerve root tension can also induce a sequence of tissue reactions leading to intraneural inflammation.[25] These tissue reactions lead to secondary functional alteration and subsequent hyperexcitability of nerve root tissue or loss of nerve function.[31] Such altered states of neurological function could be reported as pain or paresthesias and sensory deficit or muscular paresis respectively,[31] and may in fact be occurring at the same time.[25] Clinically, this dichotomous situation might account for the commonly observed phenomenon of seemingly conflicting states of neurological dysfunction represented by various combinations of paradoxical symptoms, i.e. genital pain or paresthesias and anorgasmy; or urinary frequency or urgency and sluggishness or incontinence.

The exact physiological changes involved in the production and resolution of mechanically induced PPOD are open to speculation. It does appear that mechanical disturbances of the lower lumbar or lumbosacral spine may be central to this issue. While the author has not had the opportunity to confirm or rule out lumbar disc involvement through the use of sophisticated imaging modalities as a probable etiology in the production of mechanically induced PPOD, clinical examination has consistently revealed evidence of a discogenic low back disorder as the likely cause of the patient's complaints. With rare exception, most PPOD patients do not reveal frank evidence of a radiculopathy. Rather they present with a history of back and/or leg and/or pelvic symptoms which can be provoked during physical examination by various orthopedic stress tests and manoeuvres that challenge the lumbar spine in a manner consistent with provoking symptoms of discal origin i.e.; Valsalva's, Kemp's, straight leg raise, etc. Additionally, most PPOD patients do reveal evidence of a sensory radiculopathy involving one or more of the lower sacral nerve roots. It is possible that the functional significance of a sensory radiculopathy involving the lower sacral nerve roots may be greater than one involving the lower lumbar or upper sacral (S1) roots. A sensory deficit involving the latter may result in pain or paresthesias, while one involving the lower sacral nerve roots may have the added effect of disturbing normal bladder, bowel and sexual function as a result of altering sensory input necessary to maintain an intact

TABLE 1 SYMPTOMS OF PPOD

	Pelvic pain	Bladder dysfunction	Bowel dysfunction	Gyn/Sex dysfunction
Case 1	R/L inguinal pain rectal pain dyspareunia	Frequency, urgency, dribbling, stress incontinence		Decreased gentital sensitivity, anorgasmy, depressed libido, leukorrhea
Case 2	R/L inguinal pain	Frequency, urgency, difficulty, sluggishness	Constipation, loss of rectal filling sensory perception	Genital pain and paresthesias, decreased genital sensitivity, diminished orgasm intensity

TABLE 2 CLINICAL SIGNS OF LOWER SACRAL NERVE ROOT COMPRESSION

	Case 1	Case 2
Lower sacral dermatomal alteration	Right S2 and S3 Hypoesthesia	Right S2 and S3 Hyperesthesia
Lower sacral somatic palpatory hyperpathia	Bilateral inguinal and suprapubic palpatory hyperpathia	Bilateral inguinal palpatory hyperpathia
Induced pelvic pain on straight leg raise	Right inguinal pain on ipsilateral SLR at 85° (enhanced form)	Right inguinal pain on ipsilateral SLR at 30° (primary form); Right inguinal pain on contralateral SLR at 45° (primary form)
Reflex alteration	None	None
Motor alteration	None	None

TABLE 3 DIFFERENTIAL DIAGNOSIS OF MECHANICALLY INDUCED VS INTRA-ABDOMINAL PPOD

	MIPPOD[1]	IAPPOD[2]
Onset or aggravation of PPOD associated with mechanical stress to lumbar spine	Often	Infrequent
PPOD associated with low back and/or leg pain/paresthesias	Usually	Occasionally
PPOD symptoms	Usually of wide variety and often involve multiple pelvic organs	Less numerous and more specific to organic pathology
Pelvic pain aggravated by stress provocation of the lumbar spine	Yes	No
Pelvic pain induced on straight leg raise	Yes	No
Sensory alteration in lower sacral nerve root dermatomes	Yes	No
Palpatory hyperpathia over somatic regions corresponding to the S2 and/or S3 nerve roots	Yes	Occasionally
Confirmation of nerve root involvement by pain provocation examination[22]	Yes	No
Fever and/or abdominal rigidity and/or rebound pain	No	Maybe
Laboratory and urine findings	Negative or non-specific	Dependent upon pathology

[1] MIPPOD = mechanically induced PPOD
[2] IAPPOD = intra-abdominal PPOD

micturition, defecation and sexual reflex arc.[32] As nerve roots have been shown to be susceptible to low levels of compression and tension, it may be that discogenic low back disorders in their early stages of development (i.e.; bulging or protruding disc without obvious nerve root impingement) may provide sufficient mechanical insult to alter the sensory and sensory dependent reflex action of the lower sacral nerve roots. This hypothesis is consistent with this author's previous experience in identifying and successfully resolving mechanically induced PPOD in patients who had prior CT or MRI scans revealing "diffuse posterior annular bulging" at one or more levels of the lower lumbar spine.[21]

The identification of patients with PPOD most likely to benefit from manipulative therapy of the lumbar spine is a three step process. The first consideration being the establishment of the presence of pelvic symptoms characteric of lower sacral nerve root irritation or compression. This at times may be very difficult. It has been the author's experience, as it has of others,[33] that unless the patient is guided symptom by symptom through the individual possible disorders, they commonly avert questioning into the possible existence of bladder, bowel, gynecologic or sexual dysfunction.[4] It seems that these patients, being primarily concerned with their presenting complaint, usually of low back and leg pain can become indifferent to questioning in seemingly unrelated areas. Often times the mechanically induced PPOD patient will exhibit bilateral lower extremity pain symptoms attributable to the low back,[4] although these symptoms may not be symmetrical in their distribution. Table 1 lists the individual symptoms of lower sacral nerve root compression identified in these patients. Once the presence of symptoms characteristic of lower sacral nerve root irritation or compression has been established, it is necessary to try and identify clinical evidence of lower sacral nerve root involvement.

It has been previously reported that the most reliable clinical signs are of a sensory nature.[4] These include identifying sensory alteration within the boundaries of the lower sacral nerve root dermatomes; provoking pain upon deep palpation over somatic regions corresponding to the S2 and S3 nerve roots; and the induction of pelvic pain on straight leg raise, in either it's primary or enhanced form. Table 2 outlines the clinical signs of lower sacral nerve root compression found in these two cases.

The last consideration in identifying PPOD of mechanical origin is to rule out local pelvic causes of pain or organic dysfunction. Although it is not the intent of this paper to cover in detail the myriad of intrapelvic causes of pelvic pain and/or pelvic organic dysfunction, it should be obvious that these disorders must be considered in the differential diagnosis of PPOD. Where a suspicion of intra-abdominal or intrapelvic pathology exists, various physical manoeuvres have been described to aid in the differentiation on intra and extra abdominal or pelvic causes of pain.[34,35] In addition, pain syndromes of intra-abdominal or pelvic origin generally are not reproducable or aggravated by mechanical orthopedic stress provocation of the lumbar spine and, do not show characteristic neurological involvement typical of PPOD of spinal origin.[22] Table 3 contrasts clinical features of mechanically induced PPOD and intra-abdominal PPOD.

When mechanically induced PPOD is of recent onset, it's identification is usually not difficult. As with the cases presented above, these patients frequently recall the onset of pelvic symptoms occurring close in time to some type of mechanically stressful event to the lumbar spine. By contrast, in the chronic mechanically induced PPOD patient, these same findings may at times be more difficult to detect. Many of these patients have a long history of recurrent low back pain making the relationship between the onset of PPOD and a mechanical insult to the low back more difficult to establish.[4] This is especially so if individual symptoms of PPOD have developed at various stages over a long period. To further complicate this issue, patients with long standing symptoms of PPOD have usually undergone previous (sometimes numerous) evaluations and attempts at treatment by various types of specialists, who directed efforts at what was thought to be a local pelvic disorder.[21] A prior history of pelvic surgery, especially if multiple, which have failed to satisfactorily resolve symptoms characteristic of lower sacral nerve root compression may, indicate mechanically induced PPOD of spinal origin. Many patients with long standing mechanically induced PPOD have undergone numerous attempts at treatment with only minimal, short lived or no real improvement.[6,21] These patients commonly undergo surgical procedures that include laparoscopy, hysterectomy or hernioplasty for pelvic pain; suprapubic urethrovesical suspension for urinary incontinence and a coccygectomy for coccygeal or para-anal pain.

The two cases reported herein had been successfully managed by manipulative methods. It should be pointed out that like other disorders of spinal origin the mechanically induced PPOD syndrome does carry the potential of deterioration, the most serious complication being an acute cauda equina syndrome. The management of these cases requires careful ongoing assessment of the patient's response thereby allowing the clinician the earliest opportunity to identify any sign of regression. It has been this author's experience that initial treatment of the lower lumbar spine by distractive decompressive manipulation in patients with clinical evidence of mechanically induced PPOD is often followed by an aggravation of the patient's PPOD symptomatology. This usually is manifested by an increase in the intensity or severity of the individual PPOD symptoms or, by the onset of additional PPOD symptoms characteristic of lower sacral nerve root irritation or compression. In the two cases presented, this phenomenon is illustrated by the onset of genital pain and paresthesias following the onset of treatment. In most cases however, this post treatment "irritation" only lasts for the first few treatments or occasionally 1–2 weeks. Like the acute cauda equina syndrome, the mechanically induced PPOD syndrome demonstrating progressive deterioration of bladder and bowel function despite appropriate conservative intervention similarily neccessitates surgical referral.

Summary

Mechanically induced PPOD syndrome can be quite diverse in it's presentation. It's symptomatic presentation falls into four basic areas; pelvic pain and various disturbances of bladder, bowel and gynecologic/sexual function. Two uncomplicated representative cases of the mechanically induced PPOD syndrome were presented. In this author's experience, mechanically induced PPOD is usually identified as a co-existent finding in patients presenting with a low back related disorder, most commonly in the female patient presenting with bilateral lower extremity pain symptoms attributable to the low back. Familiarity with the individual symptoms representing lower sacral nerve root irritation or compression is essential in clinically recognizing patients with this disorder. Once symptoms representative of mechanically induced PPOD have been identified, clinical evaluation should include efforts at trying to identify signs of lower sacral nerve root involvement, as well as ruling out the possibilty of intra-abdominal causes of PPOD. As demonstrated by the two cases presented, and based on this author's previous experience, distractive decompressive manipulation of the lower lumbar spine has been found to be effective in resolving the symptoms of PPOD in individuals with clinical evidence of lower sacral nerve root irritation or compression as a result of a mechanical disorder of the low back.

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