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Case Report

S1 Dorsal Root Ganglion And Inferior Hypogastric Plexus Pulsed Radiofrequency Neuromodulation May Improve Type III Coccydynia Pain: a Case Report

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Abstract

Introduction : Coccygeal pain occurs in 1% to 2.7% of people without a clear coccygeal origin, unless provoked by prolonged sitting or anatomical changes found incidentally during surgery. Pain transmission blocked by pulsed radiofrequency (PRF) neuromodulation of the inferior hypogastric plexus (IHP) and dorsal root ganglion (DRG) can improve both nociceptive and neuropathic pain.
Methods : A 16-year-old female with a one-month history of coccydynia, coccygeal numbness radiating to the heels, and normal motor, micturition and voiding functions. The CSK 15 needle was inserted through both S1 neuroforamens to reach the DRG, then pulsed radiofrequency (PRF) 42°C for 2 minutes using the Cosman G4 device, followed by triamcinolone 20mg 1mL also administered contralaterally. Thus, PRF was also performed on both sides of the IHP anterior to the sacrum through the S2 neuroforamen approach.
Results : Improvement was observed after one month (NRS 0-1), whereas coccygeal numbness improved gradually. The combination analgesic (paracetamol 300mg, diazepam 2mg, diclofenac sodium 12.5mg) or pregabalin 50mg is administered as required, while vitamin B12 50 mcg/ 12h is continued.
Discussion : The pelvic sympathetic trunk (PSN) lies extraperitoneally anterior to the sacral and anteromedial to the anterior sacral neuroforamen with four or five interconnected ganglia. It rise to the lumbar sympathetic trunk (above) and the impar ganglion (below), which involved in transmitting sympathetic nociceptive from the perineum, distal rectum, distal vagina, distal urethra and anus. The parasympathetic afferent cells within the S2-S4 DRGs associated with pelvic splanchnic and somatic nerves.
Conclusion : Neuromodulation treatment for coccydynia has good results in DRG and IHP.

Keywords : coccydynia, pain, nociceptive, neuropathic, dorsal root ganglion, inferior hypogastric

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INTRODUCTION

The clinical complaint of coccygeal pain is commonly known as "coccydynia" or tail bone pain and was first described by Simpson in 1858. The pain is felt in the coccyx, buttocks and sometimes in the lower back. About 1% to 2.7% of people complain of coccygeal pain during theuraptic treatments, but few of them fail to identify the site of the lesion from the coccyx. Neurological examinations are not clear enough to describe these disorders, unless the worsening pain is mainly aggravated by prolonged sitting. Meanwhile, the anatomical changes can be seen incidentally during surgery under C-arm fluoroscopy. In addition, there are few important underlying conditions to rule out, such as soft tissue abscess or osteomyelitis, both of which are malignant and serious problems. Direct vertical trauma to the coccyx can result in a variety of outcomes, ranging from contusion to fracture-dislocation. Excessive flexion or extension of the coccyx also plays a role in increasing pain.[1] The coccyx is the terminal segment of the spine and gets its name from the Greek word for 'cuckoo bird's beak'. Consisting of three to five fused segments, the first of these is known as the coccygeal cornua and articulates with the sacral cornua. The lower part of the filum terminale and the coccygeal ligament are attached to this segment. A clinical examination may reveal tenderness or pain around the coccyx, which could indicate a fracture, inflammation or infection. If there is no tenderness, the pain may originate in the pelvis or lower back. The examination may also reveal erythema, swelling, a rash, discharge, fistulas, or skin breakdown, which could suggest cellulitis or osteomyelitis. The coccyx has several important functions, including supporting the anus.[2] It can classified as 6 types. *Type I:* gentle ventral curvature with caudally pointed apex of the coccyx (> 50%); *Type II:* more prominent of ventral curvature with apex pointing anteriorly (8–32%); *Type III:* acute anterior angulation without subluxation (4–16%); *Type IV:* subluxation at sacro-coccygeal or inter-coccygeal Joint (1–9%); *Type V:* retroverted with posteriorly angulated apex (1–11%); *Type VI:* scoliotic or laterally subluxated coccyx (1–6%).[3]

Coccydynia, or coccyx pain, may not be thought of as the cause of persistent chronic pain. Instead, the sign and symptom as clear as increased pain when sitting and localised pain known as coccydynia or coccygodynia. This is typically aggravated by pressure over the coccyx or prolonged sitting and is most common in people with abnormal coccygeal mobility. Although male and female with similarities of prevalence but like five folds more common in women with an average age of 40 years.[4]

The risk factors for developing coccydynia, such as obesity and being female (five times more common than male), are so common in teenagers and adults that rapid weight loss can cause the mechanical cushion to be lost. The underlying causes include falls, dislocation or ligament laxity, fracture, infection (osteomyelitis) and neoplasm (such as chordoma). Internal trauma is most commonly associated with the birth of a child during labour, particularly in the setting of a difficult or instrumented delivery.[5] In addition to articulating with the sacrum, the coccyx is connected to various structures via five main ligaments. The anterior longitudinal ligament originates at the front of the sacrum and extends to the front of the coccyx. The superficial division begins at the edge of the sacral hiatus and attaches to the dorsal surface of the coccyx. The deep division begins at the posterior orifice of the fifth sacral segment, inside the sacral canal, and extends down to the dorsal surface of the coccyx, beneath the superficial division. The lateral sacrococcygeal ligaments connect the lower lateral part of the sacrum to the transverse processes of the coccyx on the same side, as well as to the ischial spines of the ipsilateral sacrum and coccyx. The sacrotuberous ligament connects the ischial tuberosity to the sacrum and coccyx. The coccygeus, gluteus maximus, external anal sphincter, and two levator ani muscles (pubococcygeus and iliococcygeus) all attach to the coccyx. Somatic nerves and the sympathetic ganglion impar carry nociceptive signals from the coccygeal plexus, formed by the fourth and fifth sacral nerves and the coccygeal nerve. The two sacral sympathetic chains converge to form the terminal end of the sympathetic trunk, known as the ganglion impar. This is located close to the midline of the anterior aspect of the coccyx, below the sacrococcygeal joint. The ganglion provides nociceptive and sympathetic innervation to the coccygeal region. This includes afferent innervation from the perineum, distal rectum, anus, distal urethra and lower vagina.[6] Non-traumatic coccydynia is caused by degeneration, laxity or hypermobility of the ligaments of the sacrococcygeal joint, infection, etc. Pain may radiate into the toes and may be confused with sciatica. Comprehensive treatment is required, and patients may have to suffer; correct ergonomic posture is essential, physical manipulation of the levator ani muscles, anti-inflammatory prescriptions, nerve block, steroid or single local anaesthetic or dextrose 5% prolotherapy ganglion impar block may improve in some patients. And few patients need repeat injections. Implantable spinal cord stimulation (SCS) can provide a satisfactory cure, but cost may be an issue. However, coccygectomy is not recommended by many surgeons as the benefits are not worth the risk.[7]

DRG neuromodulation by RF is a promising treatment for coccydynia and may avoid surgical treatment. Steroid blocks are an effective short-term temporary treatment.[8] Injection treatment may be useful for diagnostic prediction and treatment, although the site of injection is controversial. There is a need for more adequate studies of sacrococcygeal and intercoccygeal joint, tender point or ganglion impar injection. Ganglion

impar blocks have been shown to result in approximately 75% improvement at 6 months, while neuromodulation using SCS in the conus region from L2–S2 has also shown improvement in severe perineal pain.[3] Meanwhile, the point entry approach to reach the impar ganglion is challenging as in the case presented a Type III coccyx. We therefore performed a bilateral inferior hypogastric plexus (IHP) block at the S2 segment, which connects to the impar ganglion and the S1 dorsal root ganglion (DRG). The DRG is a key structure in sensory transduction, modulation and pain transmission. Many studies suggest that targeting the DRG can treat both nociceptive and neuropathic pain syndromes.[10,11] Despite the potential complications and adverse effects that accompany it, long-term analgesic medication remains a viable option for managing coccydinia syndrome. Therefore, we hypothesised that nerve root and sympathetic blockades might lead to a better outcome.

METHODS

A 16-year-old female complained of coccydynia for more than 1 month, which. The symptoms occurred after a felt sit down during sport. The motor function, micturation and defecation are normal, tingling or numbness in the perianal area and in both feet. Conservative and medical physiotherapy management has been undertaken, but the symptoms are still experienced as bothersome. The pain is particularly severe with prolonged sitting and can even feel like a burning sensation. Magnetic resonance imaging (MRI) results revealed a Type III coccyx fracture and interventional radiofrequency pain management under local anaesthesia was planned. Radiofrequency (RF) neuromodulation procedures are performed by consultant pain and minimally invasive neurologists who are trained and experienced in complex pain interventions since 2012.

Surgical preparation to operating room standards, emergency equipment, 0.9% normal saline infusion 20 drops per minute, premedicated cefazolin 1 gram antibiotic, C-arm fluoroscopic guidance, Cosman G4 radiofrequency generator, and aseptic and antiseptic measures in the surgical area (regions L4–5 to S1–2). The patient is placed on the operating table in a pronated position with the C-arm fluoroscope in the postero-anterior (PA) position, visualising the L4–5 and S1–2 regions. At the landmark point on the left S2, the posterior neuroforamen was aligned under C-arm fluoroscopy and the CSK 15 RF electrode needle was inserted in a gentle pass through the neuroforamen after 2 mL of lidocaine 2% injection. The depth of the needle could be monitored by switching the C-arm fluoroscope to a lateral view. When inserting the needle inline at the S2–3 articulation, the direction can be maintained. The tip should be stopped when it reaches approximately 0.5 cm anterior to the sacrum, just anterior to the S2 foramen. Confirm the position by injecting 1 mL of Iopamiro 370 dye contrast and it will appear to form a thickened line anterior to the sacrum, indicating that it corresponds to the inferior hypogastric plexus. The C-arm fluoroscope is positioned in PA view to confirm that the needle position is in line with the area of the inferior hypogastric plexus. Sensory stimulation with the Cosman G4 RF device provided a tingling sensation if the readings were above 1.00Hz, followed by motor stimulation to ensure that no nearby viscera were involved. Pulsed RF (PRF) 42°C for 2 minutes followed by injection of triamcinolone 20mg 1ml to prevent neuritis. Similar procedures were performed at the S2 neuroforamen on the right side. Sensory stimulation should not exceed 1.00Hz and motor stimulation should not be used to induce visceral contraction. After confirmation of the stimulated response, pulsed RF at 42°C was applied for 2 minutes and the same procedure was repeated on the opposite side. The PA was viewed at the left S1 neuroforamen, which was the next targeted anatomical landmark for DRG-selective nerve roots. After RF CSK15 electrode needle insertion and the tio is around the DRG, so the 1 mL of dye contrast injected for confirmation. Sensory and motor stimulation was performed to confirm that the RF electrode needle was just around the DRG. Pulsed RF neuromodulation was then performed with the same settings and duration. Postoperative medical therapy included levofloxacin 500 mg/ 24h for 4 days and vitamin B12 50 mcg/ 12h. Analgesics were given in the form of a combination capsule (paracetamol 300mg, diazepam 2mg and diclofenac sodium 12.5mg) and pregabalin 50mg for persistent pain. RF technology provides better pain relief than steroid ganglion block or open surgical treatments.[1]

It is so widely used in the treatment of coccydynia by ganglion impar block because it can reach the nociceptive and sympathetic fibres simultaneously. According to a recent study, it can provide long-term pain relief with minimal risk, except when caution is required regarding neuritis or inadvertent injection from the use of neurolytic agents. In patients with unusual coccyx anatomy, such as in this study of Type III coccyx fractures, difficulties have been observed in approaching the fracture. Therefore, we need an optional approach technique by treating the IHG and S1 DRG. This study adds S1 DRG level for interruption of pain transmission as this may be clinically relevant for chronic intractable pain.[12]

RESULTS

After one month of follow-up, we found that the intensity of the pain had decreased (NRS 0–1), although the hypesthesia around the coccyx could come and go. The analgesic drugs with dose reduced, except when the pain occurs spontaneously. The neurotrophic drugs vitamin

B12 50 mcg/ 12h, the analgesic (paracetamol 300mg, diazepam 2mg and diclofenac sodium 12.5mg) or pregabalin 50mg could be administered if the pain persisted. Overall, she felt much better than before, accompanied by personal treatment while sleeping on the bed with knees bent, then swinging from side to side in a relaxed manner (3–4 times a day). She also avoided sitting for long periods, squatting or sitting in a low position, or sitting on her back.

DISCUSSION

In women, pelvic pain syndromes can be a serious problem as there are many possible causes. These include: urogynaecological (ovarian cysts, endometriosis, premenstrual syndrome), gastrointestinal (mesenteric adhesions, irritable bowel syndrome), neuromusculoskeletal (nerve root compression, fibromyalgia), interstitial cystitis or psychosomatic.[13] Determining which tissue is the source of pain in people with LBP requires a comprehensive assessment and analysis based on clinical examination and imaging. Pain types and syndromes can help to focus on the main problems. The pain experienced by people with LBP is caused by structural abnormalities of the muscles, bones, joints, ligaments, vasculature or nerve fibre units involved. For example, sciatic nerve entrapment is characterised by electrical, radiating pain in the foot, cluneal nerve entrapment manifests as buttock pain, or coccydinia in women may be related to visceral organs. Similarly, the patient who had pain manifested as aggravation with prolonged sitting without radiating pain. As branches S1 to S4, forming the cluneal nerve, innervate the skin of the buttock close to the sacrum and coccyx, this nerve may have been injured after spinal surgery or felt while sitting.[14] The sacral nerves are divided into lateral and medial branches which appear to pass originally through the dorsal sacral neuroforamens. The lateral branches pass posteriorly to the sacrotuberous ligament (STL) and then to the posterior sacrococcygeal plexus (PSCP) or posterior sacral plexus. The adjacent tissue, the posterior sacroiliac ligament, could become entrapped and cause low back pain (LBP). This may be of greater concern if the patient is over 76 years of age. The medial branches contribute from S1 to coccyx on 5 sides (50%) and from S1 to S5 (30%), whereas at S3–S4 and S4–S5 these branches do not contribute. The sacral dorsal rami from S1-S2 formed the LT in 37.5%, S1–S3 in 12.5%, S1–S4 in 31.3% and S2–S4 in 18.8%.[15] The inferior hypogastric plexus (IHP) lies anterior to the sacrum and ventromedial to the sacral foramina S2, S3 and S4. It receives efferent sympathetic fibres from the hypogastric and pelvic splanchnic nerves, preganglionic parasympathetic fibres from the pelvic splanchnic nerves and visceral afferent fibres from the pelvic viscera.[13]

Pain blocks are usually performed on the superior hypogastric plexus (SHP), but this does not provide optimal pain relief. The lower pelvic organs are innervated by the IHP, although this is rarely used for pelvic pain management with effective and safe for improving on chronic pelvic pain. Recently, intrarectal manipulation has been used to adjust the coccygeal joints, which influences the intercoccygeal and sacrococcygeal joints. This increases the coccyx's range of motion by at least 50% and typically provides pain relief within six months. However, patients were not satisfied with this approach in terms of pain relief.[6] Another approach involves injecting glucose to trigger acute inflammation and promote the repair and regeneration of damaged tissue. It has been observed that a low concentration of 5% dextrose reduces pain by influencing sensory peptidergic nerves. Reinjection can improve neural sensation and terminate neuropathic pain. One of our studies revealed that a 5% dextrose solution increases the expression of angiogenic factors, including vascular endothelial growth factor A (VEGF-A), platelet-derived growth factors A and B (PDGF-A and PDGF-B), and insulin-like growth factor I (IGF-I). The study also demonstrated an increase in apoptotic factors, such as caspases 3 and 8, in cultures of adult fibroblasts.[16] Then, inject a solution containing 15% dextrose and 40 mg of lidocaine into the lesion site on the coccyx. This is followed by two injections: the first is given caudally, followed by cranially. Both are administered at a volume of 23 cc. A second set of caudal and cranial injections using a solution containing 20% dextrose and 40 mg of lidocaine at a volume of 4 cc are administered 24 weeks later. These procedures aim to tighten loose tendons, ligaments and joint capsules by multiplying and activating the fibroblasts associated with the pathological structures. The pain score decreased gradually from 8 to 4 and then to 01 by the end of the four-week procedure. If pain relief is not satisfactory, the procedure may be repeated.[17] Surgical treatment, such as amputating a coccyx fragment just above the sacrococcygeal junction, carries a risk of complications and is not always an effective way of relieving pain. For this reason, this procedure is generally not recommended.[18]

Spinal cord stimulation (SCS) technology involves implanting an electrode into the sacral epidural space to interrupt the transmission of pain impulses to the dorsal horn. This neuromodulation technique can provide long-term pain relief by reducing levels of excitatory amino acids and preventing antidromic modulation of the gamma-aminobutyric acid (GABA) and adenosine-dependent systems. SCS covers the S1–S4 anterior and posterior rami, which form the sacral plexus and supply the gluteal, sciatic, posterior cutaneous and pudendal nerves. The posterior rami of the S5 and coccygeal nerves supply sensory innervation to the coccyx region and join with a branch of the anterior ramus of S4 to form the coccygeal plexus and the anococcygeal nerves. These

nerves innervate the sacrotuberous ligament on the dorsal aspect of the coccyx –– the site of pain in coccydynia.[19,20] Another clinician with experience in this field performed the SCS procedure, placing electrodes on the bilateral L1 dorsal root ganglia (DRGs). Positive outcomes were reported in terms of pain relief and reduced requirements for opioid medication.[18] However, proper electrode placement, therapy costs and routine evaluation of electrode implantation must be considered.

Fluoroscopic guidance may be helpful in approaching the dorsal foramen S2, which is usually chosen because it is better visualised than others. Care must be taken when blocking the SHP because of its proximity to the bladder, bowel and common iliac artery, with the potential risk of nerve injury, paraesthesia, haematoma, vascular injury and rectal puncture. The study found that the L1–S3 anterior sacral nerve roots can even be affected by IHP blocks through the S2 neuroforamen level.[13] Approach to the blocked IHP through the S2 foramen appears to extend to the upper (S1) and lower (S3) levels, as recently observed with a success rate of approximately 73% of patients achieving analgesia. Bilateral blocks performed close to the midline and by advancing the drug volume would also increase the success rate of analgesia with this technique.[21] The autonomic fibres from the L4 to S1 segments, corresponding to the T12 and L1 vertebrae. The pelvic spinal nerves (PSNs) separate from the spinal nerves as they exit the sacral foramina and then enter the presacral tissues as a highly intertwined bundle of fibres. The sacral hypogastric fascia gives rise to the sacral nerve roots S1–S4, while the PSNs arise from S2 to S4. They run anterolaterally into the inferior hypogastric plexus (IHP) to supply the pelvic viscera. They also lie superolaterally within the presacral tissue. This IHP is essential for autonomic function, in particular supplying the cervix, the vaginal fornix and the posterior aspect of the bladder and ligaments, either the coccyx or the sacrum. This means that afferent somatic or autonomic fibres from the coccyx area continue proximally to the brain, passing through the sacral DRGs and also the IHP.[11] The autonomic nervous system (ANS) also plays an important role in chronic pain due to clinical and traumatic entities. Sacral fractures and spinal cord injuries can affect the peripheral nervous system (PNS) and the ANS. The coccygeal attachments to the sacrotuberous ligaments are anatomically related to the dural sac and anatomically related to the filum terminale. If the altered structures cause tension or strain on the muscles and tendons that make up the pelvic floor, complaints of low back or coccygeal pain will result. Traumatic sitting can cause changes in the sacrococcygeal angle as the protective mechanisms change. This can lead to instability, hypermobility and hyperflexion of the coccygeal segments in excess of 25 degrees.[22]

The pelvic sympathetic trunk lies extraperitoneally anterior to the sacral bone and anteromedial to the anterior sacral foramen with four or five interconnected ganglia. Above, it continues into the lumbar sympathetic trunk; below, the two trunks converge into a single small ganglion, the impar ganglion, which lies close to the sacrococcygeal joint and the coccygeal apex. The ganglion thus plays an important role in the development of pelvic and coccygeal pain by transmitting sympathetic efferent signals to nociceptive afferent signals from various areas including the perineum, distal rectum, distal vagina, distal urethra and anus.[1] The electrical impulses from the DRG are involved in the expression of pain due to peripheral nerve damage. Pelvic innervation plays an important role in understanding coccydynia and pelvic pain. The cell bodies of the parasympathetic afferents lie within the S2–S4 DRGs and then run together with the pelvic splanchnic and somatic nerves. DRG neuromodulation appears to be a good option for blocking pain transmission or using neuromodulation technology such as radiofrequency or SCS implantation. This may prove that by treating the DRGs, good results can be observed in both nociceptive and neuropathic pain syndromes, which are thought to play a role in chronic coccydynia.[23] Radiofrequency treatment setups are classified as either continuous (CRF) or pulsed (PRF). The applications of these differ depending on temperature, cycle time and the number of repetitions. CRF relies on the thermal energy produced when temperatures are in the range of 60–90 °C. This commonly results in the ablating of tissue and irreversible changes to the targeted nerves. In contrast, PRF is delivered at temperatures below 42°C and neuromodulates pain transmission from the targeted nerves.[24] A combination of superior hypogastric plexus (SHP) treatment and pulsed radiofrequency (PRF) of the sacral roots (S2, S3 and S4) was found to be slightly more effective at relieving chronic pelvic and perineal pain than SHP treatment alone. Following the second procedure, the effectiveness rate ranged from 62% to 72%, demonstrating that sacral neuromodulation can effectively treat pelvic pain. Sacral nerve stimulation may also reduce pain severity and improve quality of life. It is thought that the analgesic effect of PRF is due to electromagnetic waves inducing neuroplastic changes rather than thermal destruction. Furthermore, PRF may alter noradrenergic and serotonergic descending pain inhibitory pathways, as well as reducing tumour necrosis factor-α (TNF-α) and interleukin-6 (IL-6) in neural tissues. Additionally, c-Fos expression increases in laminae I and II of the dorsal horn, while microglia activity is suppressed at the lesion site.[25]

CONCLUSION

Neuromodulation for coccydynia produces good results when treating the sacral dorsal root ganglion (DRG) and

inferior hypogastric plexus (IHP). It also provides easier access to the impar ganglion than the transarticular approach, provided the needle is inserted correctly. However, the anterior angulation of the coccyx can present an obstacle that requires skill to overcome.

CONFLICTS OF INTEREST

We have no conflicts of interest or funding support in relation to this study.

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