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# Avascular Necrosis (AVN) of the Coccyx as a Cause of Coccydynia (Tailbone Pain)

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# Avascular Necrosis (AVN) of the Coccyx as a Cause of Coccydynia (Tailbone Pain)

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## **Abstract**

**Introduction:** Coccydynia (coccyx pain, tailbone pain) has many etiologies. Avascular necrosis (AVN, osteonecrosis) of the coccyx is a rarely reported cause of coccyx pain.

Case report: We present a case of a 35-year-old male who presented with complaints of four years of worsening coccyx pain. He had a history of bilateral hip replacement surgeries approximately 3 years earlier due to AVN of the bilateral femoral heads after multiple exposures to oral steroids and steroid injections at his bilateral hips. This raised our concern that his coccydynia may have been due to AVN of the coccyx. However, magnetic resonance imaging (MRI) of the sacrum and coccyx fortunately revealed no evidence of AVN.

**Discussion:** Our medical literature search found only two prior publications documenting AVN of the coccyx. While diagnostic workup in our particular patient did not reveal sacrococcygeal AVN, it was an important step to have assessed for that possibility, given his history of pelvic AVN and his risks for AVN via both oral and injectable steroids.

**Conclusion:** Although AVN of the coccyx is a rare cause of coccydynia, clinicians should consider this possible diagnosis in coccydynia patients who have risk factors for AVN.

## Introduction

Coccydynia, more commonly known as tailbone pain or coccyx pain, can be due to a variety of causes. Etiologies commonly include direct trauma to the area (e.g., resulting in fracture, dislocation, alignment abnormalities), referred pain to the coccyx region (often due to medical conditions at adjacent body areas like the pelvis or rectum), and idiopathic causes (Foye). A less commonly postulated cause of coccydynia is steroid-induced avascular necrosis (AVN) of the coccyx. Avascular necrosis, also known as osteonecrosis, is a well documented source of pain in a variety of joints including the hip, knee, and carpus. AVN typically follows compromise to the vasculature supplying the respective region (Lourie). A

2007 study found that osteonecrosis of the vertebrae is most commonly seen in the vertebral bodies at levels T12, L1, and L2, and usually presents following nonunion due to ischemia after compression fractures (Yu). The caudally located coccyx receives its blood supply primarily from the median sacral artery, a midline terminal branch of the abdominal aorta (Lourie). Diagnostic confirmation of AVN typically requires a focused history with physical exam and radiographic evidence. This paper reports a case in which a patient with a history of chronic steroid use and pelvic AVN of the bilateral hip joints presented with coccydynia, thus raising concern for possible coccygeal AVN.

## Case Summary

Our patient is a 35-year-old male with coccyx pain that began insidiously four years prior to him presenting to our university-based Coccyx Pain Center. The patient's history was notable for a work-related injury at age 29, when he began to experience groin pain while doing heavy lifting at work. The patient had been diagnosed with a groin tear and started on oral steroids. The patient continued to experience pain and was treated with oral steroids on and off over the next two years. Then, further workup revealed an acetabular labral tear on x-ray which was subsequently treated with intra-articular hip steroid injections and labral tear repair surgery. Shortly after the patient underwent labral tear repair surgery at age 31, he began to experience coccyx pain. The onset of the coccyx pain was insidious, but the pain became dramatically more pronounced one year later, around the time the patient underwent bilateral hip replacement surgeries at age 32 due to AVN of the bilateral femoral heads.

Following the bilateral hip replacements, the patient underwent eight months of physical rehabilitation that included myofascial release and internal manipulation of the coccyx. He also developed signs and symptoms of "body spasticity" concerning for multiple sclerosis and was evaluated by two neurologists. Workup, including magnetic resonance imaging (MRI) and electromyography, could not identify any neurological pathology underlying the spasticity.

Upon presentation to our Coccyx Pain Center, the

patient described his coccyx pain as feeling a small structure "floating in the coccyx area" that sometimes "cracked or popped" with bowel movements and with any flexion or extension at the hip joint. The pain was associated with radiation down the right leg to the foot and occasional numbness in the right thigh, outer hip, and toes. The patient also reported occasional pain with neck flexion that radiated from the neck to the tailbone. Conservative measures including donut and wedge cushions had failed to provide relief. He had tried multiple medications, including NSAIDs (ibuprofen, naproxen), GABA analogs (gabapentin, pregabalin), opiates (oxycodone, oxycontin, buprenorphine) and topical lidocaine. However, these provided only partial relief. The patient had also undergone lumbosacral injection for low back pain at age 31 that provided no relief.

Physical exam in our office revealed sacrum and coccyx tenderness, which was maximal over the lower sacrum and upper coccyx. There was also some diffuse tenderness to palpation over the lumbosacral paraspinal muscles, lower lumbosacral facet joints (zygapophyseal joints), sacroiliac joints (posterior superior iliac spine region), trochanteric bursae, piriformis muscles, and ischial bursae. Lumbosacral extension while standing reproduced discomfort at the lower lumbosacral facet joints. Motor exam revealed full strength throughout the upper and lower limbs. Regarding upper motor neuron signs, upper and lower limb muscle stretch reflexes were 3+ and symmetric bilaterally, and Hoffman's test was positive at the right hand. Straight leg raise was equivocal on the right and negative on the left.

Sacrum/coccyx x-rays including supine, standing, and seated views revealed degenerative joint disease of the sacrococcygeal joint and of the joint between the first two coccygeal vertebral bodies. X-rays also revealed a bone spur at the distal coccyx. Weight-bearing (seated) x-rays did not reveal any coccygeal dynamic instability.

The patient was interested in receiving a coccyx injection to treat his coccyx pain. However, we were concerned firstly regarding the possibility of AVN of the sacrum or coccyx, given his history of repeated use of both systemic (oral) steroids and steroid injections at his pelvis, as well as his history of bilateral hip AVN which had been diagnosed around the same time that his coccyx pain was worsening.

Coccyx injections commonly include placement of corticosteroid, which may be contraindicated at the site of AVN, so we decided to defer interventional pain management procedures until further evaluation for AVN was completed. Thus, the patient was instructed

to first obtain magnetic resonance imaging (MRI) of the sacrum and coccyx before we would determine the best pain management strategy.

Fortunately, MRI of the sacrum and coccyx (without contrast) revealed no evidence of osteonecrosis of the sacrum or coccyx. MRI also showed no significant bone marrow edema within the sacrum or coccyx to suggest bone contusion or fracture and no subluxation or dislocation of the sacrococcygeal segments. An approximately 90-degree angulation of the coccyx was observed, which was consistent with a prior MRI (from 6 years earlier, which was prior to his coccyx pain) and possibly represented a normal anatomic variant. There was mild subcutaneous soft tissue edema posterior to the sacrococcygeal junction. No presacral mass was identified. The musculature adjacent to the sacrum and coccyx appeared symmetric and normal in signal.

## Discussion

Nontraumatic coccydynia can result from various etiologies, including degenerative joint or disc disease, dynamic instability (including listhesis, hyperflexion, or hypermobility), hypomobility of the sacrococcygeal joint, infections, and anatomical abnormalities such as bone spurs (Lirette). Coccydynia may also reportedly be caused by radicular or referred pain, however physical exam maneuvers such as straight leg raise, slump test, lumbosacral flexion/extension, and palpation of lumbosacral facets, sacroiliac joints (posterior superior iliac spine), trochanters, piriformis, and ischial bursae can often rule out those causes of pain. Rare causes of nontraumatic coccydynia include neoplasms and psychological disorders (Lirette).

AVN is a source of pain that has been widely reported to be considered as a differential diagnosis for the hip, knee, and carpal bones. Results from our literature search, however, revealed AVN of the coccyx has only been reported in two published studies (Lourie and Hoekstra).

A review of histological findings from 16 coccygectomy cases by Lourie, et al., identified AVN ("fat cysts" and collections of small foamy cells in the marrow, consistent with early bone necrosis) in the excised coccyx specimens from 2 cases (12.5%) of the 16 cases (Lourie).

Hoekstra, et al., conducted a review of patients with primary sarcoma of the pelvic girdle treated with surgery combined with intraoperative radiotherapy (IORT) at the National Cancer Institute from 1980 to 1988. One of the patients developed AVN of the coccyx seven months post-operatively. Of note, this particular patient underwent six 28-day cycles of chemotherapy with doxorubicin and cyclophosphamide following surgery with IORT (Hoekstra).

The coccyx receives its blood supply mainly from the median sacral artery (MSA), which runs anterior to the sacrum. The MSA is the last artery to branch off of the abdominal aorta. Cadaveric and CT/MRI studies of the anatomy of the MSA have characterized the MSA as a midline vessel branching off of the left common iliac artery, continuing down the ventral surface of the lumbosacral spine, and terminating at the sacrococcygeal junction (Singhatanadgige). Damage to this small vessel from trauma may contribute to avascularity of the coccyx (Lourie).

MRI has emerged as the modality of choice for the evaluation of AVN of bone due to its high sensitivity and specificity. Findings that may indicate AVN include a low signal intensity in the subchondral bone bounded by a low signal intensity border. This border may sometimes appear as a dark line adjacent to a bright line known as the "double line sign" (Richardson).

In our case, the patient had prior AVN of bilateral hips, most likely due to a history of oral steroids and multiple bilateral steroid hip injections. During that time, the patient had also developed coccyx pain. Radiographic and MRI imaging since the onset of coccyx pain had not included the coccyx and we therefore sent him for an MRI of the sacrum and coccyx to rule out AVN. If the MRI results had revealed findings consistent with AVN, then we would have considered steroid injection at the coccyx to be essentially contraindicated. Fortunately for our patient, the MRI did not show any evidence of sacrococcygeal AVN. So, we considered it to be medically reasonable to proceed with a coccygeal corticosteroid injection under fluoroscopic guidance to treat his musculoskeletal causes of sacrococcygeal pain (specifically the degenerative changes and distal coccyx bone spur). Further follow-up and management will be ongoing.

## Conclusions

Our case and the associated literature review emphasizes the importance of including AVN as a differential diagnosis in the setting of coccydynia as it potentially alters the treatment plan for interventional spine physicians. Although the workup fortunately revealed no evidence of AVN in this particular patient, it was worthwhile and medically necessary to have this

reassurance prior to interventional treatment. Physicians should be aware of the possibility, albeit rare, for coccygeal AVN.

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