

INTRAOPERATIVE FINDINGS OF PATHOLOGIC CALCIFICATION SURROUNDING AN INTRATHECAL PAIN PUMP: A CASE REPORT

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Background: Pathologic calcification is a common complication of surgically implanted devices, increasing the incidence of subsequent infection. Most literature describing this phenomenon occurs with cardiac implanted devices. Documented reports of this complication, specifically in the setting of intrathecal pain pump (ITP) and spinal cord stimulator (SCS) implants, are minimal, with presumably low incidence.

Case Report: We present a case of a 58-year-old healthy man with a history of motorcycle accident status post L4-S1 fusion complicated by post-laminectomy syndrome. In February 2023, the patient required replacement of his ITP pump and SCS as both were at the end of life. During the replacement procedure, milky fluid was present with extensive calcifications along the ITP pocket and surrounding the catheter, as well as a small amount around the SCS implantable pulse generator.

Conclusions: This case illustrates the potential effect on the administration of intrathecal medication and neuromodulation if pathologic calcification occurs.

Key words: Chronic pain, intrathecal pain pump, calcification, implanted device, case report

BACKGROUND

Current literature suggests that pathologic calcification surrounding surgically implanted devices seems to be most common with cardiac implanted devices, such as pacemakers and implantable cardioverter-defibrillators. There are few reports of this phenomenon with implanted intrathecal pain pumps (ITPs) and spinal cord stimulators (SCSs) (1,2). One of the main risk factors for pathologic calcification is the length of time that the device remains in place (3,4). There are a few proposed mechanisms for this phenomenon, including pathologic calcification of devitalized cells, encapsulation due to fibrous tissue growth in response to a foreign body, repeated mechanical stress within the device pocket, and acidophilic environments causing microcalcifications (5-8). One of the most concerning complications

of any implant is infection. There have been several studies (9) outlining the association with fibrosis, adhesions, and calcifications resulting in a higher incidence of infection. This can be life threatening in some cases, especially with certain implants, such as ITPs or SCSs that are continuous with the intrathecal and epidural space, respectively. We present a case report outlining the intraoperative findings of significant fibrosis and calcification surrounding an implanted ITP in an asymptomatic patient. This patient underwent both SCS implantable pulse generator (IPG) and ITP replacement at the same time.

CASE PRESENTATION

The patient is a 58-year-old man with severe intractable back pain that started after a motorcycle

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accident in 2007. As a result of his accident, the patient underwent L4-S1 fusion, after which he was prescribed increasing amounts of opioids to attempt to control his pain. He continued to experience back pain and subsequently had an SCS implanted in 2007. Then due to incomplete pain relief, he had an ITP placed at an outside hospital in 2009. Per patient's history and chart review, the patient was responsive to both treatments as he no longer required any oral opioid medications. The patient is otherwise relatively healthy with comorbid conditions of well-controlled hypertension, insomnia, and asthma. He takes nifedipine extended release and lisinopril for his blood pressure, zolpidem as needed for insomnia, and gabapentin and nortriptyline for his chronic pain.

The patient established care with our pain clinic in 2020 for management of his chronic back pain due to post-laminectomy syndrome. He continued to receive significant pain relief from both intrathecal morphine and SCS at that time. On February 14, 2023, the patient was scheduled for replacement of his SCS IPG and his ITP as both had reached their respective end of service.

On the day of the procedure, he denied any signs or symptoms of infection, including fevers, chills, swelling, erythema, or drainage at the implant sites. Preoperative x-ray of the lumbar spine and pelvis (Fig. 1) demonstrated presence of both intrathecal pump and SCS implants with no radiographic evidence of excessive calcification, lead migration, or catheter migration. Physical exam was unremarkable, except for 2 well-healed surgical scars over the respective implanted devices.

The patient was taken to the operating room (OR) and placed in a prone position on the OR table. Sites were prepped and draped in the usual sterile fashion. Decision was made to replace the ITP first followed by the SCS IPG. After incision over the pump was made, there was a moderate amount of milky-white fluid emanating from the ITP pocket. Although the patient did not have any systemic signs of infection as stated previously, the procedure was halted in order to further investigate if this fluid accumulation was due to infectious etiology. Immediately the fluid was sent for culture and gram stain. Once the pump was externalized, we noted several calcifications along the pocket lining (Fig. 2) and the pump itself (Fig. 3). These were scrapped and sent for frozen tissue analysis. These samples along with 3 fluid specimens were hand-delivered to the lab next to the OR. While waiting on

fluid culture results, attention was turned to the pocket itself where there were large calcifications surrounding the catheter as it tunneled from the pocket. This was carefully dissected away from the catheter in order to provide mobility and patency. Pulse lavage with vancomycin and saline was performed after all large calcifications were dissected out of the pocket. Given that part of the ITP catheter was stiff and calcified, we elected to splice this portion and replace this section with a new catheter. The decision was made not to perform a dye study as the patient had been receiving therapeutic benefits from his ITP therapy prior to the replacement procedure. Of note, there was clear fluid determined to be intrathecal morphine dripping from the catheter once it was disconnected from the pump. Therefore, we were confident that the catheter was working appropriately and in the correct location within the intrathecal space.

We were informed by the pathology team within 30 minutes that the frozen tissue samples and fluid specimens showed only diffuse calcifications with no evidence of infection or neoplasm. The SCS IPG site was not addressed until pathology results were received, at which time we made an incision over the previously healed surgical scar and dissected it down to the IPG. Once the IPG was externalized, there was a very small amount of similar milky-white fluid noted within the pocket; however, no calcifications were observed. Since this fluid resembled the fluid sent for culture, the decision was made to proceed with the replacement. Both incisions were closed with 0-Vicryl, 2 layers of 3.0 Vicryl continuous for subcutaneous tissue, and absorbable barbed sutures for the skin.

CONCLUSIONS

Most of the current literature centers on cardiac devices when it comes to the discussion of pathologic calcification involving implanted devices, such as pacemakers and implantable cardioverter-defibrillators. There is currently one case report (10) describing calcification in an SCS IPG pocket in a patient with end-stage renal failure on dialysis. To our knowledge, our case is a novel finding of a calcified surgical pocket surrounding an ITP in an otherwise healthy patient. This demonstration opens up the need for further discussion regarding pathologic calcification within pain management practices.

Our patient never experienced a lapse in treatment from his ITP, and therefore the calcification was an

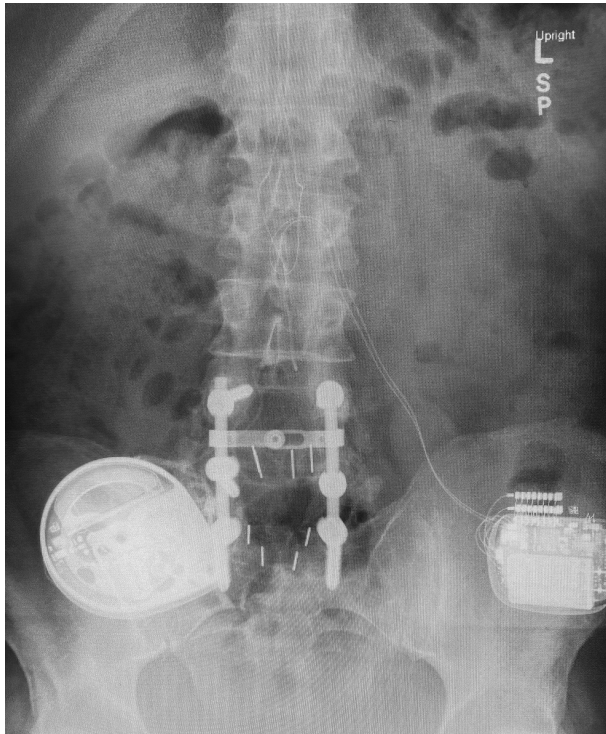


Fig. 1. An x-ray of the patients's lumbar spine showing previous hardware, spinal cord stimulator, and intrathecal pump.

incidental intraoperative finding. However, given the extent of calcification within the pocket and surrounding the catheter, there was a theoretical risk of catheter impingement and possible obstruction within the pocket, which could have reduced the efficacy of his treatment. Even though SCS and ITP pumps have an average life span of 5 to 7 years, patients could have decreased treatment effects earlier due to developing calcification (3,4). Internal cardiac defibrillator distal pacing electrodes implanted in the heart have demonstrated high impedance due to ectopic calcification surrounding the leads, so it is reasonable to conclude that this could also negatively affect neuromodulation devices (11).

Additionally, pathologic calcification of a surgical pocket can closely mimic an infection, and awareness of this phenomenon can help the provider further assess a potentially infected surgical pocket. If a surgical pocket has fluid accumulation, swelling, and white-appearing fluid when undergoing ITP or SCS replacement, one could quickly presume the pocket is infected and abort the procedure. Our case demonstrates the need to increase understanding about pathologic calcification

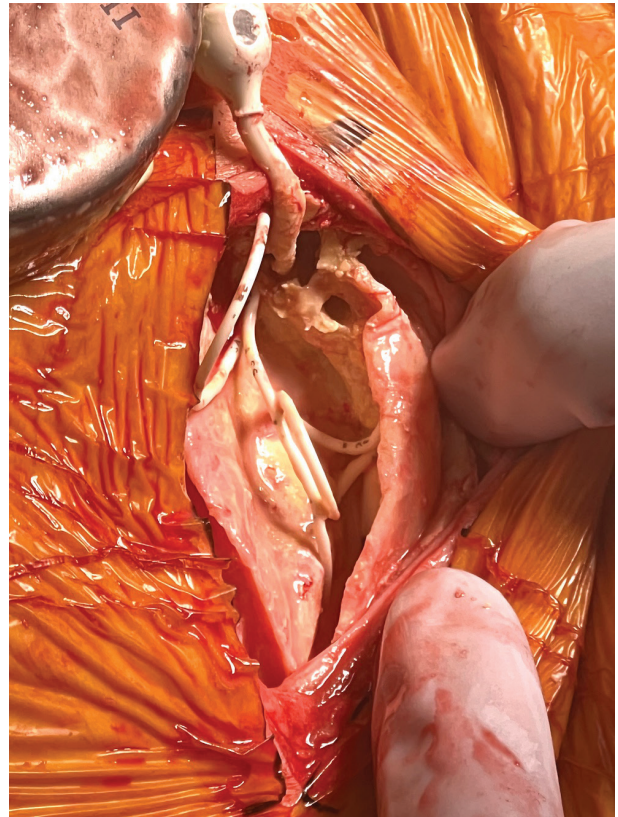


Fig. 2. An intraoperative photo demonstrating calcifications within the surgical pocket surrounding the intrathecal pump.

as aborting the procedure in this setting would only increase the possibility of developing a calcification-related complication, such as high impedance or low effectiveness as seen in cardiac devices (11).

Currently, there is not significant discussion within interventional pain management practices regarding the possibility of calcification occurring or affecting implanted devices, such as SCS and ITP. Unlike with cardiac implanted devices, pathologic calcification is not known to be a commonly encountered complication with implanted devices in the pain setting, though now there is evidence that this possibility needs to be further explored. It is important for both the patient and physician to understand the risks associated with any device implantation and its subsequent complications. Our novel case report shows that more research needs to be done to define these risks when it comes to pathologic calcification in the pain setting. Some preventative strategies have been studied in pacemakers, such as using a decellularized

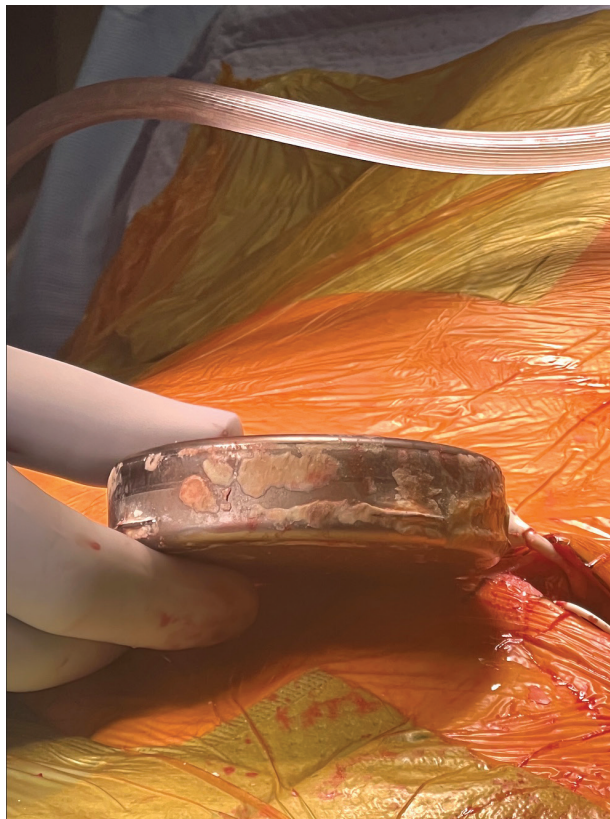


Fig. 3. Externalized intrathecal pump with calcifications surrounding the pump and catheter.

extracellular matrix envelope that surrounds the implant when placed into the surgical pocket (12). This has been shown to improve tissue remodeling around the device, subsequently decreasing fibrosis and calcification. Our case report demonstrates that

more investigation and discussion are warranted regarding pathologic calcification incidence, potential risk factors, subsequent complications, and preventative strategies within the interventional pain management setting.

REFERENCES

1. Basarakodu K, Lakkireddy D, Li H, Tchou P. "Porcelain pocket": Dystrophic calcification of an implantable cardiac defibrillator pocket presenting as impending erosion. *Heart Rhythm* 2005; 2:787-788.
2. Bieffer HRC, Hurlimann D, Grunenfelder J, et al. Generator pocket adhesions of cardiac leads: Classification and correlation with transvenous lead extraction results. *Pacing Clin Electrophysiol* 2013; 36:1111-1116.
3. Legrand AP, Marinov G, Pavlov S, et al. Degenerative mineralization in the fibrous capsule of silicone breast implants. *J Mater Sci Mater Med* 2005; 16:477-485.
4. Steckiewicz R, Swieton EB, Kołodzinska A, Bogdanska M, Fus Ł, Solarz P. Morphometric parameters of cardiac implantable electronic device (CIED) pocket walls observed on device replacement. *Folia Morphol (Warsz)* 2017; 76:675-681.
5. Anderson HC. Mechanisms of pathologic calcification. *Rheum Dis Clin North Am* 1988; 14:303-319.
6. Massaro G, Leone O, Valzania C, et al. Pocket histology at cardiac implantable electronic device replacement: What's new? *Heart Rhythm* 2023; 20:198-206.
7. Trop I, David J, El Khoury M, Gautier N, Gaboury L, Lalonde L. Microcalcifications around a collagen-based breast biopsy marker: Complication of biopsy with a percutaneous marking system. *AJR Am J Roentgenol* 2011; 197:353.
8. Schoen FJ, Levy RJ. Calcification of tissue heart valve substitutes: Progress toward understanding and prevention. *Ann Thorac Surg* 2005; 79:1072-1080.
9. Cakir E, Kuzeyli K, Usul H, Peksoylu B, Karaarslan G, Yildiz K.

- Shunt dysfunction due to calcification of a ventriculo-peritoneal shunt: A case report. *J Clin Neurosci* 2004; 11:210-211.
10. Bamps S, Dejaegher J, Sprangers B, Theys T. Severe calcification in the abdominal pocket of an implantable pulse generator (IPG) in a patient on long-term hemodialysis. *J Clin Case Rep* 2017; 7:1-2.
 11. Hauser RG, Sengupta J, Casey S, Tang C, Stanberry LI, Abdelhadi R. High shocking and pacing impedances due to defibrillation lead calcification. *J Interv Card Electrophysiol* 2020; 58:253-259.
 12. Liu K, Omar M, Samy Elayi C, Xiang KK, Catanzaro JN. Use of a novel extracellular matrix to mitigate severe fibrosis and calcification at time of permanent pacemaker generator change. *J Am Coll Cardiol* 2022; 79:2493.

