

Posture-Induced Radial Nerve Palsy in a Patient with Advanced Pancreatic Cancer: A Palliative Care Case Report

ABSTRACT

Radial nerve palsy is an established compressive mononeuropathy but is rarely described in palliative care. A 76-year-old man with metastatic pancreatic body adenocarcinoma developed acute left wrist drop with finger extension weakness and radial sensory disturbance after repeatedly sleeping leaning forward over an overbed table for symptom relief. Examination localised the deficit to the radial nerve. He was managed conservatively with splinting, physiotherapy, occupational therapy input and posture modification, with full recovery in four weeks. This case highlights a potentially preventable complication of adaptive sleeping posture in advanced cancer.

KEYWORDS

radial nerve palsy, wrist drop, palliative care, pancreatic cancer, compressive neuropathy.

INTRODUCTION

Radial nerve palsy is a peripheral mononeuropathy that typically presents with weakness of wrist, finger and thumb extension. The radial nerve arises from the posterior cord of the brachial plexus, supplies the extensor compartments of the arm and forearm, and provides sensory innervation to parts of the posterior arm, posterior forearm and dorsoradial hand.¹ Injury can therefore produce wrist drop, impaired finger extension and variable sensory loss, depending on lesion level.

Posture-induced compressive radial neuropathy, classically termed Saturday night palsy, occurs after prolonged external pressure on the radial nerve. Han et al. described sleep-related cases after the arm was compressed over a chair armrest or under a pillow. In that series, wrist drop was the commonest presentation, sensory symptoms often affected the dorsum of the first to third fingers, and subjective improvement began after a mean of 2.4 weeks.²

In advanced cancer, new focal neurological deficits may raise concern for metastatic nerve compression, plexopathy, radiculopathy or treatment-related neurotoxicity. In palliative care, additional factors such as cachexia, immobility, sedating medication and altered sleeping posture may plausibly increase vulnerability to compression neuropathy, although direct evidence in this setting is limited. Cancer cachexia is associated with loss of skeletal muscle, and strong opioids are commonly used for pain in advanced progressive disease.^{3,4}

This report describes acute posture-induced radial nerve palsy in a patient with metastatic pancreatic cancer who adopted a compensatory sleeping position because of pain and abdominal discomfort. Consent was obtained from the patient prior to starting the case report, with the identity and personal details kept anonymous.

CASE PRESENTATION

A 76-year-old right-hand-dominant man with metastatic pancreatic body adenocarcinoma with hepatic metastases was diagnosed in 2024. Imaging at diagnosis demonstrated an inoperable pancreatic body mass with vascular invasion and liver lesions compatible with metastatic disease. His history included previous invasive gastric adenocarcinoma treated with neoadjuvant FLOT chemotherapy and near-total gastrectomy in 2022, and previous panproctocolectomy for adenomatous polyposis. He had received 12 cycles of FOLFIRINOX (folinic acid, fluorouracil, irinotecan and oxaliplatin) for pancreatic cancer and was later admitted to the Palliative Care Unit for symptom control.

During admission, the patient developed acute left-sided wrist drop on waking. He had marked weakness of left wrist and finger extension and reduced sensation over the dorsal web space between the thumb and index finger. The right upper limb was normal, and there was no associated neck pain. The affected limb was his non-dominant side.

A key feature was the repeated adoption of an unusual sleeping posture. Because lying in bed worsened his pain and abdominal discomfort, the patient slept in an armchair leaning forward with his trunk flexed, arms supported on an overbed table and head resting on his arms (Figure 1). This posture was considered a plausible mechanism for prolonged radial nerve compression during sleep.

At onset, he was receiving ketamine and morphine by continuous subcutaneous infusion, with additional subcutaneous morphine as required for breakthrough pain. These drugs were clinically relevant because sedation and reduced spontaneous repositioning may have contributed to sustained pressure in an abnormal position.

Neurological examination showed an isolated left radial nerve pattern deficit. Left wrist extension was MRC grade 1/5 and left finger extension was MRC grade 1/5. Elbow flexion

Figure 1. Representation of the sleeping posture described in the case. The patient is seated and leaning forward with trunk flexed, arms supported on an overbed table and head resting on the arms.



and extension were 5/5, and shoulder flexion and extension were 5/5. Sensory examination showed reduced sensation over the first dorsal web space. The left brachioradialis reflex was diminished, while the triceps reflex was normal.

The pattern favoured a radial nerve lesion proximal to the posterior interosseous nerve, because posterior interosseous neuropathy is predominantly motor and should not cause superficial radial sensory loss.⁵ Preserved shoulder and elbow strength made brachial plexopathy less likely, while the absence of neck pain and the focal radial pattern made cervical radiculopathy less likely. Metastatic nerve compression was considered because of his malignancy, but the acute onset on waking after prolonged abnormal positioning supported a compressive mononeuropathy.

Chemotherapy-induced peripheral neuropathy was also considered. FOLFIRINOX includes oxaliplatin,⁶ and chemotherapy-induced peripheral neuropathy classically develops in a distal glove and stocking distribution.⁷ This background was relevant, but it did not explain an acute unilateral wrist drop with focal radial sensory involvement.

No imaging, nerve conduction studies or electromyography were performed specifically for the wrist drop, as these were not expected to change management in the context of the patient's palliative goals of care. The diagnosis was therefore clinical. Management was conservative and included wrist splinting, physiotherapy, occupational therapy input, education regarding posture modification, and advice to avoid pressure over the table edge. The patient made a full clinical recovery over approximately four weeks.

DISCUSSION

The most likely mechanism was posture-induced compression of the radial nerve, probably at the spiral groove or mid-humeral region. The temporal sequence was important: the patient slept in a fixed compensatory posture and woke up with acute wrist drop, severe wrist and finger extensor weakness, and sensory disturbance in the radial nerve distribution. Subsequent complete recovery with conservative management supports a reversible compression injury rather than malignant infiltration or a progressive neurological disorder.

This case differs from the traditional description of Saturday night palsy, which is often linked to alcohol-related deep sleep or intoxication. In this patient, the precipitating factors were cancer-related pain, abdominal discomfort, cachexia, palliative symptom burden and analgesic treatment. The posture that relieved discomfort also placed the arm at risk of sustained compression. This is clinically relevant because palliative patients may prioritise a position that reduces pain, even if that position increases pressure on superficial nerves.

The neurological findings were useful for localisation. Sensory loss over the first dorsal web space implied involvement of the superficial radial sensory distribution. Diminished brachioradialis reflex suggested a lesion proximal enough to involve brachioradialis fibres, while preserved triceps strength and

reflex argued against a very high radial nerve lesion. Preserved shoulder and elbow power further reduced the likelihood of a major plexus or root lesion.

The case also has limitations. The diagnosis was not confirmed by neurophysiology or imaging, so the exact site and severity of nerve injury could not be proven. Alternative structural causes could not be excluded with absolute certainty. It is a single case, so no conclusions can be drawn regarding the frequency of radial neuropathy in palliative care. Follow-up was limited to clinical recovery over four weeks.

Despite these limitations, the case illustrates a practical and potentially preventable cause of morbidity. Regular assessment of sleeping posture, padding of hard surfaces, arm support, caregiver education and early physiotherapy and occupational therapy input may help preserve function in palliative care patients with pain-driven positioning or reduced mobility.

CONCLUSION

Radial neuropathy may occur as an unintended consequence of adaptive sleeping postures adopted for symptom relief in advanced malignancy. Palliative care patients may be especially vulnerable because of cachexia, immobility, pain, sedating medication and prolonged positioning. Early recognition, posture correction, splinting and multidisciplinary rehabilitation may prevent avoidable morbidity and support functional recovery.

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