



Correction to: Lumbosacral plexopathy caused by the perineural spread of pelvic malignancies: clinical aspects and imaging patterns

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Correction to: Acta Neurochirurgica (2022) 164:1509–1519
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In the comments section, the references are missing. The correct comments are shown below.

Comments:

The paper by Lee et al. describes 9 patients collected over 15 years with lumbosacral plexopathy (LSP) associated with a variety of pelvic malignancies (e.g., prostate, cervical and rectal) and perineural spread. It is gratifying to see some of our contributions [1] not only corroborated but extended. This case series supports the notion that the entity of neoplastic lumbosacral plexopathy is not as rare as thought: being underdiagnosed, misdiagnosed and underreported. In the past, these patients were assumed to have LSP due to radiation, chemotherapy, or inflammation or that is idiopathic.

The neural highways [2] underlying neoplastic LSP originate at the affected pelvic organ (or staple line) where perineural invasion transitions to perineural spread. Perineural spread continues via pelvic autonomic nerves (i.e., inferior hypogastric plexus) to the lumbosacral plexus and can extend proximally to spinal nerves, dura, and, even to the other limb [3] and/or distally to the sciatic nerve. Perineural spread can go considerable distances [4]. Clinical presentations and radiologic patterns are emerging based on the pathoanatomy and sequential features can be interpreted based on the pathoanatomic findings.

In this paper, neoplastic LSP was established by radiologic features (enlarged nerves with increased T2 and perifascicular enhancement on MRI; often with increased

avidity on PET). Only 2 patients had tissue confirmation. At our institution, we believe "Tissue is the issue" and utilize the technique of image-guided targeted fascicular biopsy [5] whenever feasible. We search for an extrapelvic site whenever possible and often identify blue infiltrates within the nerve [6]. Occasionally, we are forced to treat patients empirically who are thought to have perineural spread of a pelvic cancer but confirmatory tissue is unobtainable and suspicion is high.

Several words of caution. Radiologic findings by themselves can be non-specific. One cannot establish diagnosis on T2-weighted images alone and patterns of contrast enhancement may be variable with few truly pathognomonic features that would negate the need for tissue diagnosis. We suggest PET-CT or MR correlation in all cases (note in this series, only 7 of 9 patients had PET and 1 of these PET studies was not avid). The radiologic differential diagnosis is broad. We have treated several patients with imaging features who were thought to have perineural spread from the primary cancer but who were found to have a radiation-induced malignant peripheral nerve sheath tumor [7] (note 5 patients in this series had previous radiation). Furthermore, the presence of myokymic discharges on EMG (which is supportive of a diagnosis of radiation plexopathy and was used to exclude potential patients in this series) does not exclude concomitant cancer.

Ultimately optimal targeted treatment must consider the specific diagnosis, the mechanism (perineural spread) and the extent of disease (with/or without concomitant distant haematogenous or lymphogenous spread). Surgical resection is often not feasible. Adjuvant therapy needs to be considered.

Perineural spread is well known when involving the head/neck with certain types of cancer in particular (e.g.,

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melanoma, squamous cell carcinoma, etc.). It is time to expand our knowledge to other sites, including the lumbosacral plexus and brachial plexus, and other types of cancers [8, 9].

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The publisher regrets this error.

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