A CASE REPORT

Electroconvulsive Therapy for Neuropathic Pain: A Case Report and Literature Review

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Objective: To describe a case of intractable brachial plexopathy-induced neuropathic pain syndrome treated with electroconvulsive therapy after a failed trial of conventional drugs and interventional pain management.

Case Report: A 32-year-old male had chronic intractable neuropathic pain of the right upper extremity and shoulder for about 10 years, due to brachial plexopathy. He tried multiple pain medications and underwent various interventional pain procedures without significant pain relief. When the patient subsequently developed severe depression with suicidal ideation, he underwent electroconvulsive therapy, which significantly improved the depression and pain for two months.

Discussion: There is a growing list of non-pain psychiatric conditions that may be treated with electroconvulsive therapy. Chronic intractable pain with or without depression has been on and off the list for years. Further studies may eventually demonstrate efficacy of ECT for intractable neuropathic pain syndromes.

Keywords: Electroconvulsive therapy, neuropathic pain, depression, intractable pain

Neuropathic pain is defined as pain that results from damage or abnormal function of the central or peripheral nervous system. Neuropathic pain can be difficult to manage using conventional treatment modalities. The usual treatments include opioids, anticonvulsants, antidepressants, antiarrhythmics and interventional procedures (e.g., spinal cord stimulators, nerve blocks, and neurosurgical ablation). Unfortunately, many patients fail these treatment modalities. Though not well studied, a few case reports illustrate the benefit of electroconvulsive therapy (ECT) in the management of chronic pain syndromes.

The exact origin of ECT is unknown. It probably dates back to the medical usage of eels by the Romans to treat headache and later the experiments of Italian naturalists with electricity (1). Further, the physician Galen employed electricity in the treatment of gout and other illnesses. Later, a Hungarian psychiatrist, Laszlo Meduna, was credited with the first modern use of convulsive therapy. He was intrigued by the studies of Nyiro and Jablonski (2) in the 1920s, which demonstrated that patients with schizophrenia seemed to have a lower incidence of epilepsy than the general population. They postulated that dementia praecox (the old term for schizophrenia) had a curative effect on epilepsy, and Meduna wondered if the reverse might be true. He found that convulsions induced with intravenous Metrazol or intramuscular camphor could treat psychosis. Though Meduna is credited as the father of convulsive therapy, it was not until the late 1930s that Drs. Bini and Cerletti in Italy were successful in using electric currents to induce therapeutic seizures (3).

We describe a case of a 32-year-old man with a neuropathic pain syndrome who had received various treatments without satisfactory results. Subsequently, he was treated with electroconvulsive therapy for concomitant depression, which improved the depression and alleviated the chronic pain.

CASE DESCRIPTION

Our patient was a 32-year-old male with a 10-year history of chronic right arm and shoulder pain due to chronic idiopathic brachial neuritis. His pain started spontaneously as an aching, burning pain in the right shoulder, which then spread down the arm to the hand, and proximally to the lateral portion of the right side of the neck. He also complained of pain radiating into the scapular area and toward the thoracic spinal column. He described the pain as burning, sharp and crushing in nature. There was no particular activity that worsened the pain. Of note, in the early 1990s for a year or two, his pain nearly resolved spontaneously. Following a bout of prostatitis, the pain flared up again. Subsequently, the pain became progressively worse, to the level of 9-10/10 on the visual analog scale.

His past medical history included hypertension, seizure disorder and depression. He was treated as an inpatient and outpatient multiple times for depression. On physical examination, his right arm was noted to have decreased sensation to light touch and cold along the posterior aspect of the arm. There was hyperesthesia to pinprick in the right posterior shoulder area, right posterior arm, and the distal lateral wrist, encompassing an area of approximately 5 cm by 7 cm. There was obvious atrophy of the pectoralis muscle. He was able to demonstrate at least 3/5-muscle strength in the shoulder.

MRI of his brachial plexus showed no anatomical abnormality, and MRI of the cervical spine showed no cord or nerve compression. An EMG showed chronic changes consistent with brachial...
plexopathy with involvement of the infra-
spinatus and supraspinatus muscles.

Treatment included continuous in-
fusions of local anesthetics via an inter-
pleural catheter, an intravenous lidocaine
test, stellate ganglion blocks, TENS and
interscalene brachial plexus block, all of
which resulted in temporary mild pain re-
lied, but no long-lasting relief. Medi-
cations included methadone 100 mg a day
in divided doses, hydromorphone 2 mg
oral every 8 hours as needed, gabapen-
tin 1200 mg 3 times a day, clonidine 0.1
mg daily, clonazepam 1 mg four times a
day, and ibuprofen 800 mg 3 times a day.

Polypharmacy, interventional pro-
cedures, and behavioral pain manage-
ment were unsatisfactory. He developed
recurrent severe depression and attempt-
ed suicide. He was admitted to a psychi-
atriatric hospital for depression and suicidal
ideation. While hospitalized, he under-
went five right unilateral electroconvul-
sive treatments under methohexital (80
mg) and succinylcholine (100 mg) anes-
thesia, with an average seizure duration
of 62 seconds. He experienced relief of both
the chronic pain and depression.

The patient spent approximately 2
weeks in the psychiatric hospital and then
was discharged home. Subsequently, he
did relatively well and began to taper his
medications, including the opioids. Dur-
ing the recovery phase, he rated his pain
as 3/10 on a visual analog scale. He was
happy with the response and increased his
activity level significantly. Unfortunately,
eight weeks later, the pain recurred. Al-
though it is tempting to attribute the en-
tire pain syndrome to a psychiatric disor-
der, this patient had bona fide evidence of
brachial plexopathy and appeared to be in
pain. The ECT treatments correlated with
the 8-week period of pain relief.

**Discussion**

It is widely observed and acknowl-
edged that there is an association between
chronic pain and depression. Conse-
quently, many physicians initially believed
that antidepressants and psychotropic
drugs might relieve chronic pain by
improving depression. It is true that relative-
ly small doses of tricyclic antidepressants
can improve neuropathic pain, and adren-
ergic and/or serotonergic mechanisms are
hypothesized.

Although ECT may be considered a
nonpharmacologic method of treating
depression, there are undoubtedly signifi-
cant pharmacologic effects of ECT on the
brain. ECT is often the only known effec-
tive treatment for severe depression resis-
tant to medications. ECT may also be an
effective and relatively safe treatment for
psychosis and mania.

Not surprisingly, ECT is rarely con-
sidered for chronic intractable pain syn-
dromes today, although there is an old, but
rather sparse literature on the therapeutic
use of ECT for pain. Mandel (4) report-
ed that ECT alleviated symptoms for four
of six patients suffering from chronic pain
and depression. All of those patients had
failed treatment with tricyclic antidepress-
sants. Thus, he suggested that unilateral
ECT might be the treatment of choice for
patients suffering from chronic pain and
intractable depression.

Bloomstein et al (5) reported that
out of twenty-one patients who received
ECT for concurrent affective symptoms,
twenty experienced improvement in their
pain levels, and concluded that ECT
could be an effective treatment modality
for patients who have chronic pain com-
plified by affective symptoms. Further,
King et al (6) described a patient with
depression who developed RSD and was
helped by ECT. The patient had total res-
olution of pain for several hours after the
first treatment. Over the course of the
full ECT series, pain, vasculature chang-
es of RSD, and depression resolved com-
pletely.

On the other hand, McCance et al
(7) reported that ECT was not effective in
the treatment of post-stroke thalamic
pain. The authors performed a pilot
study in 3 patients who received six bilat-
eral ECT treatments over a period of two
weeks and observed no improvement in
thalamic pain or mood. Furthermore,
Salmon et al (8) could not verify a posi-
tive response to unilateral ECT treat-
ment for thalamic pain. They report-
ed that unilateral ECT applied to four
patients with intractable thalamic pain
did not improve pain or affective pro-
files. The authors also measured venous
plasma endorphins during the course of
ECT therapy and found no significant
 correlations with ECT treatment. Inter-
estingly, unilateral ECT in these patients
was not effective for depression, but bi-
lateral ECT was effective. They hypothe-
sized that bilateral ECT resulted in cur-
rent passage through the diencephalon,
including the thalamus and hypothala-
mus (9, 10). These structures are well
known pathways for pain sensation and
perception.

Even though there is little definitive
evidence that ECT is effective for chron-
ic pain, the idea is intriguing. Indeed, it
has been suggested that some complaints
of chronic pain represent a symptom of
underlying depression (4, 10, 11). Lason
et al (12) suggested that the transient im-
provement in pain observed in four out
of ten patients after ECT might be related to
temporary ECT-induced delirium. In-
deed, changes in a number of neurotrans-
mittner systems may occur with ECT. The
 persistence of EEG slow wave activity fol-
lowing ECT and its correlation with clin-
ical improvement suggests that a physi-
cal alteration in cerebral activity has oc-
curred. Both antidepressants and ECT
seem to alter serotonergic neurotransmis-
sion. ECT up-regulates 5-HT2, and an-
tidepressants down-regulate receptor
expression (13).

Interestingly, Lason et al (12) ob-
served a differential effect of ECT on the
prodynorphin and pro-opiomelanocortin
system in the rat. They studied the
 effect of single and repeated ECT on im-
 munoreactive (ir) dynorphin and beta-
endorphin levels in the rat brain, spinal
cord and pituitary. They reported that
there was a reduction in ir-dynorphin
and beta-endorphin levels in the hypo-
thalamus and beta-endorphin in the pi-
titary, when ECT was applied as a sin-
gle dose. However, repetitive applica-
tions resulted in significant increases of
ir-dynorphin, but not beta-endorphin in
the hypothalamus. They concluded that
the observed prolonged changes in dyn-
orphin levels after chronic ECT may play
a role in enhanced post-shock opiate-like
behavior, as well as provide a mechanism
for ECT therapy. Furthermore, Hong et
al (14) and Sarne et al (15) have report-
ed that ECT led to a long-lasting increase
in met- and leu-enkephalin levels in the
hypothalamus as well as in the rat lim-
bic system.

Even though ECT was used for vari-
ous types of intractable chronic pain dur-
ing the 1940s and 1950s, it is not now of-
ten used for pain, for obvious reasons.
There are no controlled studies to clarify
the role of ECT for intractable pain and
there are only a few case reports with vari-
able results. The negative image associat-
ed with ECT makes it unlikely that con-
trolled trials will ever be done. Despite
the fact that the risk of prolonged amnesia
or induced psychosis from ECT is well under 1% and the estimated mortality rate following ECT is less than 0.03% (16). More recent reports confirm the safety of ECT, even for elderly patients (17). Nonetheless, concerns about such factors as amnesia and injury from ECT make the option rather unpopular, despite the fact that more recent evidence, again from case reports, suggests that ECT may benefit subsets of patients with chronic pain. Recently, ECT has been reported to help patients with phantom limb pain (18), headache (19), and CRPS (20).

In conclusion, although ECT is widely accepted as a treatment for severe depression, it is seldom used in the treatment of chronic pain, despite the fact that chronic pain and depression frequently occur together. Our report is intended to rekindle a discussion about the possible use of ECT in depressed patients presenting with intractable pain syndromes. Although prospective randomized studies are needed to demonstrate the effectiveness of ECT for the treatment of chronic pain, such studies most likely will never be done. Nonetheless, there is anecdotal evidence that ECT may alleviate symptoms of depression and pain in some patients who have failed conventional treatment.

**REFERENCES**


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