

acid in the spinal cord.⁸ Moreover, gabapentin may modify central perception of itch by a modulation of μ -opioid receptors.⁹

Pregabalin is very similar to gabapentin regarding structure and mechanism of action. Compared to gabapentin, pregabalin is characterized by a more rapid time of response. Based on this characteristic, pregabalin has been preferred to gabapentin to obtain the most rapid response for a very distressing symptom. Pregabalin does not bind to plasma proteins and it is not subject to hepatic metabolism; these characteristics make it particularly attractive for patients with advanced cancer, who often present with low levels of plasma proteins and/or hepatic failure.¹⁰

In our patient, pregabalin seemed to be effective in the treatment of cetuximab-related itch. Drowsiness was the only adverse effect registered; it was transient and did not require specific treatment. We observed a dose-response effect. Therefore, we suggest a careful titration of doses to obtain the best response and minimize the adverse effects. Compliance with cetuximab-based chemotherapy was maintained and scheduled chemotherapy was not modified.

To our knowledge, this is the first report on the treatment of itch with pregabalin. Considering the expanding role of cetuximab in the management of solid tumors, and the significant incidence of itchy skin reactions, pregabalin should be considered a promising drug to treat this distressing adverse effect. Further evaluations are warranted to confirm or refute our observation.

Giampiero Porzio, MD
Federica Aielli, MD
Lucilla Verna, MD
Supportive Care Task Force
Medical Oncology Department
University of L'Aquila
L'Aquila, Italy

Claudio Porto, MD
Neurophysiopathology Unit
San Salvatore Hospital
L'Aquila, Italy

Marianna Tudini, MD
Katia Cannita, PhD
Corrado Ficorella, MD
Medical Oncology Department
University of L'Aquila
L'Aquila, Italy

doi:10.1016/j.jpainsymman.2006.07.006

References

1. Segaert S, Can Cutsem E. Clinical signs, pathophysiology and management of skin toxicity during therapy with epidermal growth factor receptor inhibitors. *Ann Oncol* 2005;16:1425–1433.
2. Segaert S, Tabernero J, Chosidow O, et al. The management of skin reactions in cancer patients receiving epidermal growth factor receptor targeted therapies. *J Dtsch Dermatol Ges* 2005;3:599–606.
3. Lacouture ME, Basti S, Patel J, Benson A 3rd. The SERIES clinic: an interdisciplinary approach to the management of toxicities of EGFR inhibitors. *J Support Oncol* 2006;4(5):236–238.
4. Perez-Soler R, Delord JP, Halpern A, et al. HER1/EGFR inhibitor-associated rash: future directions for management and investigation outcomes from the HER1/EGFR inhibitor rash management forum. *Oncologist* 2005;10:345–356.
5. Scheinfeld N. The role of gabapentin in treating disease with cutaneous manifestations. *Int J Dermatol* 2003;42:491–495.
6. Winhoven SM, Coulson IH, Bottomley WW. Brachioradial pruritus: response to treatment with gabapentin. *Br J Dermatol* 2004;150:786–787.
7. Yesudian PD, Wilson NJE. Efficacy of gabapentin in the management of pruritus of unknown origin. *Arch Dermatol* 2005;141:1507–1509.
8. Fehrenbacher JC, Taylor CP, Vasko MR. Pregabalin and gabapentin reduce release of substance P and CGRP from rat spinal tissues only after inflammation or activation of protein kinase C. *Pain* 2003;105:133–141.
9. Yoon MH, Choi J, Jeong SW. Spinal gabapentin and antinociception: mechanisms of action. *J Korean Med Sci* 2003;18:255–261.
10. Shneker BF, McAuley JW. Pregabalin: a new neuromodulator with broad therapeutic indications. *Ann Pharmacother* 2005;39:2029–2037.

A Case of Visceral Post-Stroke Pain

To the Editor:

I report a patient who was a right-handed, married, male, noninsulin-dependent diabetic (treated with metformin 500 mg b.d.) former crane driver who, in 1991, at the age of 52, had a stroke, which caused left-sided ataxia and nystagmus. This resolved after a short (but unknown) time. Six months later, he experienced a burning sensation in his right

shoulder, which lasted for a few weeks and then disappeared. Three months after this (i.e., 9 months after the first stroke), he noticed a burning, scalding sensation superficially in his lower chest and internally in the upper abdomen, with occasional radiation down into the left groin. He said that his pain was “aggravated” by contact with the bedclothes at night. The pain, which was said to be constant, was, to some extent, relieved by a hot bath.

Dosulepin (dothiepin) hydrochloride 75 mg daily was prescribed; he had been taking it for 9 months before referral to my clinic, together with a compound tablet of codeine phosphate 30 mg and paracetamol (acetaminophen) 500 mg (up to 8 tablets per day), which he said made him constipated. The patient said he had put on 14 pounds in the “few weeks” preceding his visit to the clinic.

This very obese man (height 1 m 64 cm and weight 99.3 kg) was referred to me and initially seen some 18 months after the first stroke. At that time, his principal complaint was of (internal) abdominal pain, worse on the left than on the right, and very occasionally going into the top of the left thigh; he also suffered from “occasional” girdle pains in the lower thorax. He insisted that the pain was “inside, like a stomach ache,” with additional (and separate) surface girdle pains. He rated his pain at worst as 100 on a visual analog scale (VAS) and at best as 45.

On examination, he had a slight degree of rombergism. Power and tendon reflexes

were normal and equal on the two sides, with the exception that the left abdominal reflex was considerably reduced in comparison with the right; the plantar reflexes were downgoing.

There was tactile allodynia in the T8 segment. On the anterior abdominal wall in the T7 and 8 dermatomes, v. Frey, sharpness, and heat pain thresholds were equal (i.e., unaffected); warmth threshold was raised 8°; skin-fold pinch and cold thresholds were not tested.

Amitriptyline 25 mg t.d.s. and sodium valproate 200 mg b.d. were substituted for the dosulepin. When seen three months later, he said he was “much better” but still had some pain, particularly in the left iliac fossa, where there was tenderness and guarding (he was fairly severely constipated); he no longer felt any pain in his thigh. At this time, he rated his worst pain at 70 on the VAS scale, and three months after that, at 50. Two-and-a-half years after the first incident, he felt no pain, only “numbness,” provided he was taking amitriptyline; but pain returned after 48 hours if he stopped taking the tricyclic.

In 1992, an unenhanced magnetic resonance scan was performed 22 months after the first ictus and 14 months after the onset of abdominal pain. The only supratentorial abnormalities seen in axial and coronal views consisted of increased density in the right anterior superior insular region (Fig. 1), and a slightly suspicious appearance in the symmetrical region on the other side. It is possible that a lesion

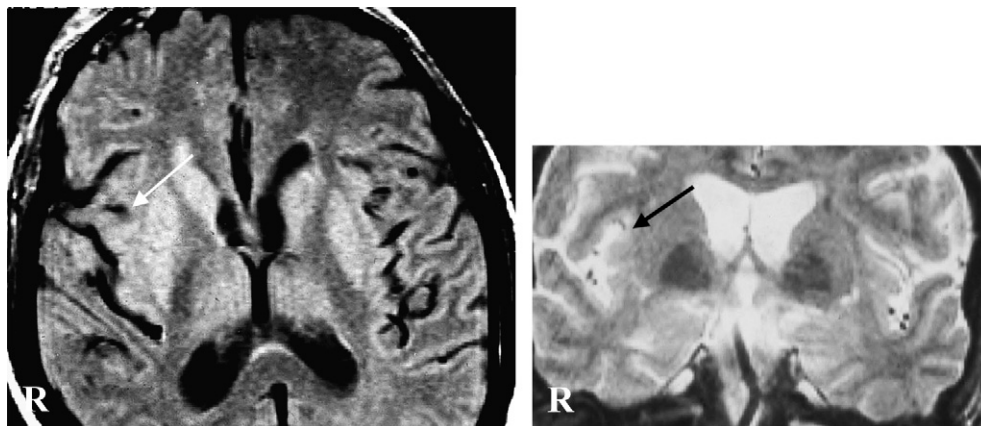


Fig. 1. Axial (left image) and coronal (right image) views of right anterior insular lesion (unenhanced MRI, 1992). Bilateral viscerosomatic abdominal pain (left > right), described as burning and tingling.

in this area could be responsible for the apparently visceral pain experienced by this patient.

A parasagittal view showed a very small change in density in the upper medulla oblongata close to the position of the vestibular nuclear complex, corresponding to a similar left-sided anomaly seen in coronal view. Whether this could be a lesion causing his initial and evanescent left-sided dysequilibrium must remain unanswered.

Unfortunately, this patient was an extremely poor witness. However, he did insist that the pain "in his stomach" was distinct from the pain in his thoracic and abdominal wall. He was constipated, and the pain and tenderness in his left iliac fossa was redolent of a local rather than a neurological cause. So far as could be ascertained, constipation resulted from the medication he had been receiving for his pain; the pain "in the stomach" preceded the constipation.

While we have not been able to find other reports of visceral pain following stroke (central post-stroke pain or CPSP), representation of the viscera in the insular cortex is well-known.¹ A functional magnetic resonance imaging (fMRI) study² and a positron emission tomography (PET) study³ specifically note activation of the anterior insula following painful gastric or lower esophageal distension. Despite the reservations, which are necessary because of this patient's lack of reliability as a witness, it would seem that this may be a genuine case of visceral pain following infarction in the anterior insula.

Acknowledgment

The author is grateful to Dr. Simon Underhill, sometime Consultant Anesthetist at Wrexham Maelor Hospital, who referred the patient to the author, and to the patient's GP, Dr. Pickles of Wrexham.

David Bowsher, MD, ScD, FRCPEd
Pain Research Institute
University Hospital Aintree
Liverpool, United Kingdom

doi:10.1016/j.jpainsymman.2006.07.007

References

1. Jänig W, Haber HJ. Physiology and pathology of visceral pain. *Schmerz* 2002;16:429–446.

2. Strigo IA, Duncan GH, Boivin M, Bushnell MC. Differentiation of visceral and cutaneous pain in the human brain. *J Neurophysiol* 2003;89:3294–3303.

3. Vandenberg J, Dupont P, Fischler B, et al. Regional brain activation during proximal stomach distention in humans: a positron emission tomography study. *Gastroenterology* 2005;128:564–573.

Central Post-Stroke Syndrome Treated with Parenteral Lidocaine

To the Editor:

Central post-stroke syndrome, also known as Dejerine-Roussy Syndrome, is a form of neuropathic pain caused by damage to the central nervous system, usually the thalamus. It is challenging to both diagnose and treat this condition.

We admitted a 45-year-old woman to an inpatient hospice unit for end-of-life care following right temporoparietal hemorrhage with midline shift. Clinical prognosis was 24–72 hours. The patient complained of severe right retro-orbital pain, which was stabbing in nature and was refractory to the opioid infusion she had received at the referring hospital. Her past medical history was pertinent for AIDS with multiple complications, hepatitis B and C, and polysubstance abuse. Physical examination was pertinent for a cachectic-appearing woman with left hemiplegia (arm greater than leg). Dose escalation of infusional hydromorphone to 24 mg/hour resulted in somnolence, but no relief of pain. High dose of dexamethasone (20 mg daily) also provided no relief. Lidocaine 2 mg/kg over 20 minutes followed by 1 mg/kg/hour infusion resulted in complete relief from pain. The hydromorphone infusion was decreased by 50%. Each attempt to reduce the rate of lidocaine infusion resulted in recurrence of the severe pain. Based on the nature of her pain and the response to lidocaine, a diagnosis of central post-stroke syndrome seemed most likely. The patient became more interactive. Family dynamics were tense due to her behavior during her long history of polysubstance abuse. Nevertheless, during this period, partial reconciliation with her estranged daughter occurred. In addition, her sister reported that she