syncope occurred during oxaliplatin infusion, there were none after it was discontinued. Other probable causes such as electrolyte imbalance, ischemic heart disease and central nervous system problems had already been excluded. Given no other identifiable cause of QT-interval prolongation, it is reasonable to speculate about a possible relationship between oxaliplatin and the QT-interval prolongation observed in this patient.

Oxaliplatin is a third-generation platinum compound with activity against colorectal cancer. It has been associated with cell membrane channelopathies [3]. Adelsberger et al. suggested that oxaliplatin may alter sodium (Na^+) channel kinetics in an *ex-vivo* rat nerve preparation [4]. Because the Na^+ channel plays a central role in impulse conduction in cardiac myocytes and cells of the His-Purkinje system [5], alteration of Na^+ channel kinetics is a possible predisposing factor for arrhythmias caused by oxaliplatin. The actual mechanism is unclear and more experimental and clinical evaluations are needed to validate the correlation between oxaliplatin and LQTS. To the best of our knowledge, our patient's LQTS had oxaliplatin as its probable cause: Physicians should be aware of arrhythmias occurring immediately after oxaliplatin infusion.

References

[1] Bazett HC. An analysis of the time-relation of electrocardiograms. Heart 1920;7:353–70.
[2] Khan IA. Long QT syndrome. Diagnosis and management. Am Heart J 2002;143:7–14.
[3] Ng M, Cunningham D, Norman AR. The frequency and pattern of cardiotoxicity observed with capecitabine used in conjunction with oxaliplatin in patients treated for advances colorectal cancer (CRC). Eur J Cancer 2005;41:1542–46.
[4] Adelsberger H, Quasthoff S, Grosskreutz J, Eckel F, Lersch C. The chemotherapeutic oxaliplatin alters voltage-gated Na+ channel kinetics on rat sensory neurons. Eur J Pharmacol 2000;406:25–32.
[5] Grant AO. Molecular biology of sodium channels and their role in cardiac arrhythmias. Am J Med 2001;110:296–305.

Regression of cervical spinal cord compression in a patient with chordoma following treatment with cetuximab and gefitinib

OLA LINDÉN[1], LARS STENBERG[2], & ELISABETH KJELLÉN[3]

[1]*Department of Oncology, Lund University Hospital, Lund, Sweden,* [2]*Department of Diagnostic Radiology, Lund University Hospital, Lund, Sweden, and* [3]*Department of Oncology Lund, Sweden and Department of Radiation Sciences Umeå University, Umeå, Sweden*

To the Editor

A previously healthy woman born in 1932 presented in 1996 with a chordoma of the second cervical vertebra, dislocating the spinal cord. She underwent laminectomy and occipitocervical fixation. She received radiotherapy at a dose of 50 Gy in 28 fractions. In February 2006 and April 2007 she was reoperated due to deteriorating ability to walk.

The second operation did not bring about any improvement, and in August 2007 her condition worsened with incontinence, increasing right-sided weakness and her right hand became completely useless. An MRI showed spinal cord compression and a 6.5×5.5 cm large right cervical mass.

Based on a case report of successful treatment of a patient with metastatic chordoma using cetuximab and gefitinib [1], the decision was made to offer the patient treatment with these drugs. The patient was started on cetuximab weekly and gefitinib 250 mg daily. After a week she reported the return of continence and after two weeks she was able to walk using a walking frame and to use her right hand to cut while eating. At the 4-week check-up the therapy was temporally stopped due to facial acne. The therapy was resumed a couple of weeks later

Correspondence: Ola Lindén, Department of Oncology, Lund University Hospital, 221 85 Lund, Sweden. E-mail: ola.linden@med.lu.se

(Received 11 April 2008; accepted 10 June 2008)

ISSN 0284-186X print/ISSN 1651-226X online © 2009 Informa UK Ltd. (Informa Healthcare, Taylor & Francis AS)
DOI: 10.1080/02841860802266672

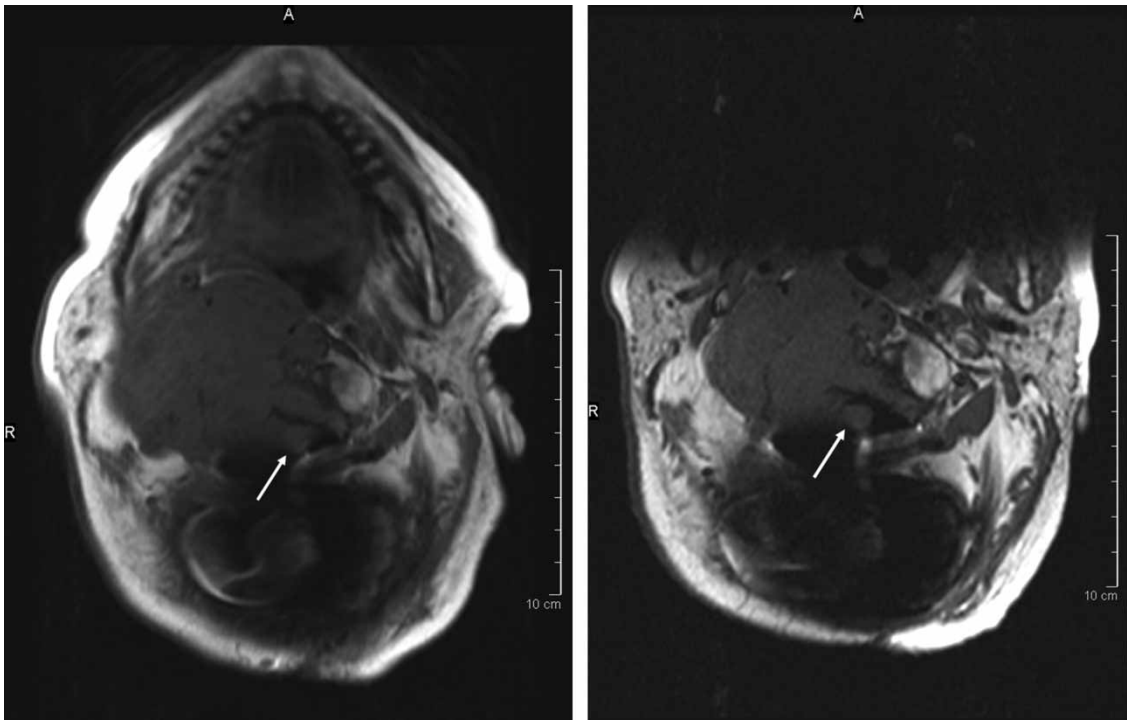

Figure 1. MRI scan of the tumour before and after treatment with cetuximab and gefitinib. The arrows indicate the spinal cord.

and after 4 month of treatment an MRI scan showed regression of the spinal cord compression and the tumour had shrunk to 4.9 × 4.1 cm (see Figure 1).

Discussion

The standard treatment for chordoma is surgery and radiotherapy. There is no recognised effective chemotherapy. The above cited case using two different agents to target the epidermal growth factor receptor, EGFR [1], was considered to provide a basis for offering our patient the same treatment [2]. The present case shows that neurological function may return with the regression of spinal cord compression, using targeting drugs. The fast return of function may be related to the way in which targeting drugs work. Unlike radiotherapy which, to a large extent, relies on post-mitotic apoptosis and may thus allow the tumour to grow before it shrinks [3], targeting drugs induce apoptosis in tumour cells [2] without their going through mitosis. Furthermore, since radiotherapy is fractionated a tumour may grow until a sufficient dose to halt it has been absorbed. Minimal tumour growth is usually of no importance in settings where neurological function is not compromised, however, when it is, an extra mm in tumour size may be extremely undesirable. Targeted therapy is thus theoretically an attractive option when neural function is compromised. This observation may encourage research into targeted drugs in spinal cord compression due not only to chordoma, but more widely, as indicated by a report of renal cell carcinoma [4].

References

[1] Hof H, Welzel T, Debus J. Effectiveness of cetuximab/gefitinib in the therapy of a sacral chordoma. Onkologie 2006;29: 572–4.

[2] Huang S, Armstrong EA, Benavente S, Chinnaiyan P, Harari PM. Dual-agent molecular targeting of the epidermal growth factor receptor (EGFR): Combining anti-EGFR antibody with tyrosine kinase inhibitor. Cancer Res 2004;64:5355–62.

[3] Thomlinson RH. Measurement and management of carcinoma of the breast. Clin Radiol 1982;33:481–93.

[4] Trinh QD, Cardinal E, Gallina A, Perrotte P, Saad F, Karakiewicz PI. Sunitinib relieves renal cell carcinoma spinal cord compression. Eur Urol 2007;51:1741–3.