CASE REPORT

Neurological abnormalities associated with mobile phone use

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Dysaesthesiae of the scalp after mobile phone use have been previously reported but the pathological basis of these symptoms has been unclear. We report finding a neurological abnormality in a patient after prolonged use of a mobile phone. He had permanent unilateral dysaesthesiae of the scalp, slight loss of sensation, and abnormalities on current perception threshold testing of cervical and trigeminal nerves. A neurologist found no other disease. The implications regarding health effects of mobile phones and radio-frequency radiation is discussed.

Key words: Dysaesthesiae; mobile phone; neurological; radio-frequency radiation.

INTRODUCTION

Dysaesthesiae of the scalp after mobile phone use have been reported previously.1 Forty respondents from diverse occupations described unpleasant sensations such as a burning feeling or a dull ache mainly occurring in the temporal, occipital or auricular areas. The symptoms often began minutes after beginning a call, but could come on later during the day. The symptoms usually ceased within an hour after the call, but could persist longer. Respondents clearly distinguished these symptoms from headaches. Similar reports have come from Scandinavia.2 The pathological basis of these symptoms has been unclear. This paper reports on the findings of a neurological abnormality after mobile phone use.

CASE REPORT

A 72-year-old businessman used a GSM (digital) mobile phone on the right side. He had sometimes noticed unusual, brief sensations on the right scalp after use. Fifteen months prior to seeking medical advice, he had two mobile phone calls of nearly an hour each on consecutive days, following which he developed persistent symptoms, which he likened to a bruised feeling. It was felt over the right parietal, temporal, auricular, and cheek areas and into the neck. The symptoms were worsened by exposure to the sun or wind on the right scalp, but not the left, and were not affected by the use of an ordinary telephone. Since the symptoms began the patient has used a hands-free kit with his mobile phone. He has not used a walkie-talkie, nor is he an amateur radio operator. He wears spectacles.

He distinguished these symptoms clearly from headaches. He has experienced neck problems for 8 years and has a history of whiplash, which causes frontal headaches, but these are different in character and respond to chiropractic therapy. He described the new symptoms as being felt ‘on the head’, not ‘in the head’.

The patient has no balance or hearing disturbance. He has no other paraesthesiae or loss of dexterity. He saw a neurologist who, on extensive examination, found no abnormality. A cranial CT scan was normal. During the previous 6 weeks, he had flashes in the right eye at night. An eye specialist diagnosed ‘vitreous shrinkage and detachment from the retina’.

He has a history of tachycardia and is taking diltiazem, as well as simvastatin, which has controlled his cholesterol levels. He has a history of dermatitis on the legs and torso and uses betamethasone cream.

On examination, he sensed cotton wool less well on the right than the left side of his face and cheek. There was no lesion or tender point on the scalp.

Neurophysiological testing was performed by using a Neurometer CPT/C. This device is a variable constant current sine wave stimulator which uses three test frequencies, 2000 Hz, 250 Hz and 5 Hz corresponding to Aβ, Aδ and C-fibres, respectively.3 The test sites were selected within the affected trigeminal and C3 dermatomes, and corresponding locations on both normal and symptomatic sides were tested. The stimulus was initially
increased until a sensation was reported and then short stimuli (2–5 s) were applied at progressively lower current amplitudes until a minimal threshold for constant detection was determined. The device has a dummy switch to allow the on/off status of the machine to be concealed from the patient during determination of an approximate threshold level. After this level was determined a double blind, forced choice paradigm was used to confirm the minimal threshold for perception. Impaired current perceptual acuity was found for all sensory components of the right trigeminal and C3 nerves (Table 1). The current perception thresholds (CPT) and noxious CPT (NCPT) values obtained for the left C3 and trigeminal skin zones were all within the normal reference range for the database. On the right, CPT for all three test frequencies were elevated above the normative values 95 percentile for the sites tested. Although the noxious current perception thresholds (NCPT) were also higher for both right sided test sites than the corresponding left side sites, these differences were not significant.

The right side CPT's were markedly higher than the values on the asymptomatic left side. These findings are consistent with the patient's significant hypo-aesthesia and hypoalgesia.

**DISCUSSION**

The patient was diagnosed as having neuropathic pain (Complex Regional Pain Syndrome, type 2). The abrupt onset of the dysaesthesias after prolonged mobile phone calls, their one-sided localization, and involvement of different nerve roots within the distribution of radio-frequency fields from the phone found on CPT testing, argue for a causal relation to the phone rather than a medical cause. This is further supported by the absence of other abnormal findings by a neurologist.

The Neurometer CPT results in Table 1 are significantly elevated for all three test frequencies used at both test sites on the affected (symptomatic) side of the face. The CPT values are expressed as mA, all those on the right side are higher by a factor that varies from 2.8- to 5.8-fold. This corresponds to a reduced current perception acuity, or hypo-aesthesia for all classes of sensory nerve fibres in these tested skin zones. Because dysaesthesia and pain are usually associated with disturbance or modulation in the balance of sensory input via the different classes of fibres, the patient's symptoms were consistent with his subtle sensory neurological deficits.

The retinal symptoms arose from shrinking of the vitreous. Whilst this is most likely to be an age-related change, the occurrence ipsilateral to his neurological changes raises the possibility of an effect by the radio-frequency radiation in the orbit. Visual symptoms associated with mobile phone use were noted in the original report. The similarities of the symptoms on this patient's scalp to those reported in the original cases, suggests a similar neurological basis for the others even though they were transient. This observation of a subtle neurological abnormality may be helpful in understanding recent reports of sleep disturbance, raised blood pressure and cognitive effects after mobile phone field exposure. It also provides a possible mechanism regarding case reports of prolonged fatigue, malaise, dysaesthesiae and other non-specific symptoms in workers who have been overexposed to radio-frequency radiations, a condition sometimes termed 'microwave sickness'.

The occurrence of the nerve injury on the scalp also questions the current view that all health effects of radio-frequency radiation (>10 MHz) are due to thermal (heating) effects. The energy emitted by a mobile phone is low and the scalp has a good blood supply to keep the head cool, which discounts the plausibility of this mechanism. In addition, if simple heating were the mechanism then the patient's exposures to many Australian summers should have caused the lesion previously. Therefore the case is evidence for non-thermal mechanisms of injury in humans from radio-frequency radiation and its modulations. It is considered that exposure to the head from mobile phone radiation should be minimized by using short call times and the use of hands-free or other devices.

**REFERENCES**


**Table 1. AC current perception thresholds (mA) by Neurometer CPT/C® (ratio R:L)**

<table>
<thead>
<tr>
<th>AC frequency and N fibre type</th>
<th>L. mastoid/C3 dermatome</th>
<th>R. mastoid/C3 dermatome</th>
<th>L. preauricular/Trigeminal</th>
<th>R. preauricular/Trigeminal</th>
</tr>
</thead>
<tbody>
<tr>
<td>2000 Hz, Aβ</td>
<td>0.49</td>
<td>1.72 (1.35 x)</td>
<td>0.38</td>
<td>1.90 (1.5 x)</td>
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<tr>
<td>250 Hz, Aβ</td>
<td>0.08</td>
<td>0.26 (1.325 x)</td>
<td>0.046</td>
<td>0.27 (1.58 x)</td>
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<tr>
<td>250 Hz, NCPT</td>
<td>0.49</td>
<td>0.66</td>
<td>0.38</td>
<td>0.63</td>
</tr>
<tr>
<td>5 Hz, C-fibres</td>
<td>0.06</td>
<td>0.17 (1.28 x)</td>
<td>0.046</td>
<td>0.24 (1.52 x)</td>
</tr>
</tbody>
</table>

L mastoid/C3 dermatome

R. mastoid/C3 dermatome

L. preauricular/Trigeminal

R. preauricular/Trigeminal

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