Acoustic Neuroma

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Medical history, cigarette smoking and risk of acoustic neuroma: An international case-control study

Acoustic neuroma (AN) incidence has increased very significantly in the past decade; this paper explores some of the possible risk factors.

Thus far the only confirmed external cause of AN is exposure to intense ionising radiation. AN is usually unilateral unless it is associated with neurofibromatosis, an inherited disorder. There has been increased speculation that mobile phones could be a cause but research results usually find that there is no significant association.

The present study attempts to identify statistical associations between AN and some common factors such as smoking and gender. Further study of AN and emfs should take any such factors into account. The study was of case (n=563) control (n=2703) design and was set in 5 north European countries. Eligible cases were individuals diagnosed with acoustic neuroma between September 1999 and February 2005 aged between late teens and 60 to 70. Controls, who had no history of any form of brain cancer, were frequency matched to age, gender and region or at random from GP registers. Exposure data was obtained by interview.

Participation rates were 77% for cases and 50% for controls.

There was no significant association of AN with allergies (asthma, hay fever, eczema, food allergy) or allergy medications.

Those with a more than 10 year history of epilepsy were more likely to have AN OR = 4.1 (95% CI = 1.9 to 8.8) and the association strengthened where antiepileptic medication had been taken. Although more likely to have brain tests the reason these people with epilepsy were diagnosed with AN was because each had been observed to have signs of tumour. Around 0.5% of the UK population has epilepsy, but those from poor backgrounds are more likely to have it. The received wisdom is that head injuries received at birth, head injury, brain tumours and alcoholism are risk factors for epilepsy.

Head injury, involving loss of consciousness or hospitalisation or both, was not a risk factor for diagnosis of AN.

Other brain tumours did not increase the risk of AN.

In women, having had a baby was associated with increased risk OR = 1.7 (95% CI = 1.1 to 2.6) but risk did not vary with age of first child, number of children, breast feeding or time since last live birth. There was no association with ever use of oral contraceptives or hormone treatments. The risk increased if the woman had ever smoked.

Cases were significantly less likely to have ever been regular cigarette smokers up to 1 year prior to the reference date than controls OR = 0.7 (95% CI = 0.6 to 0.9) but this was only significant for current smokers or those who had only given up within the past 5 years.

Comment
The study was of a significant size and benefited from certainty as to diagnosis. Much of the information sought could be validated but in any case was unlikely to be mis-remembered (with the usual caveats for smoking history).

It could be that smoking really does have a protective effect or that smokers are less likely to be diagnosed for some other reason. The evidence in this research was that the neuroma size at diagnosis was not larger in smokers as would be expected for delayed diagnosis but, size at diagnosis is very variable.

Both smoking and pregnancy could indicate a hormonal effect.

Epilepsy, smoking and parity should be assessed in any future research into causes of AN.

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