tics of myelomatosis. The exact diagnosis can be established through biopsy, thus leading to correct treatment.  

EMILIA KERTY  
PER H NAKSTAD  
Department ofNeurology, Rikshospitalet, The National Hospital, Oslo 1, Norway  
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References


Neurotoxicity of halogenated hydroxyquinolines

Sir: In 1979 we (Baumgartner et al, J Neurol Neurosurg Psychiatr 1979;42:1073) recorded the results of a clinical analysis of cases of suspected hydroxyquinoline neurotoxicity reported from outside Japan between 1935 and 1977. The criteria employed in this assessment were fully discussed. Of the 222 cases from 201 reports, 42 were considered probably to have been related to the administration of clioquinol or similar compounds. In 69 there was a possible relationship. In 42 a relationship was thought to be unlikely, and in 59 insufficient information was available for assessment.

We have subsequently analysed a further 137 cases reported between 1977 and April 1983, although in a number of these the drug had been administered considerably before 1977. In these further cases, a relationship to the administration of hydroxyquinolines was considered probable in 27, possible in 28 and unlikely in 17. Insufficient information was available in 31, and 14 were excluded from evaluation because the symptoms were not neurological or because documentation of hydroxyquinoline intake was not presented.

Combining the assessments from the two series yields a total of 359 reported cases with the following attributability distribution: probable 69, possible 97, unlikely 59, no relationship 30, insufficient information 104. As was emphasized in our previous report, it is striking that the number of cases reported from outside Japan is of quite a different order from the large numbers encountered in that country before clioquinol sales were stopped in 1970. The reason for this disparity remains uncertain, but the greater consumption of clioquinol in Japan is likely to have been the most important factor.

In our assessment of the cases reported since 1977, the semielogical categorisation again included cases of acute fully reversible toxic encephalopathy with amnesia as a prominent feature. This usually occurred following the intake of a large amount of the product over a short period. Also included were cases of isolated optic atrophy of subacute or insidious onset, most commonly in children. Thirdly there were cases of myelopathy, usually of subacute onset, either in isolation or accompanied by optic neuropathy. It is of interest that no cases of peripheral neuropathy were identified in the probable category, either in isolation or associated with optic neuropathy or myelopathy. It is now clear that the neurotoxic effects of the halogenated hydroxyquinolines are substantially confined to the central nervous system. The term subacute myeloptic neuropathy (SMON) is therefore probably a misnomer.

G BAUMGARTNER  
O GILLAND  
H KAESER  
CA PALLIS  
F CLIFFORD ROSE  
HH SCHAUERBURG  
PK THOMAS  
NH WADIA

*Neurologische Universitätsklinik, Kantonsst, Rämistrasse 100, Zürich, Switzerland.
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G Baumgartner, O Gilland, H E Kaeser, C A Pallis, F C Rose, H H Schaumburg, P K Thomas and N H Wadia

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