Short report

CT scan prediction of late post-traumatic epilepsy

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SUMMARY Out of 233 patients admitted for head trauma during 1977-1978, 93 had a CT scan examination within the first 48 hours. Forty-nine of these had at least one clinical risk factor for post-traumatic epilepsy. Ten of this group developed post-traumatic epilepsy. In all cases early CT scan showed focal brain damage, which was related more significantly to post-traumatic epilepsy than to risk factors.

It is necessary to know whether a patient with head trauma should be treated with prophylactic anticonvulsant therapy. There have been many studies on large series of civilian and war head trauma with the purpose of establishing early factors predictive of post-traumatic epilepsy. With closed head trauma, loss of consciousness for longer than 24 hours, early seizures, focal neurological signs, depressed fracture of the skull and intracranial haematoma, are associated with a higher risk of post-traumatic epilepsy. The aim of the present study was to see whether early CT scan could be predictive of post-traumatic epilepsy.

Methods

The study was performed on the patients admitted for head trauma during 1977-1978 and alive on 1 January 1982. We excluded the following cases: (1) patients with previous epilepsy or who were taking antiepileptic drugs for any reason at the time of head trauma, (2) patients who before or after head trauma suffered another possible cause of seizure (stroke, metabolic encephalopathy, another injury to the head), (3) patients discharged with prophylactic anticonvulsant therapy. There were 233 such patients. We interviewed all the patients or their relatives by telephone and whenever they mentioned persisting symptoms we personally questioned the patients or eye-witnesses. When at least one seizure had appeared after the first week from the time of the head trauma, post-traumatic epilepsy was considered to have occurred. We divided head trauma into two groups: (1) severe, when there was a loss of consciousness longer than 24 hours, focal neurological signs, early seizures (within first week), depressed skull fracture, intracranial haematoma or brain contusion (radiologically or surgically diagnosed), (2) mild-moderate in the remainder. Ninety-three patients had been examined by CT scan within 24 h (86 patients) or 48 h (seven patients) from the time of trauma. We divided CT scan pictures into six groups: (1) normal, (2) brain swelling, (3) intracranial haematoma (subdural or epidural), (4) focal hypodensity, (5) intracerebral haemorrhage, (6) intracerebral haemorrhage associated with extracerebral haematoma.

Results

INCIDENCE OF POST-TRAUMATIC EPILEPSY

Eighty-six patients had had severe and 147 mild or moderate head trauma. Eleven patients (5%) out of 233 developed post-traumatic epilepsy. In the group with severe head trauma 11 (13%) developed post-traumatic epilepsy, whereas not one of the mild-moderate group did.

CT SCAN VERSUS CLINICAL FACTORS VERSUS POST-TRAUMATIC EPILEPSY

Early CT scan examination had been performed on 36 patients of the mild-moderate group and on 57 of the severe group (including 10 of the 11 patients who developed post-traumatic epilepsy). In the table, CT scan findings are compared to clinical risk factors of post-traumatic epilepsy. Obviously, intracranial haematoma or brain contusion are not included in the
clinical risk factors but are considered as CT scan parameters. Thirty-one patients had focal cerebral damage shown by CT scan, including five with focal hypodensity and 26 with intracerebral haemorrhage. Ten of them (32%) developed post-traumatic epilepsy: one had focal hypodensity and nine intracerebral haemorrhage.

Three out of 18 patients with intracerebral haemorrhage alone developed post-traumatic epilepsy, whereas six out of eight with intracerebral haemorrhage associated with extracerebral haemorrhage did so. None of 11 patients with extracerebral haematoma alone developed post-traumatic epilepsy. Forty-nine patients had at least one clinical risk factor; 28 had CT scan evidence of parenchymal damage and nine (32%) of them developed post-traumatic epilepsy. Statistical analysis shows that in the group of patients with at least one clinical risk factor the risk of post-traumatic epilepsy related to parenchymal damage is significantly higher (p < 0.02, chi square test, Yates correction).

Discussion

The follow up period in our series varied from 3 to 5 years. Therefore, we believe we have detected most of the patients who might develop post-traumatic epilepsy, according to epidemiological and non-epidemiological studies. In our study the incidence of post-traumatic epilepsy in the whole series and in the severe head trauma group agree with the value found by Annegers et al. As regards the incidence of post-traumatic epilepsy related to each single clinical risk factor, our cases are too few to make a comparison with other reports. Our findings are grossly in accordance with the expected frequency of post-traumatic epilepsy for each group, particularly for the higher risk of post-traumatic epilepsy when two or more risk factors are present. The most important finding in our study is that only patients with CT scan evidence of a focal cerebral lesion developed post-traumatic epilepsy. The risk of post-traumatic epilepsy seems particularly high when intracerebral haemorrhage and extracerebral haematoma are associated, suggesting the presence of a cerebral laceration.

No previous reports have been published on CT scan in the prediction of post-traumatic epilepsy. However our findings agree with CT scan studies which show a high frequency of focal lesions of the brain in series of patients already suffering from post-traumatic epilepsy. Focal parenchymal damage detected by early CT scan seems to be a definite risk factor of post-traumatic epilepsy. Furthermore, if our results are confirmed, it should be possible to select candidates for prophylactic anticonvulsant therapy on the basis of early CT scan only.

References

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