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<td><strong>Author(s)</strong></td>
<td>Fong, GCY; Fong, KY; Mak, W; Tsang, KL; Chan, KH; Cheung, RTF; Ho, SL; Ho, WY</td>
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Postictal psychosis related regional cerebral hyperperfusion

G C Y FONG, K Y FONG, W MAK, K L TSANG, K H CHAN, R T F CHEUNG, S L HO and W Y HO

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LETTERS TO THE EDITOR

Postictal psychosis related regional cerebral hyperperfusion

Postictal psychosis is a known complication of complex partial seizure in particular temporal lobe epilepsy. It usually runs a benign and self-limiting course. A postictal phenomenon with focal cerebral hypofunction (similar to Todd’s palsy), rather than ongoing seizure activity, has been postulated. Surface EEG is either normal or showing non-specific slow waves. Hence, antipsychotic medications are prescribed instead of antiepileptic drugs. Until recently, the pathogenic mechanisms have remained unknown. In this communication, we report on two patients with postictal psychosis, during which a cerebral SPECT study showed a hyperperfusion signal over the right temporal lobe and contralateral basal ganglion. As hyperperfusion in ictal cerebral SPECT is closely linked to epileptic activities, our findings support a contrary explanation for postictal psychosis.

Prolonged video-EEG telemetry study was performed in patients who underwent presurgical evaluation for epilepsy surgery. Antiepileptic drugs were withdrawn to facilitate seizure recording. A diagnosis of temporal lobe epilepsy was based on analysis of the electroclinical events and, if applicable, postoperative outcome after anterior temporal lobectomy. Psychosis was diagnosed according to the fourth edition of the diagnostics and statistical manual of mental disorders (DSM-IV) criteria of brief psychotic disorders without marked stressor. HMPAO-SPECT was performed during the psychotic period, which ranged from 2–4 days after the last seizure. Interictal cerebral SPECT, brain MRI, and a Wada test were performed as part of presurgical evaluation.

Patient 1 was a 34 year old Chinese woman with complex partial seizures since the age of 18. Her seizure control was suboptimal on a combination of antiepileptic drugs. Brain MRI showed a small hippocampus on the right. Interictal EEG showed bilateral temporal sharp waves and ictal recordings confirmed a right temporal epileptogenic focus. A Wada test confirmed right hippocampal memory dysfunction. Six hours after her last secondary generalised tonic-clonic seizure after video-EEG telemetry, she began to develop emotional lability, talking nonsense, motor restlessness, and auditory hallucination. A cerebral SPECT study was performed at day 4 after her last seizure. Her psychotic features persisted although she was taking antipsychotic medication (pimozide). Cerebral SPECT showed a clear hyperperfusion signal over the right lateral temporal neocortex and contralateral basal ganglion. An interictal cerebral SPECT study was repeated at 4 weeks after postictal psychosis which showed a complete resolution of hyperperfusion signal in the right temporal lobe and basal ganglia. Anterior temporal lobectomy was performed and she became seizure free after surgery.

Patient 2 was a 44 year old man with intractable complex partial seizures since the age of 30. His seizures were intractable to multiple antiepileptic drugs. Brain MRI showed left hippocampal sclerosis. Interictal cerebral SPECT showed a relative hypoperfusion area over the left hemisphere. Interictal surface EEG was non-lateralising but ictal EEG disclosed a right hemispheric onset. On withdrawal of antiepileptic drugs, seven complex partial seizures with secondary generalised tonic clonic seizures were recorded within a period of 72 hours. His usual antiepileptic drugs were then restarted. Thirty hours after his last secondary generalised tonic-clonic seizure, he began to develop emotional lability, talking nonsense, restlessness, auditory hallucination, persecutory delusion, and delusion of superstition. Cerebral SPECT study, performed 2 days later while his psychotic features persisted, showed two relative hyperperfused areas over the right temporal neocortex and contralateral basal ganglion in addition to the original hypoperfused area over the left hemisphere. An antipsychotic agent (thioridazine) was...
started after the cerebral SPECT. His psychotic symptoms resolved 2 weeks later with full recovery.

Cerebral SPECT performed during the interictal period (IP) and during postictal psychosis (PP) were analysed visually and areas of hyperperfusion were identified. Quantitative data at regions of interest (ROIs) were measured on coronal and axial slides containing basal ganglia (BG), mesial (MT), and lateral (LAT) temporal lobe structures. Asymmetry index (ASI) was calculated as (ROI focus−ROI contralateral)/ROI focus×ROI contralateral)×100%. We set an arbitrary change of ASI >100% to be significant. As there were only two seizures, statistical testing was not performed.

Both patients showed postictal psychosis and had a regional increase in rCBF over the right temporal neocortex and the left basal ganglia compared with their interictal study (figure). Quantitative analysis for patient 1 showed changes of ASI during IP and PP over right MT was +75% (+6.4467 to −1.6528); over the right LT was +116.7% (1.0797 to 12.5576); and over the left BG was +206.8% (+2.0737 to 2.21574). Quantitative analysis for patient 2 showed changes of ASI during IP and PP over right MT was −3.8% (13.14217 to 12.64158); over right LT was +178.6% (10.4696 to 18.7057); and over left BG was +155.9% (−5.85556 to 3.27522).

Postictal psychosis is a distinct clinical event associated with temporal lobe epilepsy. The diagnosis of postictal psychosis requires a close temporal relation between bouts of complex partial seizures and the onset of psychosis. The psychosis usually develops after a cluster of complex partial seizures precipitating a postictal psychosis (PP). Both patients showed postictal psychosis (PP) were analysed visually and had a regional increase in rCBF over the right temporal neocortex and the left basal ganglia compared with their interictal study (figure). Quantitative analysis for patient 1 showed changes of ASI during IP and PP over right MT was +75% (+6.4467 to −1.6528); over the right LT was +116.7% (1.0797 to 12.5576); and over the left BG was +206.8% (+2.0737 to 2.21574). Quantitative analysis for patient 2 showed changes of ASI during IP and PP over right MT was −3.8% (13.14217 to 12.64158); over right LT was +178.6% (10.4696 to 18.7057); and over left BG was +155.9% (−5.85556 to 3.27522).

Oncofetal matrix glycoproteins in cerebral arteriovenous malformations and neighbouring vessels

Cerebral arteriovenous malformations (AVMs) are thought to be congenital lesions exhibiting features of either mature vascular walls or embryonal anastomotic plexuses. It is generally assumed that changes in size are dependent on enlargement of the venous compartment, organisation in the setting of microhaemorrhages, and gliosis. However, recent findings are consistent with the hypothesis of ongoing angiogenesis. Previous research from this laboratory disclosed that peculiar isoforms of fibronectin (FN) and tenascin (TN) typically occur in fetal and neoplastic tissues. These isoforms are a blend of structurally different glycoproteins that result from alternative splicing of the primary transcript and are mainly expressed in the extracellular matrix. Their expression is undetectable in normal adult tissues, with the exception of the vessels in the regenerating endometrium. To gain further insight into the pathobiology of the AVMs the present report sought to ascertain whether these lesions also express oncofetal FN and TN isoforms.

Tissue samples were obtained after neurosurgical excisions of ruptured AVMs. All 10 patients had experienced an intracerebral haemorrhage as the first clinical manifestation of their disease. There was no drug history before bleeding. Control specimens from two right gyri recti and one cerebellar tonsil were obtained, respectively, from operations for ruptured aneurysms of the anterior communicating artery or for Arnold Chiari disease.

Immunohistochemical evaluations were performed on 5 μm thick cryostat sections using a protocol reported previously. Owing to the limited amount of available material, only in a few cases was some fresh tissue retained to allow western blots. Distribution of FN and TN isoforms was investigated using three monoclonal antibodies (mAbs) or two Ab fragments, obtained by phage display technology, respectively. These Abs, prepared in our laboratory, were found to work on fresh frozen material. According to the previous characterisations the BC-1 mAb and the TN-11 Ab fragments are specific for isoforms occurring almost exclusively in fetal tissues and in tumours, with the recognised TN isoform being typically associated with anaplastic gliomas (table). The antibodies were blocked using the specific antigens. The antigens were recombinant protein containing the epitope produced in E Coli. For the mAb BC-1 we used the recombinant protein containing the type-III repeats 7B–8–9. For the mAb Ab-4 we used the recombinant protein containing the type-III repeats 2–8. For the recombinant antibodies TN-11 and TN-12 the recombinant type-III repeat C and the recombinant fragment containing the BG-12 were used respectively.

All 10 AVMs were found to contain large amounts of FN and TN, as shown by intense immunostaining with the use of the IST-9 / IST-4 mAbs and the TN-11 Ab fragment. The staining was localised either in the endothelium or the subendothelial layer. A positive response was found in several artery-like vessels and in a few vessels with thinner walls surrounding the angiomatous nidus. In all these cases the wall of several vessels exhibited intense staining with the use of the TN-11 Ab fragment. Using the BC-1 mAb some of these vessels exhibited some staining (figure). In the control specimens (brain and cerebellum) both the FN isoform containing the ED-B sequence (ED-B+FN) and the type-III repeat C TN isoform were absent, despite the widespread distribution of total FN and TN in the vascular walls.

Characterisation of the employed Abs and distribution of the recognized isoforms.

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<th>Anti-FN mAb⁴</th>
<th>Anti-TN Ab fragments⁴</th>
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<tr>
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<td>IST-9</td>
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<tr>
<td>Recognised isoforms</td>
<td>Total FN</td>
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<tr>
<td>Distribution of the isofoms (t)</td>
<td>Widespread</td>
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<tr>
<td>IST-4</td>
<td>IST-9</td>
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<tr>
<td>Total FN</td>
<td>Widespread</td>
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<tr>
<td>Type III repeat C Isoform</td>
<td>Absent in several types of malignancies</td>
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