ORIGINAL ARTICLE

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Temporal Lobectomy with Delayed Amnesia Following a New Lesion on the Other Side

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ABSTRACT: ***Purpose:*** To describe a delayed severe complication of temporal lobectomy for intractable epilepsy. ***Method:*** A case of amnesia occurring 24 years after surgery is described and five similar cases from the literature reviewed. ***Results:*** Mean age at surgery (5 right) was 40 years (19-62 years), 3 female. Four of five tested had impaired visual and verbal memory preoperatively but not sufficient to contraindicate surgery. Pathology was mesial temporal sclerosis in 3, 1 cavernoma, 1 dysembryoplastic neuroepithelial tumor (DNET) and 1 normal. Postoperatively, four were seizure free 3-12 years off medication and two continued with seizures. There was no unexpected postoperative memory change until incapacitating anterograde amnesia developed 1-24 years after surgery. In five patients, including ours, this followed definite or possible status epilepticus with new mesial temporal sclerosis on the opposite side in the four that were investigated by MRI. One patient developed a glioblastoma in the opposite temporal lobe. ***Conclusion:*** Continuing or late recurrence of seizures from the remaining temporal lobe after temporal lobectomy can result in incapacitating amnesia if status epilepticus occurs. Other new lesions on the opposite side to surgery can have the same effect.

RÉSUMÉ: Lobectomie temporale avec amnésie tardive suite à une nouvelle lésion contra-latérale. ***Objectif :*** Le but de l'étude était de décrire une complication tardive sévère d'une lobectomie temporale pour une épilepsie résistante au traitement médical. ***Méthode :*** Nous décrivons un cas d'amnésie survenue 24 ans après une chirurgie ainsi que 5 cas similaires publiés dans la littérature. ***Résultats :*** L'âge moyen au moment de la chirurgie (5 lobectomies droites) était de 40 ans (19 à 62 ans) et 3 étaient des femmes. Quatre des 5 patients évalués avaient une altération de la mémoire visuelle et verbale avant la chirurgie, mais elle n'était pas suffisamment sévère pour constituer une contre-indication à la chirurgie. Trois patients étaient atteints de sclérose temporale mésiale, 1 avait un cavernome, 1 avait une tumeur neuroépithéliale dysembryoplasique et 1 était normal. Après la chirurgie, 4 patients sans médication n'ont pas eu de crises au cours d'une période d'observation qui variait de 3 à 12 ans et 2 avaient encore des crises. Aucun changement inattendu au niveau de la mémoire n'a été observé après la chirurgie jusqu'à ce que les patients présentent une amnésie antérograde invalidante de 1 à 24 ans après la chirurgie. Chez 5 patients, dont notre patient, ceci a suivi un état de mal épileptique avéré ou possible avec une nouvelle sclérose temporale mésiale du côté opposé objectivée chez les 4 patients qui ont subi une IRM. Un patient a présenté un glioblastome du lobe temporal contra-latéral. ***Conclusion :*** Une persistance ou une récidive des crises épileptiques provenant du lobe temporal restant après une lobectomie temporale peut provoquer une amnésie invalidante si le patient présente un état de mal épileptique. De nouvelles lésions du côté opposé à celui de la chirurgie peuvent avoir le même effet.

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It is well known that bilateral mesial temporal lesions result in severe memory impairment that has been known as global amnesia or more recently the temporal lobe amnesic syndrome[1]. This is defined as severe anterograde amnesia with preservation of other cognitive functions. The most famous case of amnesia followed bilateral resection of mesial temporal structures (part of hippocampus and amygdala) is HM, whose memory problem was meticulously studied and documented by Scoville and Milner[2,3]. This work resulted in the recognition of the role of the hippocampus in memory function, the end of bilateral temporal lobectomy as a treatment for epilepsy and psychiatric conditions, and the advent of careful preoperative memory examination in the assessment of epilepsy patients prior to temporal lobectomy[1].

Rarely severe anterograde amnesia has immediately followed anterior temporal lobectomy when an unrecognized lesion or memory impairment was present in the other temporal lobe[1,4]. Underappreciated is the potential for late development of amnesia long after temporal lobe surgery if a subsequent insult occurs to the remaining temporal lobe[5-8]. We present a case of severe amnesia that occurred 24 years following a successful right anterior temporal lobectomy (ATL) for medically refractory temporal lobe epilepsy (TLE) and review five other cases in the literature with similar late onset memory impairment.

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Case History

A 48-year-old right handed woman began having complex partial seizures at seven years-of-age on the background of a one hour generalized febrile convulsion without focal features at age three months. Developmental history was subsequently normal. Seizures began with a rising epigastric sensation and an indescribable taste or odor that would progress to loss of awareness with staring, lip smacking, left arm and hand automatisms with turning of the head to the left, lasting two to three minutes. Secondary generalization occurred rarely. Postictally she would feel confused and tired with a headache, but without focal deficits including aphasia. Frequency was approximately ten per month despite trials of six different anti-seizure drugs. Physical examination was normal. She graduated high school and did secretarial work.

She was investigated for surgery. Electroencephalogram (EEG) demonstrated spikes and seizures arising from the right anterior temporal lobe, with no abnormalities seen elsewhere. Computed tomogram (CT) scan (prior to availability of magnetic resonance imaging (MRI)) showed mildly enlarged ventricles including the temporal horns, slightly more prominent on the right. At 20 years-of-age she underwent a right temporal lobectomy extending 6 cm from the temporal pole in the superior temporal gyrus and 7.5 cm from the temporal pole in the inferior temporal gyrus including the anterior 3 cm of hippocampus and part of the amygdala. Pathology revealed mesial temporal sclerosis (MTS). Carbamazepine was discontinued two years after surgery and she remained seizure free for 14 years. During that time, however, she developed depression and frequently complained of short-term memory impairment significant enough to interfere with work. Physical examination was normal other than a left superior homonymous quadrantanopia.

At age 34 years, complex-partial seizures consisting of nausea, abnormal taste, staring and unresponsiveness with oral and bimanual automatisms recurred. Medication was restarted but she continued to have mainly nocturnal seizures about once a month. At age 41 years, after an episode of confusion lasting four hours, possibly nonconvulsive status epilepticus (no EEG done), she was re-admitted for seizure evaluation. The EEG showed infrequent interictal spikes arising independently from the right and left temporal lobes. Two recorded seizures were clearly left temporal in origin. Magnetic resonance imaging showed high T2 signal in the right posterior hippocampus consistent with residual mesial temporal sclerosis as well as increased T2 signal and atrophy in the left hippocampus (Figure). Her complaints of poor short term memory, thought to be depression related, remained unchanged and she was able to function normally including doing part time clerical work.

At age 44 years, while camping with her family, she had two typical complex partial seizures during the night. In the morning, she awoke feeling unwell, vomited several times and returned to sleep. In the afternoon, her family found her with a left gaze deviation and right facial jerking that progressed to a generalized tonic-clonic seizure followed by generalized status epilepticus with recurrent seizures lasting approximately five hours until emergency services were able to locate their camp site. She was transferred to a local hospital where she required propofol to stop the seizures. Investigations showed an elevated white blood cell count, pulmonary infiltrate on chest x-ray, and normal cerebrospinal fluid analysis including negative cultures. No EEGs were done in the peripheral hospitals but a subsequent one showed no persistent seizure activity. The MRI showed unchanged left MTS with no new lesions. There was a postictal right hemiparesis that cleared over several days. However, she now had persistent and marked memory impairment. Since she no longer could function independently, she was transferred to a chronic care unit.

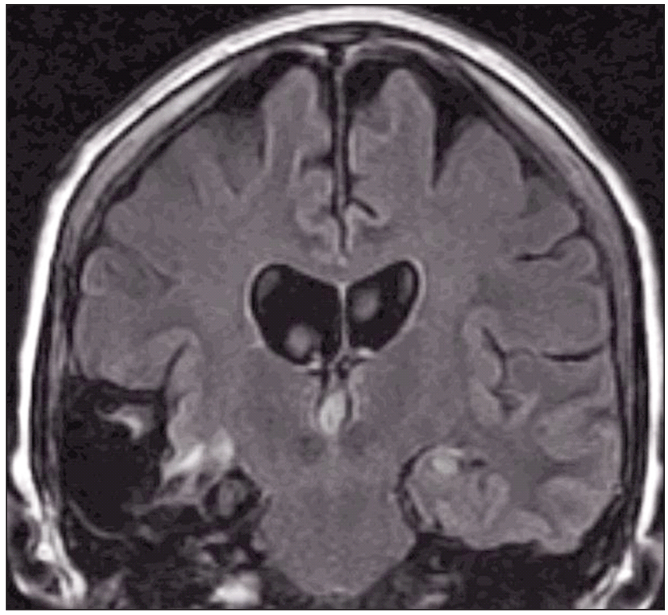

Figure: *MRI fluid-attenuated inversion recovery (FLAIR) image shows left mesial temporal sclerosis as well as the previous right temporal lobectomy with residual sclerosis in the posterior hippocampus.*

Neuropsychological testing documented severe anterograde amnesia and some retrograde amnesia (see below). Functional MRI using a novel memory encoding task showed no activation of the anterior hippocampus on either side. This was not unexpected on the right but suggested impaired memory function in the left anterior hippocampus as well. The posterior hippocampus was activated bilaterally suggesting some residual memory function in that region. After four years, memory impairment was unchanged requiring continuous supervision by family and caregivers. Complex partial seizures have continued about once a week with no further episodes of status epilepticus.

Neuropsychology

Age 20 years: pre-surgical neuropsychological evaluation showed low average IQ, impairments in both verbal and visual memory (worse for visual material), and difficulties in some visual-perceptual abilities (Mooney visual closure task). Language, attention and executive functioning were normal (Table 1). Sodium amytal testing was conducted using a protocol very similar to that previously used at the Montreal Neurological Institute[9]: for each side evaluated independently, during drug effect expressive and receptive language tested and then memory assessed by presenting four items (two pictures of objects, a real

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Table 1: Neuropsychological evaluation results - Standardized Scores for IQ; age-based percentiles for all others

	Surgery		Follow-up		
Age	20 yrs	21 yrs	35 yrs	44 yrs	46 yrs
IQ[1]					
FSIQ	88	81	91	80	83
Language					
Verbal Fluency (Oral; Letter Cues)[2]	52	76	3	0.6	3
Boston Naming Test		18	7	1	1
Attention					
Digit span[3]	75	75	63	63	50
Verbal Memory[4]					
WMS Logical Memory	*1st Ed*	*1st Ed*	*3rd Ed*	*3rd Ed*	*3rd Ed*
Immediate	11	8	9	0.8	2
Delayed			2	0.3	0.8
WMS Paired Associates					
Immediate	2	33	2		
Delayed			1		
Rey Auditory Verbal Learning					
Immediate (Total 5 Trials)			50	0.02	< 0.02
Delayed			8	< 0.02	< 0.02
Visual Memory					
WMS Visual Reproduction					
Immediate	18	2			
WMS 3 Faces					
Immediate			16	5	5
Delayed			5	2	2
WMS 3 Family Pictures					
Immediate			5	2	0.8
Delayed			5	0.8	0.8
Rey-Osterrieth Complex Figure Test					
Copy	56	33	53	0.09	52
Delayed	1	5	3	1	1
Executive function					
Wisconsin Card Sorting Test	*Modified*	*Modified*	*Standard*	*Standard*	*Standard*
Num. of Categories	99	50	11-16	11-16	11-16
Perseverative Errors	98	81	2	4	5
Trail Making Test -A			25	1	9
Trail Making Test –B			66	4	2

1. IQ - Wechsler Adult Intelligence Scale-Revised (WAIS-R) in pre-operative and first post-operative; 3rd Ed. (WAIS-III) in second post-operative; Wechsler Abbreviated Scale of Intelligence (WASI) in third and fourth post-operative. 2. Oral Fluency - 1 letter in pre-operative and first post-operative; total for 3 letters in remaining post-operative. 3. Digit Span - WAIS-R in pre-operative and first post-operative; WAIS-III/Wechsler Memory Scale, 3rd Ed. (WMS-III) in remaining post-operative. 4. WMS 1st Ed. had no delayed memory trials in standardized test, so none reported for ages 20 and 21. Note: Scores were considered 'impaired' if they fell below the 10th percentile compared to age-based norms.

object, and a nursery rhyme); post-drug effect (approximately 11 minutes for left and right sides) recall of the four items was assessed, and a recognition format used if unable to recall. A score of two or less (out of four) constituted a memory test failure. This revealed left hemisphere language with normal recognition memory after right carotid artery injection (recall slightly less, with correct recall of one picture and gist-recall of the rhyme), and severe impairment of memory after left carotid injection (no items recalled, recognition only for object when forced to guess) suggesting that the right temporal lobe could be removed without risk of an amnestic syndrome. By design, the sodium amytal memory test is less demanding than many standardized neuropsychological memory tests. The purpose is to evaluate basic levels of memory support in each temporal lobe functioning independently, as opposed to being sensitive to a range of impairment (as would be the case in more detailed and complex typical standardized neuropsychological memory measures). It is thus possible for a person with temporal dysfunction to have some degree of difficulty on tests of, for example, list learning or complex figure recall, but have adequate memory performance on the sodium amytal memory procedure - particularly recognition. This could come about for a variety of reasons, including the nature of tests used or possibly some eloquent tissue performing memory tasks (albeit to a

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Table 2: Characteristics of 6 patients with late amnesia following a second insult after temporal lobectomy

Year	Sex	HD	Pre-op Memory	Sz Onset	Sz Type	Pathology	Surgery Age	Side	Outcome	Amnesia Age	Cause of Amnesia	Off med
2013	F	R	Impaired visual/verbal	7	CPS Rare GTC	MTS	20	R	Sz free 14yrs	44	Gen SE New L MTS	yes
2006	F	R	Normal	18	CPS	cavernoma	38	R	Sz free 3 yrs	41	L temp Tumor	yes
2004	M	R	Impaired visual/verbal	21	CPS Rare GTC	DNET	62	R	Sz free 5yrs	67	NCSE New L MTS	yes
	F	R	Impaired visual/verbal	26	CPS Rare GTC	MTS	49	R	Decreased	50	NCSE? New L MTS?	no
1997	M	R	Impaired verbal/visual	13	CPS Rare GTC	MTS	19	L	Sz free 8yrs	28	Gen SE? New R MTS	yes
1957	M	R	Not stated	34	CPS Rare GTC	Normal	53	R	Sz free 2mos	54	Acute coma NCSE?	no?

HD, handedness; Sz, seizure; CPS, complex partial seizure; GTC, secondarily generalized tonic clonic; DNET, dysembryoplastic neuroepithelial tumor; NCSE, nonconvulsive status epilepticus.

reduced degree compared to the general healthy population).

Age 21 years: in the first post-operative assessment one year following surgery when she reported some subjective decline in memory, formal testing was generally similar to pre-operative findings with difficulties in both verbal and visual memory (Table 1). Subsequent testing at age 35 years again showed deficits in verbal and visual memory not substantially changed from previously, but also difficulties in some aspects of language (naming, fluency) and executive function (working memory, problem-solving) suggesting some new frontal lobe dysfunction.

Age 44 years: following the episode of status epilepticus she required daily supervision from either family or support workers because of her significant change in immediate recall and long-term autobiographical memory. Direct comparisons to her pre-surgical and first post-surgical assessment were limited given both the extended interval and because different tests or updated versions were used in the later assessments. However, comparing performance to her results from age 35 using Reliable Change Index data[10,11], testing at this time revealed significant declines in memory in comparison to the previous assessments along with deficits in language, some executive abilities, and attention.

Age 46 years: in the last formal neuropsychological assessment, there were ongoing significant impairments in memory (verbal, visual, auto-biographical), language, and possibly executive functions. The last area of those may have been influenced by her severe memory deficits (possibly forgetting task instructions). Autobiographical memory (evaluated through the Autobiographical Memory Interview,[12] and interview with patient and spouse) was variable for episodic remote information but significantly poor for events since and one year prior to the episode of status epilepticus. Baseline Wada memory testing (i.e., no drug administered) was also impaired with a score of 2/8, no false positive errors, similar to that obtained by persons with what was previously called global amnesia[9]. She performed within normal limits on measures of attention, visual construction, and other executive tasks.

Outside of formal testing, she also displayed notably poor immediate memory in general conversation. While in hospital she would forget the location of the nearby bathroom and after one week reported staying only one day. She could not recall the neuropsychologist's name within a few hours of a reminder, but across days could remember he was a psychologist. She did not remember her father's death one week after attending his funeral. In summary, testing and general function indicated a severe anterograde amnesia and limited retrograde amnesia with deficits present across modalities and types of memory measures in excess of other cognitive problems.

Cases from literature

We were able to identify five previous case reports describing patients who underwent temporal lobectomy and later developed severe amnesic syndromes following post-operative insults to the unoperated temporal lobe[5-8]. In the earliest case reported by Walker, the patient received a right temporal lobectomy for refractory epilepsy[5]. Formal neuropsychological memory evaluation was not documented, but the patient "seemed to have no memory impairment after the temporal lobectomy for over a year." Although modern imaging techniques were not available, the pathology of the resected temporal lobe was reported as "normal". One year after surgery the patient developed acute coma (no stated cause) followed by an amnesic syndrome. One may suspect nonconvulsive status epilepticus given that the patient was not seizure free following surgery, although seizures were not described. Details of the subsequent hospital

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investigation are not provided, thus other possibilities including anoxic-ischemic injury, stroke, encephalitis, or trauma cannot be excluded.

The remaining four cases provide the results of serial neuropsychological evaluations, including pre- and postoperative assessments, as well as assessments following onset of amnesia[6-8]. The clinical details of all six cases including ours are summarized in Table 2. One patient with intact memory became amnestic three years after surgery following the growth of a glioblastoma in the remaining temporal lobe. Similar to our patient, three had preoperative visual and verbal memory deficits with no significant deterioration after surgery. Of the four non-tumor cases, two were seizure free for five and eight years, both off medication, before seizure recurrence and two continued to have seizures after surgery. There was confirmed or possible status epilepticus in every case before amnesia developed. As in our patient, MRI in three patients revealed signs of new MTS on the unoperated side.

Discussion

Our patient did very well for some time after right temporal lobectomy for medically refractory temporal lobe epilepsy, remaining seizure free for 14 years post-operatively, 12 years without antiepileptic medications. She was able to work, marry and have children. Although she complained of memory deficits following surgery, these were thought to relate to depression as neuropsychological testing did not demonstrate significant change in comparison to preoperative assessment. Late recurrence of seizures from the other temporal lobe culminated in status epilepticus and subsequent amnesia 24 years after surgery. Follow-up MRI revealed new onset MTS on the unoperated side that appeared after seizure recurrence but prior to the status epilepticus, raising the question whether temporal lobe epilepsy could be a progressive condition in some cases. Four similar cases from the literature also had late amnesia after possible or definite status epilepticus with MTS demonstrated in the other temporal lobe in the three who had MRIs.

Disabling seizures can persist in approximately one-third of patients after temporal lobectomy[13] and will recur in approximately one-third of seizure-free patients after discontinuation of medication[14]. Our patient had a period of seizure freedom lasting 14 years following surgery, most of that time without antiepileptic seizure medications. Risk factors that predict late seizure recurrence after surgery have not been identified[15] but stopping medication is likely one of them. Among patients with seizure recurrence, seizures can arise from incompletely resected mesial or neocortical temporal structures, an extratemporal focus or, as in our case, relapse in the contralateral mesial temporal lobe[16].

The development of MTS of the contralateral temporal lobe after surgery, as occurred in four of the above described patients including ours, is unusual. The precise pathophysiology of MTS and mechanisms of how patients with MTS develop seizures remain unclear[17]. Damage to the hippocampus and surrounding mesial temporal structures can result from numerous etiologies, of which encephalitis, ischaemia/anoxia and surgery are the most common[18]. Temporal lobe epilepsy with MTS is thought to develop after a "latent period" following an early insult to the brain. Risk factors include seizures prior to five years-of-age, seizures associated with febrile illness, complicated febrile seizures, central nervous system infections, head trauma, and birth trauma. However, there may be no identifiable etiology. Most regard MTS as causing TLE, although others argue that MTS is the result of chronic seizures[19]. Most patients present with evidence of MTS on one side but it has been proposed that the early inciting event likely affects both hippocampi asymmetrically[20]. It is possible that our patient had mild unrecognized MTS that was aggravated by seizures that started in the unoperated temporal lobe and then even more so by subsequent status epilepticus. Preoperative concerns about memory in the opposite temporal lobe warrant an amytal study and this was not required in any of the described patients nor did they develop memory problems after surgery until seizures or, in one case, tumor compromised the other side. It is of interest that in five of the six cases, surgery was on the non- dominant right side (the single left temporal lobectomy was on the dominant side). Whether the dominant temporal lobe is more susceptible to memory failure when damaged following opposite temporal lobectomy cannot be determined from such a small sample. Further, these cases do not provide any clear predictors of late onset amnesia other than continuing or late recurrence of seizures particularly when they are coming from the temporal lobe opposite the side of surgery. Since this outcome is rare, it may occur by chance. Nonetheless, patients may want to be aware preoperatively of the risk to memory if the temporal lobe opposite the side of surgery is ever damaged by seizures, stroke, tumor, etc.

Although it is well recognized that amnesia may result from damage to both temporal lobes, the precise structures involved, requirement of bilateral hippocampal lesions, degree of hippocampal damage, and nature of the memory loss is variable and controversial. Even though our patient already had MRI evidence of MTS in the unoperated temporal lobe prior to status epilepticus, severe amnesia did not occur until after that event. Animal models and case reports have demonstrated acute neuronal injury in multiple brain regions following status epilepticus[21]. One definite and another possible episode of status epilepticus in our patient presumably caused damage to remaining cells of the hippocampus, other memory structures including the thalamus, mammillary bodies, and the fibers connecting them with the hippocampal formation, or possibly all of these. Whatever neuronal reserves were responsible for persisting memory were presumably affected by excitotoxic cell damage. However, even after severe amnesia occurred, fMRI suggested residual memory activation in the posterior hippocampus bilaterally. This was clearly not sufficient to allow functional retention of new memories. The previously used term global amnesia to describe such cases, including HM, is misleading since, as in this case, loss of memory may be severe and incapacitating but is never absolute.

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