A Case of Reversible Dementia Secondary to Intracerebral Haemorrhage

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Abstract

The causes of reversible dementia are many, ranging from neuroinfections, tumours, normal pressure hydrocephalus, subdural haematomas and vitamin deficiencies. Reversible dementias secondary to intracerebral haemorrhage are rare and have not been reported. We report a case of reversible dementia secondary to intracerebral haemorrhage, which completely reversed after 16 weeks with return to premorbid level of cognitive functioning. This case report adds to the existing literature on aetiologies for reversible dementia (German J Psychiatry 2008; 11: 156-158).

Keywords: reversible dementia, intracerebral haemorrhage

Received: 22.5.2008
Revised version: 22.12.2008
Published: 30.12.2008

Introduction

Dementia due to potentially reversible aetiologies is an important group of dementias to be identified not only because of the number of such patients encountered but also due to the potential for substantial improvement with treatment.

The actual prevalence of reversible dementias is quite low. A recent meta analysis covering 39 studies representing 7042 patients showed that only 0.6 % of dementias actually reversed (Clarfield, 2003). Causes of reversible dementias can range from cerebral tumours, normal pressure hydrocephalus, vitamin B6 deficiencies (Srikanth & Nagaraja, 2005) and neuroinfections (Cummins, 1983). Subdural haematomas can also be a cause of reversible dementia (Ishikawa et al, 2002).

There is a case report of reversible dementia with presence of lupus anticoagulant, which reversed after treatment with steroids (Van Horn, 2006).

We report a case presenting with features of dementia following intracerebral haemorrhage and which completely reversed 4 months time following partial resolution of the haemorrhage.

Case report

A 48 year old single Caucasian male (Mr M) was taken to the accident and emergency department of the local hospital after his friend had called an ambulance to his home. Mr M was feeling unwell for a few days prior to the admission and had reportedly fallen at home. He was found to be unsteady on his feet.

During assessment at accident and emergency department, he was found to be suffering from high temperature (38.7° C). The Glasgow Coma Scale score was 15/15. There were no focal neurological deficits, pupils were equal and reacting to light, there were no meningeal signs. Systemic examination was normal. Blood pressure readings were within normal limits. He was oriented in place and person but was unable to say the day, date, month and year. Immediate, recent memory and remote memory were intact. The Mini Mental Status Examination (MMSE) done at that time
was 26/30. He also gave a history of heavy alcohol con-
sumption for the past 30 years, which was more for the past
10 years, drinking up to 70 units per week. There were no
features of alcohol withdrawal at that time. Blood cultures
were taken. All blood investigations were done which
showed increased white blood cell count. Liver function
tests and electrolytes were normal. He was started on i.v.
fluids, injection thiamine was given and was also started on
amoxycillin and clavulanic acid which was continued for
about 7 days. Urinary tract infection was also ruled out. A
possible sepsis was suspected with a differential diagnosis of
Wernicke’s encephalopathy in view of his past drinking and
mental confusion at the time of admission. There was no
past psychiatric history or significant past medical history.
There was no history of hypertension or diabetes.

He was subsequently admitted to the medical ward. In the
medical ward he developed urinary incontinence, became
disoriented in time, place and person. Immediate and recent
memory was impaired and started to have confabulation. Mr
M believed that he was still employed as a lorry driver
and that his parents were still alive. According to his brother,
he had actually lost his driving licence due to drink driving
several months back and both his parents had died 10 years
back. He believed that he had 3 cars parked at 3 different
hospitals in the county even though he had only 1 car at
home. An unenhanced CT scan was done due to worsening
in cognition. It revealed evidence of a sizable parafalcine
bifrontal abnormality, which looked like a resolving intra
cerebral haematoma. There was no mass effect with any
extra cerebral collections.

He was started on lactulose on advise of the neurosurgeons.

A referral was sent to the traumatic brain injury team and he
was seen by them. A brief cognitive screen was done which
highlighted confusion, disorientation to place and time. His
anterograde memory was impaired, there was decreased
recall of new information and impaired lexical memory. His
MMSE score was 27/30. There was marked confabulation as
well. It was suggested at that time that he needed a period of
inpatient stay at the psychiatric unit in view of his cognitive
deficits, as he was not safe to be discharged home. The neu-
ropsychologist from the traumatic brain injury team then
reviewed him again a week later who carried out further
cognitive tests. Mr M was still disoriented in place and
person. Visuospatial abilities were impaired and retro-
grade/anterograde memory was very poor, confabulation
persisted. It was noted that Mr M had excellent abstract
reasoning ability with good calculation skills. Reading and
writing was preserved.

Mr M subsequently absconded from the ward two times and
was found in a pub drinking alcohol and was brought back
by his friend. He was referred to the psychiatry team for
further assessment and observation in psychiatric unit. He
was admitted to a psychiatric unit as an informal patient. He
was discharged from the medical ward to the care of the
psychiatry team on thiamine and vitamin B complex. The
MMSE on admission was 27/30.

A detailed cognitive assessment was done again. Adden-
brooke’s cognitive examination was done which revealed a
score of 97/100, with an MMSE score of 30/30. Confabula-
tion was still present. He believed that he was still employed
as a lorry driver, believed his parents were alive and that he
had spent time with them at home.

Due to the persistent confabulation, a possible diagnosis of
Korsakoff’s syndrome was considered due to the past his-
tory of alcohol use and marked confabulation. A repeat MRI
was asked for, to look specifically at the mamillary bodies
and periaqueuductal grey matter as they are generally in-
volved in this condition. A repeat MRI scan done did not
reveal any structural deficits suggestive of Korsakov’s syn-
drome and the bifrontal intracerebral haemorrhage has sig-
ificantly reduced with minimal oedema. The traumatic
brain injury team neuropsychologists continued their as-
sessments and detailed neuropsychological assessments
were carried out a week later showed areas of improvement in
verbal fluency, visuospatial function, a verbal IQ of 110 with
improvement in psychomotor speed. Mr M also reported
that his memory especially for recent and remote events was
returning.

Occupational therapy referrals were done and he was taken
on a home visit with them to check functional skills at home.
He was reviewed again by the neuropsychologist. It was felt
that Mr M's memory and higher cerebral functions was
showing continued improvement. Remote and autobi-
ographical details were also returning to the same degree.

Joint visits with the occupational therapist were done to
review his living skills. He was able to cook a meal on his
own and was aware of common dangers. His financial affairs
were looked after by his brother during his hospital stay and
efforts were being carried out to transfer it back to him on
gradual basis. Advice was also given to him to stop drinking
completely and he was referred to the drugs and alcohol
team for continuing community support.

He was subsequently discharged from the ward. At the time
of discharge his confabulation had totally stopped. He was
aware that both his parents were dead and was able to recol-
clect events leading to his hospital admission. There were no
cognitive deficits noted at the time of discharge and the
decision was taken to discharge him to his home. He was
offered follow up appointments at the community mental
health team and at the traumatic brain injury team as well.
He was seen twice in the outpatient clinic following dis-
charge and the MMSE remained at 30/30 without any defi-
cits in memory and cognitive function. He remained absti-
nent from alcohol and was also planning to apply for a job.

Discussion

We have described a case of reversible dementia secondary
to intracerebral haemorrhage. There was no evidence of
cognitive decline before his hospital admission and was
functioning independently.
His symptoms of cognitive decline reversed completely after 14 weeks of hospital admission. Serial CT scans and MRI scans showed gradual resolution of the haemorrhage with concomitant improvement in cognitive function and confabulation, which was serially tested by neuropsychologists from the traumatic brain injury team. A possible differential of Korsakov syndrome was considered in view of the persistent confabulation but was ruled out due to the complete reversibility of the recent memory deficits and total absence of confabulation at time of discharge. The most recent MRI scan done just before discharge from hospital also did not reveal any structural lesions associated with Korsakov syndrome such as mammillary body haemorrhages or structural changes in the periaqueductal grey matter.

Since discharge he has returned to his premorbid cognitive functioning level and has been followed up by community services who have not noted any decline. Mr M remains on Thiamine supplements and B complex tablets and attends groups therapy for alcohol issues. He currently remains abstinent from alcohol and has been abstinent for the last 7 months.

Intracerebral haemorrhage leading to cognitive deficits, which completely resolved over a space of 3 to 4 months is something which has not been reported and hence this case was presented. Prominent confabulation with no evidence of temporal lobe or hippocampal pathology on MRI scan which completely reversed over time is also something which has not been reported. The complete reversal of cognitive deficits took place after 16 weeks, which is quite short.

This case adds to the list of aetiologies of reversible dementia with intracerebral haemorrhage as one of the causes. More cases need to be identified to add strength to this particular aetiology.

References