

# ATENOLOL INDUCED MEMORY IMPAIRMENT: A CASE REPORT

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## ABSTRACT

*This report deals with a 54-year-old man with loss of memory. His impaired memory was found to be due to the atenolol he was on and he made a complete recovery on withdrawing the beta-blocker. This patient's experience stresses the need to consider beta-blockers as a potentially reversible cause of memory impairment.*

**Keywords:** *memory deficits, beta-blockers, atenolol*

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## INTRODUCTION

Beta-blockers, particularly the hydrophilic agents such as atenolol, are generally considered to be free from central nervous system side effects. We have a patient who suffered from progressive loss of memory as a result of atenolol therapy.

## CASE REPORT

A 54-year-old executive engineer was admitted to the hospital because of loss of memory. The patient had experienced progressive loss of memory especially of recent events for three months prior to admission. During the initial phase of his illness, the patient felt he was getting forgetful because of pressure of work. Soon his memory deficits embarrassed him both socially and professionally.

He had difficulty in concentrating on his work as an executive engineer in a large organisation. He felt uncomfortable as he could not recollect the gist of a board meeting shortly after it was over. On several occasions he could not instantly recall the names of his colleagues. He could not grasp new ideas or concepts in his job. He found it increasingly difficult to sort out simple day-to-day professional problems which he could handle with ease previously.

At home, he could not remember his shopping lists. At the time of consultation he could "only remember three or four items out of ten for the day". He resorted to reminder cards and pocket diaries to overcome his memory deficits. He had no difficulty in driving to his office. But, occasionally the roads did not appear familiar to him. Although he was perturbed by his present predicament, he denied being depressed or having suicidal tendencies. He had no marital or financial problems either. His appetite was normal and he had no loss of weight. He did not suffer from insomnia and specifically denied early awakening. The patient was not noted to be agitated either by his close relatives or colleagues. His wife observed that he carried out his domestic duties as much as he could. The patient himself denied harbouring any feeling of worthlessness or guilt at any stage. He, however, admitted to "some loss of libido" for an unspecified period of time on direct questioning.

He sought the present consultation to get a medical opinion before applying for an optional retirement to avoid further embarrassment in his professional and social circles. His past medical history, however, was remarkable for several events.

He sustained a head injury following a fall from a moving tram at the age of 8. He was unconscious for a few days in a hospital but no further details were available. He suffered from pulmonary tuberculosis when he was 16, and was on antituberculous chemotherapy for 18 months. Seven years later, he developed fever and became comatose. He was then told that he had cerebral malaria from which he recovered following treatment.

He had recurrent headache when he was 37 and was then diagnosed as suffering from "migraine" by a neurologist. He was on ergot derivatives and mefenamic acid for a few years.

Three years later, he was found to be hypertensive on a routine examination. His blood pressure was well controlled on atenolol 100 mg a day since then. Apart from atenolol he was not on any other medication at the time of present entry.

Physical examination revealed a very pleasant and cooperative patient. He had no hallucinations and was well orientated. His Mini Mental Score<sup>(1)</sup> was normal at 26 out of 30. He could not recall two of the three objects after 3 minutes. Although he could subtract serial 7s from 100 with ease, he could not spell "world" backwards.

The following laboratory evaluations were normal or negative: complete blood counts, erythrocyte sedimentation rate, blood urea, serum electrolytes, random blood sugar, thyroid function tests, serum B<sub>12</sub> and folate assays, Venereal Disease Research Laboratory (VDRL) tests, Treponema Pallidum Haemagglutination Test (TPHA), electrocardiogram (ECG) and chest X-ray. A computed tomographic scan of the brain was reported to be normal.

The atenolol was stopped. His hypertension was successfully controlled with one tablet of Moduretic (Amiloride HCL 5 mg, hydrochlorothiazide 50 mg) a day.

Four weeks later, he reported a "seventy-five per cent" recovery of his memory and made a complete recovery after a further period of eight weeks. He returned to work with greater vigour and interest. And so he decided against optional retirement and remains well two and a half years later.

## DISCUSSION

The patient was initially thought to suffer from depression, especially as beta-blockers can themselves cause this affective disorder<sup>(2)</sup>. But, apart from memory impairment, he did not have sufficient features to meet the accepted criteria for depression<sup>(3)</sup>. His loss of libido is a known side effect of beta-blocker therapy. Amnesia has been linked to toxic levels of beta-blockers<sup>(4)</sup>. Although the blood level of atenolol was not measured in this patient, the gradual onset of memory impairment with paucity of other features made acute atenolol toxicity unlikely in this patient.

Dementia has been defined as "an acquired, persistent

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impairment of intellectual function with deficits in at least three of the following five spheres of mental activity: memory, language, visuospatial skills, cognition (abstraction, mathematics, judgement and so forth) and personality<sup>(5)</sup>. This patient had one of the cardinal features of early dementia, viz memory deficit, which was severe enough to interfere with his social and occupational functioning<sup>(6)</sup>. Thus for practical purposes he was thought to suffer from some form of dementia.

The usefulness of several routine tests in dementia has been questioned recently<sup>(7,8)</sup>. The list of investigations done on this patient may be justified by the patient's clinical presentation, the desire to find a cause for his memory deficits and finally the patient's and his relatives' specific requests to ensure everything possible be done to get to the root of the problem.

The extensive investigations on this patient ruled out some of the more commonly reversible causes of dementia such as hypothyroidism<sup>(5,8)</sup>, megaloblastic anaemias<sup>(5,8)</sup> and chronic subdural haematomas<sup>(5,8)</sup>. The diagnosis of atenolol - induced memory deficit was thus one of exclusion in this patient.

Beta-blockers were first introduced in 1958. Initially, these drugs, particularly the hydrophilic agents such as atenolol, were thought to be free from central nervous system side effects. But, in due course, several case reports and reviews drew attention to the possibility of beta-blocker-induced cognitive impairment<sup>(3,9)</sup>. The exact mechanism by which beta-blockers cause memory deficits is not known.

Initially, only the lipophilic beta-blockers such as propranolol were thought to cause cognitive impairment<sup>(9,10)</sup>. But, as illustrated by this patient, the hydrophilic beta-blockers such as atenolol can also cause neuropsychiatric side effects. This adverse reaction due to atenolol has been reported earlier<sup>(11)</sup>. Unlike the lipophilic beta-blockers, hydrophilic agents may take several years before they give rise to cognitive complaints, which makes it difficult to establish causal association<sup>(11)</sup>. The acid test in this patient was the decision to withdraw atenolol from him. The rapid improvement in his memory following the withdrawal of atenolol confirmed the clinical suspicion that atenolol was the cause of his memory impairment.

Beta-blockers are widely used. It is necessary to screen patients on these agents from time to time for possible cerebral side effects. If there is any suspicion of cognitive impairment in them, then one should withdraw the beta-blockers especially when equally effective alternative drugs are available. Although atenolol was stopped abruptly in this patient with no untoward reaction, it may perhaps be safer to withdraw the beta-blocker gradually to avoid rebound phenomena.

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