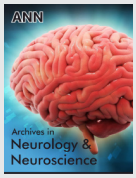

ISSN: 2641-1911

DOI: 10.33552/ANN.2026.18.000948

Archives in Neurology & Neuroscience

Iris Publishers

Case Report



# ADHD - A Syndrome with Varying Aetiologies

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**Received Date:** July 02, 2026

**Published Date:** July 17, 2026

## Abstract

The author has argued elsewhere [1,2] that the definition of 'ADHD' is problematic. It leads to restricted thinking about the causes of problems, specifically inattention and hyperactive states. There are many causes of poor attentional skills and over activity, not just an arbitrary collection of symptoms that have come to be called the syndrome of 'ADHD'. The picture has been further complicated in the UK by a 'tick-box' approach to diagnosis leading to the mushrooming of online diagnostic services many without medical doctors trained in the speciality, bestowing diagnoses to allow access to educational resources that are squeezed and trying to ration output (eg EHCPs). The below cases would both have been diagnoses as ADHD in other settings and treated with stimulants or similar.

## Case 1:

S was a 60-year-old man with a long history of being unable to focus well and having a sense of disconnection from the world in general. Despite this he has made a great success of his construction company, despite often not being able to concentrate long enough to complete quotations. He was also a keen golfer but here would lose concentration while playing a hole at times.

He was often restless and slept poorly. He also had repeated sinus problems, urinary frequency (for many years), a number of allergies (especially animals) and a tendency to bowel problems. In more recent years he had become unable to tolerate garlic and had longer standing issues with lactose.

I had seen one of his two children previously for Hyperactive subtype ADHD and he had responded well to very low doses of stimulants but was exquisitely sensitive to dose levels.

His father may well have had ADHD or a neurodivergent profile with a number of ASD-type features: he was very bound to routine, easily triggered and angry and therefore S, and his family were all subject to physical battering. Not unnaturally, he became quite anxious around this and probably masked his ADHD symptoms in order not to be hit by his father. His early memories were related to abusive experiences or threats of same. His mother had depression (possibly largely related to her marital situation and also difficulties in her own upbringing) and she had a great many allergies as well.

In Primary he did well and was top of everything, but was always quite restless. When he got to Secondary, with the increase in demands and scope of subjects widening, he struggled, particularly with homework. He was always better with coursework than exams but gained five O-Levels (some of which he took early) and one A-Level. He was always socially very adequate and had lots of

friends.

He had a good diet with home-cooked food sources avoiding lactose and did not drink alcohol as he reacted very negatively to it with vomiting or diarrhoea.

He met the screening criteria at interview and on standard instruments for ADHD (Combined subtype) and in addition to the above reported hay fever and a tendency previously to mouth ulcers. He also had acne, was hot at night and had a tendency to bloating. In addition, he was quite hypermobile. He noted that when he took some supplements I had suggested for his son, related to gut function, his own tendency to runny motions and pain reduced.

My conclusion initially was that he had marked histamine-related symptoms (often found with concentration issues and hypermobility in my experience and that of others), and that his early (paternal) experiences may have predisposed him to anxiety and mood difficulties (reported by his wife but not so much by him).

Initial investigations showed lower range B12 and low minerals responsible for controlling blood sugar levels and among other predisposing factors a GPX1 gene SNP suggesting a lowered pathway for elimination of some substances, and in particular, histamine. Stool bacterial balance was not optimal but not highly disordered.

He was treated initially with low dose lisdexamfetamine (as used successfully in his son) and an antihistamine (combined H1 and mast cell blocker/stabiliser). However, he reacted badly to the Lisdexamfetamine with a marked increase in anxiety and feeling quite depressed. This was almost certainly due to interaction with his histamine metabolism. The antihistamine helped his sleep and night sweats but not his anxiety or focus (which is why I give it). He reacted poorly to subsequent trails of guanfacine (excessive sedation) and atomoxetine (urinary retention).

He went on holiday to the Middle East and became ill with a chest infection. He was prescribed antibiotics and subsequently a B-complex injection. For one day after this his brain felt clear and he could focus – a new experience.

Subsequent investigations showed a double normal level of homocysteine implying his methylation was less than adequate. His genetic mapping (Lifecode Gx UK)did not show particular vulnerabilities on that pathway. Attempts to support with supplements led to further negative reactions and it seemed that he had become sensitive to sulphur sources.

He had a downregulation SNP on his SULT1A1 gene which may have contributed to this.

Currently he is undergoing a very slow desensitisation via diet and when sufficiently well frank attempts to support his methionine cycle (and so lower homocysteine) will restart. This is therefore not 'classic ADHD.'

## Case 2

K was a 7-year-old boy with ADHD symptoms. He was 'on the go' all day for 6am and showed episodic dyscontrol or impulsivity. He fulfilled criteria for a diagnosis of ADHD Combined subtype on interview and questionnaire measures. However, the hyperactive symptoms were clearly predominant. In terms of medical symptoms he reported headaches (when he is overwhelmed), a tendency to sore throat and cough, skin problems, excessive sweating and being hot at night, a tendency to excessive intestinal gas and pain, pains and aches in his joints and hypermobility. He had had Molluscum Contagiosum for some time and it was quite widespread when he saw me.

As an infant he had colic. Dairy was removed at eighteen months as he had a slightly upset stomach that increased with solids, and bright red cheeks. All symptoms disappeared when dairy was removed and he has subsequently been discovered to have sensitivities to soya and gluten as well. Eczema was also helped by the dietary adjustments.

In the last three years he had shown increasing bouts of tonsillitis and viruses. He saw a specialist due to recurring tonsil infections and mother implemented a supplement protocol in relation to viral fatigue, with benefit – since doing this he had only had tonsillitis once in nine months. The family history showed some similarities in behaviours in other members.

Testing showed a high level of mycotoxins in his urine – ochratoxin 48, citrinin 36, gliotoxin 353 – and a vulnerable histamine genetic pathway, along with upregulation via an IFNG SNP driven by inflammation or cortisol (both present). A protocol to eliminate moulds was hard for him to follow to completion.

He responded poorly to all ADHD medication and several other medications but showed a marked improvement in symptoms on Ketotifen 1mg at night. This was not just due to sedation (can be an issue, but was minimal here) his core symptoms improved as well. Ketotifen is a combined H1 blocker and mast cell stabiliser. He is not completely controlled but much improved and work continues via diet and supplements.

These cases serve to demonstrate that the rather narrow definition of ADHD and the presumption of treatments attaching to that label is unwarranted. A wider consideration of the causes of difficulties and a return to proper fully trained medical assessment of disease- disorders is called for. That assessment needs to encompass a full understanding of metabolic and whole person causes. This has implications for Child Psychiatric training and reconnection with its medical roots.

## Acknowledgement

None.

## Conflict of Interest

No Conflict of interest.

## References

1. Fry, Richard (2024) Debate: 'Neurodiversity' - has it outrun its usefulness? Child Adolesc Ment Health 29 (3): 314-315.
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