Attention deficit hyperactivity disorder across the lifespan

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Educational aims

- The most up to date research on Attention Deficit Hyperactivity Disorder, in a succinct complete way
- A description of a multimodal assessment for ADHD
- A complete description of treatment methodologies available for people with ADHD
- To answer questions often asked of ADHD and its treatment

Key words
Attention deficit hyperactivity disorder, multimodal assessment, medication, non-pharmaceutical interventions, evidence based

Abstract

This article is a research summary of published and non-published work pertaining to Attention Deficit Hyperactivity Disorders (ADHD). ADHD is one of the most common child mental health disorders and is under-recognised in children (5.29%) and adults (2.5%). ADHD is highly heritable with a multifactorial pattern of inheritance. Siblings and parents of a child with ADHD are 4 to 5 times more likely to have ADHD. Methylphenidate is the first line pharmacological treatment with a combined response (this includes trials of other licensed amphetamines) rate of 95%. All clinicians working in mental health should be aware of this disorder, comfortable diagnosing and treating people with ADHD. Young people with untreated ADHD are 5 times more likely to develop antisocial behaviour, substance abuse and other co morbid psychiatric disorders.

Introduction

The purpose of the review is to cover the epidemiology, aetiology, diagnostic criteria and different managements of Attention Deficit Hyperactivity Disorder (ADHD) with specific reference to practice of ADHD assessment and treatment management in Malta. This review's target audience is for all clinicians to better understand what ADHD is and explain any misconceptions there are related to the medications which are used to manage ADHD and relate this to Malta. The authors will look to answer the questions using evidence based published and unpublished research.

What is ADHD?

ADHD is among the most common neurobehavioural disorders presenting in children and adolescents.[1] It is characterised by persistent symptoms of inattention, hyperactivity and impulsivity according to Diagnostic and Statistical Manual of Mental Disorders 5th edition (DSM 5) present in two of three environments (namely home, school, clinic).[2] The onset of the symptoms must have been present before the age of 12 years; this was increased from the previous DSM- IV due to the recognition that adults may also be diagnosed with ADHD, however may not clearly remember their symptomatology in early childhood.[2] This increase in age to diagnosis was thought to create a sudden rise in the prevalence of ADHD diagnosis around the world. ADHD is also classed under the term Hyperkinetic Disorder in the International Classification of Diseases 10th edition (ICD10) with similar characteristics including early onset, disorganized, ill-regulated, excessive activity, recklessness and impulsivity.[3] The main difference between the DSM-5 and ICD-10 diagnostic criteria is that in the former a young person (YP) may be diagnosed with concentration difficulties only, also known as Attention Deficit Disorder (ADD) or hyperactivity and impulsive symptoms but no concentration difficulties or ADHD combined type (attention, hyperactivity and impulsive symptoms). As opposed to the ICD-10 diagnostic criteria where a diagnosis is made only when all three core symptoms are present.[3]

Over the last decade there has been interest in the pragmatic use of social language in children with ADHD, this is the domain that manages conversational contexts. It was reported that as many as 50% of children with ADHD[4] have less

developed pragmatic language skills (receptive and expressive) related to their typically developing peers. They also suffer from a developmental delay in onset of talking.[5,6] As a result, social problems are reported in 52-82% of children with ADHD7; such as having fewer reciprocated friendships,[8] and being more often disliked by their peers.[9] Social problems arise due to symptoms of impulsivity (e.g. interrupting, difficulty waiting their turn), and inattention (e.g. not listening). This means that a child with ADHD has a greater chance of getting into trouble at school and then when called in to explain to the teacher what really happened, struggles to verbalise the experience, as a result may be judged as defiant. Subsequently peer rejection and education failure has been associated with negative long-term outcomes such as substance abuse, delinquency and academic problems.[10]

Is ADHD a valid diagnosis in adults?

ADHD is a common behavioural disorder that is associated with significant adult psychopathology, social and academic impairments and the risk for negative long-term outcomes. There is no doubt that in many cases ADHD symptoms persist into adult life and cause significant clinical impairments. ADHD diagnosed in childhood tracks on through to adulthood, with 4-15% of adults retaining the full diagnosis and 50-66% of YP presenting in partial remission of ADHD symptoms.[11,12,13] The main clinical issue is recognition of the disorder in adults and quantifying the impact on adult mental health.[12]

To date there has been considerable debate on whether ADHD is a disorder solely present in YP or whether there is evidence that ADHD symptoms persist through to adult life. The latter hypothesis, is strongly supported by research,[11] which found that symptoms of ADHD persist in 65% of adults. Furthermore, it is thought that the ADHD symptoms do not resolve in adulthood, but rather adults develop the required social skills to control and mask their ADHD symptoms and adapt to social requirements.[11] On the other hand, Moncrieff has argued that the validity of symptoms in adults do not automatically follow those used to diagnose children and concluded that the rapid growth in interest in adult ADHD could be the result of the drug companies seizing the opportunity to expand on a lucrative market.[14]

Clinical presentation in adults

Adults with ADHD clinically present with more symptoms of poor attention (rather than hyperactivity) and ceaseless mental activity (distracted mind) such as procrastinating to start a job, then trying to multitask and carry out a number of jobs at the same, without ever finishing any of these jobs or finishing them with careless mistakes. Hyperactivity (over activity) is not as prominent symptom in adults, since adults learn to manage their behaviour, on the one hand through learning to adapt to social norms and also due to development (maturity) of the pre-frontal cortex. Mood dysregulation and lability are common symptoms in adults with ADHD. These symptoms lead to low tolerance of frustration, falling out with peers and colleagues, and as a result this effects their self-esteem and can lead to poor performance at the work place.

Adults with ADHD prefer occupations that are exciting and busy and that have an element of risk, such as: sales, stock broking, entrepreneurial ventures. They undergo frequent changes in employment, have poor planning abilities, e.g.: organising finances, handling course work at college, live and work in a messy environment, enjoy reckless driving, have trouble maintaining stability within their relationships and as a result may suffer from social isolation. They may choose to engage in leisure activities that are highly absorbing or stimulating, such as downhill skiing or high-contact sports. Adults with ADHD usually have difficulty organising their homes, such as cooking regular meals, cleaning and managing their children (e.g. packing their lunches, getting them to appointments and school on time).[15]

Newly diagnosed adults with ADHD have presented in adulthood, the commonest precipitant factors for these include: infection (e.g. Rheumatic fever), degenerative disorders (e.g. early onset dementia), acquired brain injury (e.g. punch drunk syndrome) and intoxication (e.g. heavy metal poisoning).

There is, thus, clear evidence that ADHD is evident in adults but the diagnosis of adult ADHD is complex and the diagnostic criteria may be unreliable. Untreated YP with ADHD symptoms who are exposed to high expressed emotion within their families and experience poor social interactions, have a higher risk of developing conduct disorder (anti-social personality traits) symptoms in adulthood.[16,17]

Epidemiology - how common is ADHD?

The worldwide prevalence of ADHD in children 0 - 18 years was reported to be 5.29% in a systematic review and meta-regression analysis conducted by Polanczyk *et al*,[18] with minor differences found between countries around the world. For example in the United Kingdom it was estimated that the prevalence of ADHD is 2.23% of children age 5-15 years.[19] Whilst in the United States of America, the National Health Interview Survey (NHIS) in 2006 estimated the prevalence of ADHD among children age 3-17 is 7%.[20] The possible reasons for the significant difference in prevalence rates between these two countries has been widely debated; the most common reasons for the low prevalence reported in the UK is due to the strict adherence to ICD-10[2] as opposed to the DSM-5 in the USA. Furthermore, Biederman *et al*[19] reported that the USA have higher rates of social deprivation and experiences of trauma as a country when compared to the UK. In addition, one of the reasons apart from the more lax DSM-5 criteria for diagnosing ADHD, is that for parents to get clinician reviews refunded by insurance, a diagnosis needs to be given. It is reported that ADHD is more common in YP living in urban rather than rural communities and there is a link with low socio-economic status. It is believed that ADHD is an under-identified and under treated disorder.[21]

In adulthood, the overall pooled prevalence rate for adult ADHD was 2.5%[22] reported in a robustly conducted meta-analysis. Furthermore, Simon *et al* reported that children do not outgrow the disorder (ADHD) but they outgrow the diagnostic criteria (ICD-10, DSM-5), therefore this means that there may be an underestimation of the true prevalence of this disorder in adults. Some of the reasons for the possible underestimation include: different methodological and diagnostic differences used in the different studies lead to differences in results, symptom recall bias, the use of DSM-5 diagnostic criteria to diagnose adult ADHD[23] which were written for YP, not adults,[12] and the overlap of the symptoms of adult ADHD with other disorders, as well as the adult co-morbid disorders.[12]

ADHD is more prevalent in males than females,[24] however the ratio varies depending on the study design of populations. The reported range varies between 9 males is to 1 female and 2.5 is to 1.[25] Further analysis has gone into why

there is such a discrepancy between males and females, it was hypothesised that it is almost socially accepted for boys to be hyperactive (boisterous), however girls are praised for being more obedient. It is also reported that girls tend to daydream more. However, day dreamers tend to still be quiet in class, therefore not picked up by teachers as having a concentration problem which may be effecting their overall academic potential.

The Evolving Concept of ADHD - is this a disorder created by today's world?

The diagnosis of ADHD in children has been a controversial issue for many years, with some researchers arguing that it does not exist and whilst others presenting evidence of its existence. Similar controversy as expected evolved in reaction to the diagnosis of adults with ADHD.[17] The skepticism about adult ADHD is influenced by the absence of well validated and universally accepted diagnostic criteria so adult ADHD diagnosis is significantly biased by current level of functioning.[22]

The evolution of the concept of ADHD goes back to the early 20th century when Still in 1902 described children with hyperactivity and poor attention as having a "defect of moral control".[26] The first reported use of a stimulant to reduce hyperactivity was by Bradley who used Benzedrine (stimulant) in children as early as 1937.[27] Furthermore DSM-II included the earliest form of ADHD in their criteria, describing it as "hyperkinetic reaction of childhood". This evolution of ADHD as a disorder continued with the latest edition of the DSM-5 broadening the age of onset from "on or before age 7" to "on and before age 12".

Aetiology - Is ADHD a group of behaviours acquired through one environment or is there a genetic linkage?

ADHD is a developmental disorder, which is biologically based but environmentally influenced. There is no one gene which causes ADHD, due to interactions between multiple genes of small effect size.[28] It is understood that ADHD results from an interacting combination of genetic and environmental factors.[29] Genetic studies including twin and adoption studies indicate high heritability of 0.8. Family studies report a 57% prevalence rate in children of ADHD adults, whilst parents and siblings of a child with ADHD have an increased risk of 4 to 5 times being more likely to have ADHD than the general population.[30,31]

ADHD is best viewed as a gene-environment interaction. Currently there are two predominant theoretical models used to explain ADHD with a genetic predisposition. Each based on a distinct neuropsychological deficit: first executive dysfunction underpinned by deficient inhibitory control mechanisms and second, delay aversion, underpinned by behaving impulsively in order to avoid delay. Children with a biological predisposition will manifest the disorder when placed in the correct environment, typically one characterised by chaotic parenting.[32]

The neurobiology of ADHD suggests its association with cognitive processing deficit [24] and the pathophysiology is strongly linked to dopamine and norepinephrine neurotransmitter systems in frontostriatal circuitry based on brain imaging, carried out in neuropsychological and pharmacologic studies.[25] Rutter *et al* indicated that the main problem lies in "behavioural dysregulation, executive deficits in inhibitory control and working memory, and delay aversion".[24]

Neuroimaging studies of children have shown smaller amygdala volumes which were not evident in adults with ADHD.[33] Poor functioning in the striatum, frontal lobes, and posterior periventricular regions are indicated in attention problems.[34] The hypofrontality theory suggests that ADHD is associated with subnormal activation of the prefrontal systems responsible for higher-order motor control.[35]

For complex conditions such as this, biological based phenotypes that lie in the pathway from genes to behaviour may provide insight into the link. Such endophenotypes have aided the clarification

Table 1: ADHD diagnostic criteria[2,3]

Symptoms Groups		
Inattention	**Hyperactivity**	**Impulsivity**
Does not attend	Fidgets	Talks excessively
Fails to finish tasks	Leaves seat in class	Blurts out answers
Cannot organise	Runs/climbs excessively	Cannot await turn
Avoids sustained effort	Cannot play/work quiet-ly	Interrupts others
Loses things	Always "on the go"	Intrudes on others
Forgetful		Blurts out answers
Easily distracted		
Does not listen		

DSM-IV ADHD	ICD-10 HKD
Either or both of following:	All of following
At least six of nine inattentive symp-toms	At least six of eight inattentive symp-toms
At least six of nine hyperactive or impulsive symptoms	At least three of five hyperactive symptoms
	At least one of four impulsive symp-toms

Pervasiveness

Criteria are met in one situation and impairment is present in another Criteria are met in more than one situation

of aetiology at pathophysiology of several conditions in medicine. Neuropsychological impairments, neuroimaging and electrophysiological paradigms for ADHD show potential to move molecular genetics research forward. However, familial or genetic overlaps between these constructs still remain unclear. The identification of an 'endophenotype' to help clarify which 'at risk' subjects will go on to develop ADHD could help reduce this high rate of disability.[32]

Environmental factors also play a role in the development of ADHD, these include severe neglect resulting in attachment disorders.[36] A lot of work and research has come out of the seminal Bucharest studies, these are large scale studies conducted on children raised in very deprived conditions in the Romanian orphanages in the times of Caucescu. The findings reveal that children who suffered from maternal and nutritional deprivation at the ages of 0 to 1 year are likely to have under developed right limbic systems and as a result suffer from emotional dysregulation.[37] Further environmental factors include, obstetric complications[38] although this theory is currently disputed, very low birth weight (<1000g), pre or post-natal insults, exposure to lead poisoning, head trauma[25] and nutritional deficiency were expansively shown to contribute to the development of ADHD.[39]

Proportion of co-morbidity in people suffering from ADHD

If ADHD is under or misdiagnosed or not managed well, the prognosis for YP is poor; this means that there will be negative social, academic and vocational consequences. A large proportion (78%) of YP with ADHD tend to present with at least one co-morbidity, the commonest include mood disorders (40%), substance dependence (35%), anxiety disorders (25%).

Furthermore, co-morbid psychiatric conditions are not uncommon in adults with ADHD. By comparing adults with ADHD with a sample of YP without the childhood psychopathology, the results show high rates of antisocial personality disorders with poorer prognosis,[24] these rates vary from 12%[12] to 23%[13]. Other co-morbidities include a high rate of substance abuse,[12,24] depression,[40] anxiety and bipolar disorder[41] Social impairment, repetitiveness or perseveration, rigidity and inflexibility[42] are also common co-morbidities.

Assessment process - is a one stop shop at a psychiatrist for a Methylphenidate prescription considered good practice?

According to NICE guidelines 2008[43], a diagnosis of ADHD should follow a multimodal approach. Therefore, the diagnosis needs to be made by a multidisciplinary team (MDT) specialised in ADHD. These include a clinical assessment of the YP, the ADHD symptoms in the different domains and settings over the past 6 months, substantiated by using standardised rating scales e.g. Connors',[44] SNAP–IV[45]. The initial assessment is then followed by a developmental (including prenatal, infant and early years) and neuropsychiatric history (ADHD symptoms e.g. DIVA 2.0[46] and assessment of co-morbidities e.g. anxiety, depression, learning disorders, autism spectrum disorders, tics, substance misuse), obtaining a collateral history and assessment of the YP's current mental state. A school or home observation are valuable adjuncts to reviewing the YP in their natural environment. Furthermore, obtaining a family psychiatric history, especially concerning learning problems, attention and behaviour problems, ADHD and tics and enquiring about all first-degree relatives (parents, siblings and offspring) is necessary. A physical examination to rule out medical causes of symptoms (e.g., serious head injury, seizures, heart problems, thyroid problems) or contraindications to medical therapy (e.g., hypertension, glaucoma) and to get baseline recordings of heart rate, blood pressure, weight and height are also required.

There are some controversies around the use of cognitive testing, however, there are centres of excellence such as the Tees Esk and Wear NHS Foundation Trust who recommend the use of a Weschler Intelligence scale for children - WISC V[47] and the administration of the TEACH.[48] The former assesses not only the intelligence quotient of the child, but also gives an indication of the working memory and processing speed of the YP. Lower scores in these domains could give rise to the suspicion of attention problems. Furthermore, the actual assessment process gives the psychologist the time to subjectively observe the level of attention, hyperactivity (ability to sit) and impulsivity (when answering questions) the YP displays in clinic. On the other hand, the TEACH is a computer test, which objectively measures various forms of attention e.g. sustained and joint attention.

The diagnosis of ADHD is then made at a MDT meeting where all the reports of the YP are brought together and discussed. This diagnosis is then made based on the chosen diagnostic criteria and level of functioning of the YP. The diagnosis is presented together with the strengths and weaknesses of the YP and recommendations are given at a feedback session, accompanied by a report to parents or care givers and YP.

Can a diagnosis be made in children under the age of 5?

Published literature suggests that neuropsychological symptoms of ADHD are present from birth, but the disorder is rarely diagnosed at preschool age. The reasons for this is that the brain is still undergoing neural pruning and developing cranio-frontally under the age of 5, therefore most children at the age of 3 will present with little executive function ability (which is derived from the pre-frontal cortex). As a result, the brain would appear to be almost all overactive and with a poor ability to concentrate and make rational decisions over impulsive ones. As a result, an early diagnosis would often result in a number of false positive results, furthermore, side effects from treatment with stimulants in under 5 year olds are as high as 33%, which is much higher than reported in school age children. However, research does report that ADHD symptoms could be identified as early as 15 months in females and 24 months in males.[49] Behavioural correlates to ADHD in preschool age children include difficult temperament and regulatory disturbances e.g. increased irritability, crying, hyperactivity and sleep problems.[50] A higher prevalence of externalising and internalising symptoms,[51] social problems,[52] learning problems[53] in preschool age children are all linked to an increased risk for developing and being given an ADHD diagnosis in later years.

NICE recommends that the first line of treatment for pre-school age children is parent-training and education programmes.[43] These programmes are the same as those recommended for the parents or carers of other children with conduct disorder. Drug treatment is not recommended for preschool children with ADHD.

Management

The combination of a number of interventions may have a role in managing children and adults with ADHD including pharmacological, neuropsychological and environmental interventions.[34] Methylphenidate is suggested as the first line of pharmacological treatments.[34,41] Other common treatments include Atomoxetine, and other amphetamines[34,41] such as Lisdexamfetamine a combination medication containing 25% levoamphetamne and 75% dextroampethamine. The latter is licensed for ADHD and narcolepsy but is also used as a performance enhancer in athletes and cognitive enhancer in students, apart from recreationally used as an aphrodisiac and euphoriant. However, other pharmacological treatments are used but with less supporting evidence, these include: alpha-2 agonists such as Guanfacine, Clonidine, antipsychotics such as Risperidone, Bupropion, Modafinil, and antidepressants with nor-adrenergic effects such as Amitriptyline, Imipramine, and Venlafaxine.[41] Management of ADHD must be delivered by a specialist in ADHD and is often done in combination with parenting groups such as the Incredible Years[53] and other non-pharmacological management; these include a range of psychological therapies e.g. Cognitive Behavioural Therapy (CBT), Behavioural Therapy, Family Therapy. Psychoeducation provided to the YP and parent is of outmost importance to ensure good understanding of the disorder, self-management of the symptoms and compliance to prescribed medication. This involves passing on the appropriate information and advice to parents and YP, informing GP, with school education of the care plan being provided. Special diets, avoidance of food colourings and exercise as well as other therapies such as neurofeedback should be discussed as part of the care plan.

In mild to moderate ADHD diagnosed in school age children, group-based parent training and education programmes are usually the first-line treatment. This may also include psychological treatment such as CBT and/or social skills training. Drug treatment is not indicated as the first-line treatment for all school-age children and YP with ADHD. It should be reserved for those with severe symptoms and impairment or for those with moderate levels of impairment who have refused non-drug interventions, or whose symptoms have not responded sufficiently to parent-training/education programmes or group psychological treatment.[43,54]

Pharmacological management

Stimulants (methylphenidate and amphetamines) - Why should I give my over active child a stimulant?

The use of psychomotor stimulants to treat the symptoms of ADHD has been reported in published literature as far back as 1937 by Bradley.[27] Bradley reported significant improvement in school performance after 1 week treatment of Benzedrine (stimulant) in 14 of 30 children with behaviour problems.[27] Later in 1957, methylphenidate hydrochloride was first used as a treatment for ADHD.[34] The efficacy for methylphenidate and amphetamines measured in Numbers Needed to Treat (NNT) is 4 and the effect size is 1.0, making this medication one of the most efficacious in all medicine.

There is little variety between the efficacy of slow release methylphenidate, with Concerta XL® having a NNT of 1.9 and Equasym XL® a NNT of 5.3, however, the lower NNT of Concerta XL® is thought to be a result of the poorer quality of the study design. The effect size of Atomoxetine is 0.7 with a NNT of 4.2, making it also a very efficacious medication. If a YP is diagnosed correctly with ADHD then the response rate with one of the stimulants is 95% (refer to Table 2[55]).

Methylphenidate is a central nervous system (CNS) stimulant. The mode of therapeutic action in ADHD is not exactly known.[56] However following on from the hypofrontality theory[24] it is understood that this medication stimulates the under developed dorsolateral prefrontal cortex in YP with ADHD, thereby increasing the concentration of the YP who is otherwise over attending to all diverse stimuli and providing negative neuronal feedback from the higher brain centre. This enables the YP in a classroom situation to follow the social norms and remain sitting down, rather than getting out of their place when this is not acceptable. At a neurotransmitter level, Methylphenidate blocks the presynaptic membrane dopamine transporter and thereby inhibits the re-uptake of dopamine and noradrenaline into the presynaptic neuron.[41]

Methylphenidate immediate release (Ritalin®) is a racemic mixture comprised of the d- and l-threo enantiomers.[56] This medication has a half-life of 3 to 4 hours, which means the YP must take at least 3 tablets to cover the school day and afternoon homework period. This medication has an on off effect, just like Paracetamol, and the medication is found to be out of the blood system within a few hours, therefore, there are no long-term side effects on the YP. However, the on off effect does mean that the blood level of Methylphenidate varies throughout the time in which it is effective, causing frustration to the YP who notices their concentration fluctuating throughout usage time.

The extended release form such as Metadate CD® comprises both of an immediate release component (30% of the dose) and an extended release component (70% of the dose)[55] or such as Concerta® which uses osmotic pressure to deliver methylphenidate hydrochloride at a controlled rate.[57] The extended-release form of methylphenidate was initially developed to address the issues of multiple dosing and compliance issues in children and adolescents. It was developed in the early 1980s.[57] Potential advantages for the extended-release methylphenidate might be the improved compliance and adherence to medication and avoidance of multi daily dosing. However, the 30/70% release formulation means that during

Table 2: Behaviours that reflect executive function impairments in adults[55]

Activation: organising, prioritising and initiating work
Focus: focusing, sustaining and shifting attention to tasks
Effort: regulating alertness, sustaining effort and processing speed
Emotion: managing frustration and regulating emotions
Memory: utilising working memory and accessing recall
Action: monitoring and self-regulating of activities

Table 3: Medication efficacy in psychiatry[65]

Medication Efficacy	Numbers Needed to Treat (NNT)	Effect Size (range 0-1)
Methylphenidate	4	1.0
Amfetamine	4	1.0
Atomoxetine	4	0.7
SSRI for depression in adults	10	0.5
Antipsychotics for Schizophrenia in adults	10	0.25

the morning when the YP may need to concentrate the most, is the time when the least concentration of methylphenidate is released into the blood stream, and only reaching its peak blood concentration at 12pm. Immediate release methylphenidate might have some advantages when it is used in older adolescents and adults for targeted situations such as important meetings/ events or exams.

Medikinet retard® and Equasm XL® are medium release preparations with half-lives of 8 hours, the former is released in a 50/50% release formulation, whilst the latter is released in a 30/70% formulation. The advantage of having an 8-hour preparation is that since insomnia and reduced appetite are major side effects of stimulants, not all children are awake for the full 12 hours and also, if Concerta XL® is given at 7am the YP would not develop an appetite till after 7pm, which means that the child would then have a late evening meal and then struggle to fall asleep.

Therapeutic effects of medication include improvements in ADHD symptoms, peer and family relationships, improved learning, self-esteem and social skills. A good response to methylphenidate is predicted in people with higher levels of inattention, restless behaviour and those of a younger age.

Clinical monitoring with medication include: pre-treatment, plotting height and weight on a growth chart, checking hearing clinically, assessing motor coordination and a cardiovascular examination. During treatment: measurement of weight and height, blood pressure and pulse are required.

What are the long-term side effects of taking methylphenidate?

There are no reported long-term side effects with methylphenidate, however, there are recently reported studies for the development of tolerance with very long-term use, therefore 'drug holidays' are recorded so as to reduce this risk. The commonest short-term side effects are sleep deprivation, this is avoided by not taking the medication after 4pm, and appetite suppression. xcv The latter is managed by encouraging the YP to take a good breakfast and then a large evening meal. Methylphenidate does not directly affect the growth potential of the child, however if the child is losing weight then there is a chance that the child will not achieve their potential height. Other common initial side effects include headaches, irritability, tics, tremor, dizziness, over stimulation and blurred vision, most of which disappear after a few weeks of taking the medication. Rare and dangerous side effects include psychosis, seizures, neuroleptic malignant syndrome, mania, palpitations, hypertension and sudden death.[57] There is some research on the potential for drug diversions and misuse, however Methylphenidate does not have a euphoric effect on YP that have ADHD and need this medication for therapeutic purposes.

Non-Stimulants

Atomoxetine increases noradrenaline and may also increase dopamine in the prefrontal cortex. It also blocks noradrenaline re-uptake pumps. Atomoxetine appears to be an efficacious treatment of children and adults with ADHD, and has a half-life of 24 hours so is prescribed as a once daily dose and is not a controlled drug. Its lack of abuse potential may be an advantage. The effect size is smaller (0.7) than that of the stimulants,[58] however in recent findings published by,[59] it was reported that once the controlling for parental reporting, the effect size of Atomoxetine was only 0.3 (which means a mild effect).

There are no long-term adverse effects of Atomoxetine, however the notable immediate effects include sedation and fatigue, a decrease in appetite (however this is less than for stimulants), an increase in heart rate of 6 to 9 beats per minute and an increase in blood pressure of 2 to 4 mmHg. Insomnia, anxiety, agitation, irritability, dizziness, nausea and vomiting are reported, however, these generally subside within 2 weeks of starting medication. Rare but potentially life-threatening side effects include orthostatic hypotension, hypomania, mania, suicidal ideation and very rarely liver failure.

Non-Pharmacological treatment

Non-pharmacological management for ADHD includes behavioural therapy, CBT, family therapy, social skills training, parenting groups, neurofeedback, special diets, avoiding eating food with artificial colouring from ones diet and supplementation with free fatty acids such as omega 3 and 6. This treatment could be used alone or in combination with stimulant medication and must be maintained over an extended period of time for more positive and long lasting results. The results on non-pharmacological studies remain mixed. In the large MTA follow up study[60] there was no difference found between those YP prescribed stimulant medication alone compared to those receiving a combination of medication and psychological treatment.[60] There are some small scale individual studies which demonstrate the benefit of omega 3 and 6 fatty acids,[61] cognitive training,[62] parenting groups[54] and removal of diets containing artificial colouring, however in a large and robust systematic review and meta-analysis, it was found that only in the case of food colouring was there some evidence to show the benefit on the treatment of ADHD symptoms and all the rest showed no difference when compared to placebo effect.[63]

Conclusion

ADHD is a common neurodevelopmental disorder which is possibly under-recognised in mental health settings and in community

both in children (5.29%)[18] and adults (2.5%).[18] ADHD is a highly heritable disorder 0.76 and parents and siblings of a child with ADHD are 4 to 5 times more likely to have ADHD.[30,31] ADHD is easy to treat with 95% of correctly diagnosed patients (children or adults) responding to treatment[11,64] Methylphenidate is suggested as the first line of pharmacological treatments.[34,41] For most patients with ADHD symptoms, these should be safely managed by the use of a single medication, however there is evidence where methylphenidate immediate release has been added to augment the effect of methylphenidate extended release and also that of Atomoxetine, when the clinical response remains inadequate. There is initial good evidence which suggests that stimulants and alpha-2 agonist combinations may have an additive effect, improving effectively and reducing adverse side effects. The evidence for non-pharmacological treatment of ADHD remains mixed,[63] there is some evidence to support the removal of food supplements from the diet of YP with ADHD and combination of psychological treatments and parenting groups with medication could have an added effect.[60] All general psychiatrists should be aware of this disorder and be comfortable with making the diagnosis and treating both children and adults with ADHD.

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Key points

- ADHD is a common neurodevelopmental disorder which is possibly under-recognised in children and adults
- ADHD is easy to treat with 95% of correctly diagnosed patients responding to treatment
- To ensure the accuracy, a diagnosis of ADHD should be made following a multimodal assessment which is carried out by a multidisciplinary team trained in ADHD
- In younger children there is evidence to support that parenting groups and psychological treatment is effective, although the evidence is mixed regarding if combination with medication could have an added effect
- All general psychiatrists should be aware of and comfortable with making the diagnosis of ADHD and treating both children and adults

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