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Comorbidity of Attention Deficit Hyperactivity Disorder and Post-Traumatic Stress Disorder

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Attention Deficit Hyperactivity Disorder (ADHD) and Post-Traumatic Stress Disorder (PTSD) show a high degree of comorbidity in traumatized children. Two hypotheses may help explain this relationship: children with ADHD are at higher risk for trauma due to their impulsivity, dangerous behaviors, and parents who may have a genetic predisposition for impairment of their own impulse control; and hyperarousal induced by severe trauma and manifested by hypervigilance and poor concentration may impair attention to create an ADHD-like syndrome. Four illustrative cases are presented, and implications for treatment are discussed.

KEY WORDS: Attention Deficit Hyperactivity Disorder; Post-Traumatic Stress Disorder; comorbidity; hyperarousal; treatment.

Attention Deficit Hyperactivity Disorder and Post-Traumatic Stress Disorder are complex clinical entities that pose diagnostic challenges to the clinician and have been the subject of significant debate regarding their nosological definition and validity. In addition, both of these disorders have significant comorbidity with other psychiatric disorders; however, there has been little attention given to comorbidity between ADHD and PTSD.

The diagnostic definition of ADHD has evolved over the last 30 years from "minimal brain damage" to "hyperactive child syndrome" to the more recent focus on attention as the primary symptom of the disorder. There

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is strong evidence from genetic and neurobiological studies that ADHD is a biologically based disorder (Zametkin, 1989). As currently defined, ADHD is characterized by symptoms of motor hyperactivity, impulsivity, poor concentration and distractibility which begin prior to age 7. Eight of 14 symptoms are required to satisfy DSM-III-R criteria (APA, 1987; see Table 1). Children with ADHD are frequently described as constantly in motion, "motor mouths," and unable to wait their turn or share toys. Parents often feel unable to manage them, and relationships with peers are usually difficult. Their low frustration tolerance and impulsivity frequently leads to temper tantrums and aggressive outbursts.

Barkley (1990) has estimated that the overall prevalence of ADHD in the childhood population is 5%, with a male to female ratio of approximately 3:1. Further, ADHD also occurs together with other psychiatric disorders. For example, Szatmari, Offord, and Boyle (1989) found that up to 60% of their sample had at least one other psychiatric diagnosis, 30% had two, and 10% had three other diagnoses. Conduct disorder was the most common comorbid diagnosis (40%), however, rates for other emotional disorders were also high (ranging from 16.7% to 50% in the different age groups). Munir, Biederman and Knee (1987) reported very similar results, with high rates of conduct disorder (36%), oppositional disorder (60%), major affective disorders (bipolar and unipolar, 32%) and anxiety disorder (27%).

In addition to this high degree of comorbidity, ADHD has been subtyped into different categories. For example, because a child does not have to demonstrate hyperactivity in order to fulfill the DSM-III-R criteria for ADHD, one method of subtyping is based on the presence or absence of hyperactivity. However, Barkley (1990) has suggested that attention deficit disorder without hyperactivity may represent "a separate and unique childhood psychiatric disorder" and not merely a subtype of ADHD. Others have described this group as hypoaroused, drowsy and daydreamy (Lahey, Schaughency, Hynd, Carlson, & Nieves, 1987). Hunt, Lau and Ryu (1991) have described another group of ADHD children at the other end of the spectrum who are severely hyperactive and hyperaroused.

PTSD is a relatively new addition to the psychiatric nomenclature first appearing as a diagnosis in 1980 (APA, 1980), with criteria specific to children being incorporated into the DSM-III-R (APA, 1987). PTSD occurs in response to a severe physical or emotional trauma which would be markedly frightening to anyone. Examples of such traumas include life-threatening accidents, rape, witnessing murder or other acts of severe violence, war, and natural disasters such as earthquakes. Symptoms of PTSD involve three major areas: intrusive memories of the event which are manifested by repeated and vivid memories, nightmares, flashbacks, and repetitive, post-traumatic play (in children); dissociative symptoms such as

numbing of responsiveness, withdrawal from activities, and amnesias; and symptoms of hyperarousal such as difficulty sleeping, poor concentration, irritability and anger outbursts. The onset of these symptoms may be immediate and acute, or delayed many months or years.

Although PTSD is precipitated by psychological trauma, research has revealed chronic central nervous system changes in PTSD patients. For example, Kosten, Mason, Giller, Ostroff, and Podd (1987) reported sustained urinary norepinephrine and epinephrine elevations in adult PTSD patients in comparison to other patient groups. In addition, there is evidence suggesting paradoxically reduced levels of corticosteroid in patients with PTSD (Bourne, 1970). In one of the few reports on PTSD in children, Ornitz and Pynoos (1989) studied the startle response in children with PTSD and found "a significant loss in the inhibitory modulation of startle response, suggesting that the traumatic experience had induced a long-term brainstem dysfunction (p. 866)."

The diagnosis of PTSD can be difficult to establish because denial and problems with recall are common in chronically traumatized patients. This may be particularly true with children: Traumatized children can be diagnosed with multiple different diagnoses depending on the child's state of mind on any given day (Terr, 1991). Possible diagnoses include conduct disorder, borderline personality, major affective disorder, attention deficit hyperactivity disorder, phobic disorder, dissociative disorder, among others. Seriously traumatized children with symptoms of hyperarousal and hypervigilance often have problems with attention and hyperactivity. These symptoms may be secondary to the trauma or reflect an underlying attention deficit disorder. Such children may require a significantly different clinical approach than non-traumatized children with ADHD. Four comorbid cases of ADHD and PTSD are presented for illustration and discussion. Tables 1 and 2 summarize the diagnostic criteria for ADHD and PTSD and how each of the patients presented meet the criteria.

CASE 1

Amy was a 12-year-old caucasian girl who was referred for evaluation of symptoms including poor concentration, inappropriate laughing, talking to herself, poor peer relationships and uncooperative behavior at home with frequent rage outbursts. Amy's mother also reported enuresis at home, but not at school. Amy's sleep was quite disturbed with frequent nightmares, sleep-walking and talking in her sleep.

Amy had been diagnosed with ADHD and a learning disability at age six years. She was treated with methylphenidate (unknown dosage) until

age nine with modest success. Her social skills were always very poor and her mother reported she was very difficult to manage. Her mother often allowed Amy's uncle to care for her even though she, herself, had a sexual relationship with him in her early teens. From age 9 to 11, Amy was sexually abused by her uncle. He gave her drugs (alcohol, LSD, cocaine), posed her for pornographic photographs and engaged in sexual acts with her. It was during this time that Amy's nightmares and enuresis began and her behavior problems worsened.

During the evaluation, Amy showed an array of symptoms including distractibility, impulsivity, making self-critical remarks, hearing thoughts "like voices" and seeing "visions" of stop-signs. Child Behavior Checklist and Conner's Parent Ratings indicated serious problems with attention, impulsivity and hyperactivity. Psychological testing revealed themes of people "both being nice and hurting you." There were indications of dissociative phenomena from both clinical interviews and psychological testing, to which her symptoms of laughing and talking to herself were attributed. There was no evidence of psychosis. Amy was also very withdrawn and difficult to engage. She met DSM-III-R criteria for both ADHD and PTSD (see Tables 1 and 2) and was subsequently treated with group and individual therapy by a private therapist in her community.

Table I. Summary of Case Symptomatology Meeting DSM III-R Diagnostic Criteria for Attention Deficit Hyperactivity Disorder

		Cases			
		1	2	3	4
A.	*Disturbance of at least 6 months with at least 8 of the following:*				
	1. Fidgeting/squirming	X	X	X	X
	2. Difficulty remaining seated	X	X	X	X
	3. Easily distractable	X	X	X	X
	4. Difficulty awaiting turns		X		X
	5. Blurts out answers				
	6. Difficulty following instructions	X	X	X	X
	7. Difficulty sustaining attention	X	X	X	X
	8. Shifts activities	X	X	X	X
	9. Difficulty playing quietly		X	X	
	10. Talks excessively				
	11. Interrupts/intrudes		X	X	X
	12. Not listening	X	X	X	X
	13. Loses necessary things	X			
	14. Physically dangerous activities		X	X	
B.	*Onset before age seven*	X	X	X	X
C.	*Does not meet criteria for pervasive developmental disorder*	X	X	X	X

Table 2. Summary of Case Symptomatology Meeting DSM III-R Diagnostic Criteria For Posttraumatic Stress Disorder

	Cases			
	1	2	3	4
A. *Traumatic event*	X	X	X	X
B. *Re-experiencing trauma (at least one)*				
1. Recurrent/intrusive recollections	X			
2. Recurrent distressing dreams	X	X	X	X
3. Acting/feeling as if event recurring		X	X	X
4. Distress when exposed to reminders		X		X
C. *Persistent avoidance of stimuli or numbing of responsiveness (at least three)*				
1. Avoiding associated thoughts/feelings	X	X	X	X
2. Avoiding associated activities/situations				
3. Inability to recall important aspects				
4. Diminished interest/developmental regression	X	X	X	X
5. Detachment/estrangement	X	X	X	X
6. Restricted affect	X			
7. Sense of foreshortened future				
D. *Persistent symptoms of increased arousal (at least two)*				
1. Difficulty falling/staying asleep	X	X	X	
2. Irritability/anger outbursts	X	X	X	X
3. Difficulty concentrating	X	X	X	X
4. Hypervigilance		X		
5. Exaggerated startle response		X	X	
6. Physiological reactivity with reminders		X		X
E. *Duration of disturbance at least 1 month with delayed onset (symptom onset at least 6 months after trauma)*	X	X	X	X

CASE 2

Bobby was a 5-year-old Caucasian boy referred for severe problems with attention, hyperactivity, temper tantrums and aggressive behavior. He had previously been diagnosed with ADHD and treated with multiple medications, including methylphenidate (up to 15mg twice a day), imipramine (up to 75mg daily), haldol (dosage unknown), and thioridazine (up to 50mg at bedtime), all with minimal success. Methylphenidate had caused an increased in agitation and irritability and had been discontinued. At the time of referral, he was taking imipramine (50mg at bedtime) and thioridazine (50mg at bedtime).

Bobby had a history of severe physical and sexual abuse. He was removed from his natural parents at 9 months of age for unknown reasons

and was placed in a foster home where sexual and physical abuse was documented. The sexual abuse had especially occurred at bath-time. He was removed at age three and placed in his current home. Bobby was virtually phobic of the bathroom, required much reassurance to use the toilet, and had to be bathed in the kitchen sink. He was also enuretic and frequently tried to engage other children in sexually aggressive play. Bobby's sleep was seriously disturbed. His foster parents felt he was detached and difficult to bond with even though he and his brother had been there for 2 years and adoption was actively being considered.

On examination, Bobby was constantly in motion and difficult to control. He shifted activities quickly, showed an exaggerated startle response and was easily distracted by external stimuli. He was hyperaroused and driven in his play. Bobby clearly met criteria for both ADHD and PTSD (see Tables 1 and 2). Due to concerns voiced by his foster-parents, the thioridazine and imipramine were tapered. his behavior worsened and clonidine was offered as an alternative medication. He showed decreased arousal and much improved sleep patterns as the medication was titrated up to 0.05mg four times a day. Bobby remained, however, highly impaired in his functioning.

CASE 3

Chris was a five-year-old boy who witnessed his father get seriously burned during a house fire. After watching this and losing all his possessions, he had to be left with a neighbor as an ambulance drove his mother and father to the hospital. He began to have nightmares about the fire and difficulty sleeping. he became enuretic and much more aggressive both at home and with peers. His parents felt they could not reach him or connect with him and Chris was frequently uncontrollable and violent.

During the evaluation, Chris was noted to engage in repetitive, anxious play about fires, while refusing to talk about the fire he had experienced. In a family meeting, Chris climbed on furniture dangerously and, when his mother asked him to stop, he attacked her and had to be restrained by the therapist. In addition, at times he was noted to have a short attention span and to be easily distracted. Conner's questionnaires were consistent with these observations. His parents reported Chris to be very active prior to the fire, but still within the range of normal. Subsequent to the fire, he showed serious attention problems. He was diagnosed with both ADHD and PTSD (see Tables 1 and 2). Play therapy and behavior management techniques were very effective in treating this child.

CASE 4

Donna was an 8-year-old girl who was repeatedly sexually abused by several men prior to age 5. Following removal to foster care, she disrupted several placements with sexually provocative and aggressive behaviors. Her foster parents described her as detached and difficult to bond with. Symptoms of PTSD at the time of referral included verbalized fears that someone would hurt her, going to sleep at night fully clothed, and fears of going to the bathroom alone. She avoided talking about the abuse, had recurrent nightmares of "bad people" who hurt her and her family, and repetitively played out sexual behaviors and acts in which smaller dolls were hurt or victimized by larger ones. She reported seeing things such as "the devil with my mother's face" on several occasions. She was also enuretic and encopretic. She was frequently noted to appear sad and reported occasional suicidal ideation, however, self-report measures of depressive symptoms were noted to be low to moderate. She was also noted to lack empathy towards others. She reacted intensely to mild frustration, tearing school books and clothing and scratching herself over minor mistakes in schoolwork. Although these post-traumatic symptoms were dramatic, Donna was referred for evaluation due to her teacher's concerns about inattention and impulsivity. Conner's Scales corroborated the clinical impression of ADHD.

Donna met DSM-III-R criteria for both ADHD and PTSD (see Tables 1 and 2). Her classroom behavior and ability to complete schoolwork improved markedly on methylphenidate (10mg in the morning and 5mg at noon), and placement in a small structured classroom. However, her sexual acting out, aggression and self-mutilation continued, leading to disruption of her local foster placement. Donna was moved to a new community where her therapist reported that she continued to manifest aggressive, destructive, and sexually provocative behaviors despite trials of several antidepressants, lithium and carbamazepine.

DISCUSSION

These cases portray traumatized children who meet the criteria for both ADHD and PTSD. They illustrate two possible mechanisms for the development of this comorbid relationship. The first case (Amy) gives a clear premorbid history of ADHD with a concomitant learning disability and illustrates how children with ADHD may be at higher risk for trauma. A single parent overwhelmed with managing a very difficult child accepted help offered by the man who subsequently abused her child. In addition, children with ADHD may place themselves at risk by their impulsivity and

their tendency to provoke angry responses from caretakers, most notably parents who may have a genetic predisposition for impairment of their own impulse control. It also follows that children with this background would have a poorer outcome after trauma than children with no prior psychiatric problems. These children have deficient coping strategies and defense mechanisms due to their attentional and associated cognitive impairment, rendering them more vulnerable to being overwhelmed by trauma and exacerbating their pre-morbid symptoms.

Cases 2, 3, and 4, however, do not show clear indications of either a premorbid history or family history of ADHD. Because this information is frequently not available, it is possible that these three children all have a genetic predisposition for ADHD. In case 3, for example, Chris could have had undiagnosed ADHD which was exacerbated by the traumatic event. His father had a history of school difficulties and temper outbursts which further supports this hypothesis. The genetic component for Bobby and Donna, however, cannot be established. We do know that their early lives were wrought with chaos and terror. Difficulties with attachment, relationships and affect modulation are prominent with both these children. Chronic trauma and severe secondary stressors (e.g., multiple foster placements) have created a complex psychosocial matrix in which these children must be viewed. The diagnostic difficulties with Bobby and Donna mirror those which Terr (1991) has described. As a group, Bobby, Chris, and Donna have remarkably similar symptomatology and closely resemble the children described by Hunt et al. (1991).

The lack of a premorbid history or available family history of ADHD raises the question of whether PTSD itself could create an attention deficit-like syndrome. The chronic central nervous system changes and post-traumatic symptoms which are associated with PTSD could be accounted for by neurobiological and information processing theories (Litz & Keane, 1989; Pitman, 1989). When these theories are applied to children, one could formulate a hypothesis for PTSD-related attention deficit symptoms. The human's "fight-flight" response in the face of impending danger causes an increase in the activity of the sympathetic nervous system manifested by symptoms of physiological arousal, such as a readiness to act, hypervigilance, and an increased heart rate and blood pressure (Guyton, 1976). Typically, such responses are adaptive in allowing the child to perceive and avoid potential trauma. However, this mechanism may become pathological in PTSD because the child is rendered helpless and unable to ward off the potential danger. Litz and Keane (1989) have described how this fear-related information may be encoded in the brain's memory to facilitate cognitive, motor and psychophysiological responding. They report clear evidence of hyperarousal in PTSD patients. Hunt et al. (1991) have suggested that hyperarousal can indirectly impair attention because the

child in a highly aroused state perceives many routine or trivial stimuli as significant. They hypothesized that the resulting excessive processing of stimuli leads to an overloading of the child's cognitive processes which impairs selective and sustained attention. The child with PTSD can thus be understood as constantly in surveillance of potential trauma, leading to the inability to filter out extraneous stimuli. The child's information processing abilities become overloaded. Ambiguous stimuli are perceived as threatening, producing a cascade of hyperarousal and anxiety which impairs attention and concentration. In addition, severe trauma occurring during preschool years might be hypothesized to be especially disorganizing, as children are already neurologically sensitive to overstimulation. This is a time of peak information processing abilities for the child; however, it coincides with reduced inhibition and increased facilitation of pathways associated with startle response (Ornitz, 1991).

The implications for treatment are significant. Although there are no clear, research supported guidelines for medication treatment in this population, when PTSD and hyperarousal symptoms predominate the clinical picture, the authors prefer to use clonidine or imipramine as first line medications. As in children with co-existing ADHD and anxiety disorders, traumatized children may have a poorer response to methylphenidate (e.g., Bobby). Of course there are many instances, as in Donna's case, where methylphenidate is used successfully. At times the combination of methylphenidate and clonidine can be helpful, addressing symptoms of both inattention and hyperarousal. In addition, there is also need for trauma-specific psychological modalities to resolve post-traumatic systems.

ADHD encompasses an extremely heterogeneous group of children and these cases may represent a distinct subcategory. In addition, there are other possibilities which could explain the co-existence of ADHD and PTSD. For example, because attention and concentration problems are common to most psychiatric disorders, if the criteria for ADHD are too broad, one would expect to find a great deal of comorbidity. Alternately, since ADHD and childhood trauma are both relatively common, perhaps the co-existence of these two disorders is a spurious finding. Further studies should focus on the frequency of this comorbidity and what, if any, criteria might distinguish these children as a distinct subgroup responding to specific treatment combinations.

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