State of the Art in the Management Of Post Traumatic Stress Disorder

Essay

Submitted for Partial Fulfillment of Master Degree in Neuropsychiatry

By

Nabil Nasif Saber El-Gendi
M.B., B.Ch.

Supervised by

Prof. Aida Seif El Dawla
Professor of Neuropsychiatry
Faculty of Medicine- Ain Shams University

Ass. Prof. Nivert Zaki
Assistant Professor of Neuropsychiatry
Faculty of Medicine- Ain Shams University

Dr. Marwa Abdel-Rahman Soltan
Lecturer of Neuropsychiatry
Faculty of Medicine-Ain Shams University

Faculty of Medicine
Ain Shams University
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الحديث فى علاج إضطراب كرب ما بعد الصدمة

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نبيــل نصيــف صابــر الجنــدي

بكالوريوس الطب والجراحة

تحت إشراف

أ.د. عايــدة سيــف الدولــة

أستاذ الطب النفسي

كليّةُ الطبِّ جامعةً عين شمس

أ..د. نيفــرت زكـــي

أستاذ مساعد الطب النفسي

كليّةُ الطبِّ جامعةً عين شمس

د. مـــروه سلطـــان

مدرس الطب النفسي

كليّةُ الطبِّ جامعةً عين شمس

كليـــة الطـــب
جامعـــةً عيـن شمـــس
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Contents

List of Abbreviations

5-HT	**:**	5- Hydroxy tryptamine.
ACC	**:**	Anterior Cingulate Cortex.
ASD	**:**	Acute Stress Disorder.
CAPS	**:**	Clinical Administered PTSD Scale.
CBT	**:**	Cognitive Behavioural Therapy.
CPT	**:**	Cognitive Processing Therapy.
CRH	**:**	Corticotrophin Releasing Hormone
CSF	**:**	Cerebrospinal Fluid.
Dex-CRH	**:**	Dexamethasone-Corticotrophin Releasing Hormone.
DSM -IV	**:**	Diagnostic and Statistical Manual of Mental Disorders.
DST	**:**	Dexamethasone Suppression Test.
DTS	**:**	Davidson Trauma Scale.
EMDR	**:**	Eye Movement Desensitization and Processing.
FDA	**:**	Food and Drug Administration.
fMRI	**:**	Functional Magnetic Resonance Imaging.
GAPA	**:**	Gama Amino Butyric Acid.
GR	**:**	Glucocorticoids Receptors.
HPA	**:**	Hypothalamic-Pituitary Adrenal Axis.
ICD-10	**:**	International Classification of Diseases 10.
IES	**:**	Impact of Event Scale.
LPS	**:**	Lipopolysaccharides.
MAOIs	**:**	Monoamine Oxidase Inhibitors.
m-CPP	**:**	m-chlorophenylpiprazine.
MRI	**:**	Magnetic Resonance Imaging.
MRS	**:**	Magnetic Resonance Spectrography.
NAA	**:**	N-Acetyl Aspartate.

PBMC	**:**	Peripheral Blood Mononuclear Cells.
PDS	**:**	Posttraumatic Diagnostic Scale.
PET	**:**	Proton Emission Tomography.
PHA	**:**	Phytohaemagglutinin.
PNSS	**:**	Positive and Negative Syndrome Scale.
PTSD	**:**	Post Traumatic Stress Disorder.
RCTs	**:**	Randomized Controlled Trials.
RTA	**:**	Road Traffic Accidents.
SAM	**:**	Sympathetic Adrenal-Medullary.
SCL-90	**:**	Symptom Checklist-90.
SDS	**:**	Sheehan Disability Scale.
SPECT	**:**	Single Photon Emission Tomomography.
SSRIs	**:**	Selective Serotonin Receptor Inhibitors.
TCAs	**:**	Tricyclic Antidepressants.
TOP	**:**	Treatment Outcome PTSD rating scale.
VBM	**:**	Voxel-Based Morphometry.

Introduction

Posttraumatic stress disorder (PTSD) is a condition marked by the development of symptoms after exposure to traumatic life events such as rape, war, terrorist attack or natural disasters. The person reacts to this experience with fear and helplessness, persistently relives the event, and tries to avoid being reminded of it .The lifetime incidence of PTSD is estimated to be 9 to 15 percent and the lifetime prevalence of PTSD is estimated to be about 8 percent of the general population, although an additional 5 to 15 percent may experience subclinical forms of the disorder. Among high-risk groups whose members experienced traumatic events, the lifetime prevalence rates range from 5 to 75 percent ***(Sadock B and Sadock V, 2007)***.

The term posttraumatic stress disorder was first used in the third edition (1980) of the Diagnostic and Statistical Manual of Mental Disorders, in which it was classified as an anxiety disorder ***(American Psychiatric Association, 1980).***

In this third edition, a diagnosis of PTSD required exposure to trauma that would provoke symptoms and signs of PTSD in almost everyone. In the 1987 revision of the Diagnostic and Statistical Manual of Mental Disorders, 10 diagnostic criteria were modified to emphasize the avoidance phenomena in which there are deliberate efforts to avoid thoughts, feelings, activities, and situations that simulated trauma recollection.

In the fourth edition (1994) of the Diagnostic and Statistical Manual of Mental Disorders PTSD remains an anxiety disorder American Psychiatric Association **(*Diagnostic and Statistical Manual of Mental Disorders1994*)**. There was a redefinition of the trauma. The traumatic event had to involve actual or threatened death or serious injury or a threat to the physical integrity of self or others. Also, the subject with PTSD had to respond with intense fear, helplessness, or horror. Furthermore, there had to be clinically significant distress and impairment in social, occupational, or other important areas of functioning for at least 1 month.

PTSD symptoms and signs are readily identifiable by the general internist. There is, however, considerable overlap between features of PTSD and other anxiety disorders, mood disorders, and substance abuse. Not uncommonly, patients with PTSD will be found to have several other psychiatric disorders. Specific questions must be asked about the occurrence of a traumatic event and subsequent onset of the clinical features of PTSD. Practitioners may be reluctant to ask about and patients may be reluctant to reveal distressing events that might involve shame or secrecy ***(Lange et al., 2002).***

Essential features of PTSD include:

(1) Exposure to a traumatic event that involved actual or threatened death or serious injury.

(2) Reexperiencing that event with distressing recollections, dreams, flashbacks, and/or psychologic and physical distress.

(3) Persistent avoidance of stimuli that might invite memories or experiences of the trauma.

(4) Increased arousal.

PSTD comprises an original traumatic event with the later emergence of a triad of symptom behaviors that includes avoidance/numbing, and hyperarousal/ hypervigilance of sufficient severity of re-experiencing to interfere with important aspects of the person's life. Internists, in their role as primary care providers, are likely to be the first health care professionals to see patients with PTSD ***(Lange et al., 2002).***

When the diagnosis of PTSD was introduced in 1980, traumatic events sufficient to induce this condition were considered rare ***(Schnurr et al., 2002).*** Since then, epidemiologic surveys have documented such events to be highly prevalent, with 50% to 90% of the population receiving exposure over the course of a lifetime ***(Schnurr et al., 1995; Breslau et al., 1996).***

Lifetime prevalence of PTSD is approximately 8% ***(Schnurr et al., 1995).***

Medical reports of recent military experiences in Afghanistan and Iraq described a striking increase in the prevalence of PTSD largely related to the number of firefights engaged in by the veteran ***(Hoge et al., 2004).***

Overall the most common traumatic events are witnessing a severe injury or death, involvement in a fire or other natural disaster, and involvement in a life-threatening accident (all 3 are more common in men than women) ***(Schnurr et al., 2002).***

Physical attack, combat-related trauma, being threatened with a weapon, captured, or kidnapped are also more common among men than women. Rape, sexual molestation, and parental neglect and physical abuse during childhood are more common among women than men. Note that trauma exposure meeting criteria for increased PTSD risk may be acute, such as assault or accidental injury, or chronic and recurrent, such as childhood abuse or domestic violence. Sexual differences in the type of trauma experienced also may drive aspects of PTSD. Women are more than 4 times more likely to develop PTSD than men after normalizing for the traumatic event. Rape is the trauma most likely to lead to PTSD and occurs more commonly among women (9%) than men (1%) ***(Schnurr et al., 2002).***

Other risk factors for PTSD include youth, lower socioeconomic status, preexisting psychiatric condition, childhood conduct problems, parental neglect, family history of a psychiatric condition, severity of reaction to initial trauma including dissociation, and poor social support system ***(Schnurr et al., 2002).***

Other anxiety disorders and depression in varying degrees of severity commonly accompany PTSD and may make the diagnosis of each condition more difficult ***(Schnurr et al., 2002).***

Substance abuse is a common condition developing after the onset of PTSD. Temporal discordance is the clearest evidence of comorbidity. That is, depression and/or an anxiety disorder preceding the traumatic event leading to PTSD and

substance abuse after the traumatic event offer the most compelling evidence for multiple psychiatric conditions ***(Boscarino, JA 1997).***

Not uncommonly, especially among older patients, medical conditions may be comorbid with PTSD. Exposure to trauma increases the risk of poor physical health **(*Schnurr et al., 2000*).**

It was reported that there is a direct link between trauma and a broad spectrum of medical conditions in a 20-year follow-up of men initially exposed to severe stress ***(Obscurant JA. 1997).***

PTSD subjects are more likely to have diabetes mellitus, heart disease, obesity, and osteoarthritis than their substance abuse counterparts ***(David et al., 2004).***

The American Psychiatric Association has recently published guidelines for the treatment of patients with PTSD ***(American Psychiatric Association. 2004).***

The first task is to reduce or eliminate the symptoms and signs of PTSD and any trauma-related comorbid conditions.

Next, the clinician seeks to improve adaptive functioning and return the patient to a psychologic state of safety and trust. Finally, general treatment focuses on limiting any generalization of the initial trauma and protecting the patient with PTSD against subsequent relapse.

In randomized clinical trials, most patients with PTSD treated with psychotherapy improve or recover ***(Bradley et al., 2005)*.**

Non pharmacologic models of treatment for PTSD are built on a biopsychologic understanding of this condition. ***(Brewin et al., 2003).***

Disturbances in PTSD are associated with a broad range of psychologic processes including attention, beliefs, cognitive-affective responses, coping styles, memory, and social-support systems ***(Brewin et al., 2003).***

After a traumatic event, no specific drug or combination of drugs prevents the emergence of acute stress disorder or later of PTSD. However, selective serotonin reuptake inhibitors are considered the first-line drug treatment for PTSD and the only ones to have received Food and Drug Administration indication ***(American Psychiatric Association, 2004)*.**

Selective serotonin reuptake inhibitors may reduce or eliminate the clinical features of the 3 symptoms clusters of PTSD (reexperiencing, avoidance/ numbness, and hyperarousal). Other antidepressants including tricyclic antidepressants, serotonin and norepinephrine reuptake inhibitors, and monoamine oxidase inhibitors may be of benefit in treating the patient with PTSD ***(American Psychiatric Association, 2004)*.**

Benzodiazepines may reduce anxiety and improve sleep in PTSD. They may not control or eliminate the core triad of PTSD ***(American Psychiatric Association, 2004).***

Rationale of the work

PTSD is a prevalent psychiatric disorder that may have important implications for school, social and academic function.

Psychopharmacological approaches to the treatment of PTSD have expanded over the past 20 years and increasing empirical evidence helps guide current clinical practice.

Aim of the work

1- Review incidence and prevalence of PTSD.
2- Review the diagnostic criteria of PTSD.
3- Review recent advances of the different treatment plans, both pharmacological and non pharmacological, for Posttraumatic stress disorder (PTSD).

Historical background

Posttraumatic Stress Disorder has been known under various names, especially throughout the military history, including the "irritable heart of the soldiers", "combat war neurosis" and "shell-shock".

These terms represent a complex of symptoms making the soldier unfit for further military combat and undermining the striking power of army units ***(Gersons and Carlier, 1992).***

Documented war experiences have provided an early description of the disorder among soldiers during the American Civil War (1861-1865). A combat soldier with palpitations and chest pain was said to have a functional cardiac disturbance which was called "soldiers heart" or "effect syndrome" ***(Kinzie, 1989).***

During World War I (1914-1918) the British army reported that no fewer than 7%-10% of the officers and 3%-4% of other ranks suffered mental breakdowns. In all 80,000 shell-shocked troops passed through the army hospitals. They were diagnosed as suffering from "shell-shock". On the grounds of this diagnosis, 200,000 troops were exempted for further active service. The English psychiatrists working on home ground, with the English Channel separating them from the front line, came to the conclusion of that traumatized soldiers exhibited signs of hysteria and suggested that the best remedy was to ignore it and not to talk about it. Unfortunately, but not surprisingly, this led to consolidation of the symptoms. Te shell

shock symptoms were anything but temporary, and the soldiers affected often simply disappeared into psychiatric institutions. In their despair at the unknown sickness before them, some doctors claimed that the cause lay in micro-sections of exploded bombs having entered the brain (which is where the term shell-shock comes from). It was not long, however, before others came forward with data showing that shell-shock also occurred in soldiers who had never experienced any kind of explosion, which meant that this direct explanation had to be abandoned. Other researches suggested that there was no question at all of a mental disorder but of stimulation and it was even proposed that these so called patients should be imprisoned or summarily executed ***(Gersons and Carlier, 1992).***

Theories of the disorder's psychological etiology began to compete with physical causation theory in the early 1900's. Under the influence of the psychodynamic theory, war neurosis was viewed as the result of the reactivation of an unresolved conflict in a predisposed individual ***(Kinzie, 1989).*** In other words the war trauma had served primarily to open a tin of libidinous worms, and had been reformulated in the term of the patient's early childhood emotional war with members of the family ***(Stone, 1985).***

Word War II (1939-1945) brought further clinical experience not only with combatant but also with civilians: survivors of prisoners of war camps, Nazi death camps and survivors of atomic bombing of Japan. An early description of symptoms among civilians caught in the disastrous Boston