Acute stress disorder in adults: Treatment overview

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INTRODUCTION

Acute stress disorder (ASD) is characterized by acute stress reactions that may occur in the initial month after a person is exposed to a traumatic event. The disorder includes symptoms of intrusion, dissociation, negative mood, avoidance, and arousal. Some patients who experience ASD go on to experience posttraumatic stress disorder (PTSD), which is diagnosed only after four weeks following exposure to trauma.

Treatment for ASD is aimed at curtailing symptoms of acute stress responses and preventing their development into PTSD.

The treatment of ASD is discussed here. The epidemiology, pathogenesis, clinical manifestations, course, and diagnosis of ASD are discussed separately. The epidemiology, pathogenesis, clinical manifestations, course, diagnosis, and treatment of PTSD are also discussed separately.

- (See "Acute stress disorder in adults: Epidemiology, clinical features, assessment, and diagnosis".)
- (See "Posttraumatic stress disorder in adults: Epidemiology, pathophysiology, clinical features, assessment, and diagnosis".)
- (See "Posttraumatic stress disorder in adults: Treatment overview".)
- (See "Posttraumatic stress disorder in adults: Psychotherapy and psychosocial interventions".)

APPROACH TO TREATMENT

We suggest trauma-focused cognitive-behavioral therapy (CBT) as first-line treatment of patients with acute stress disorder (ASD) rather than other psychotherapies or medication. There are no clinical trials comparing trauma-focused CBT with other interventions with efficacy in the treatment of ASD or prevention of posttraumatic stress disorder (PTSD). Trauma-focused CBT is the most extensively studied treatment for ASD with the most extensive evidence of efficacy. (See 'Trauma-focused CBT' below.)

CBT for ASD patients should typically be provided by a trained clinician over six weekly sessions of 60 to 90 minutes; additional sessions can be added if necessary. The intervention is typically delivered at least two weeks after trauma exposure. This allows the individual additional time for transient symptoms to abate and for post-trauma stressors to ease. The commencement of therapy should be timed with respect for other stressful events stemming from the trauma. The patient may find it difficult to focus attention on therapy if distracted by trauma-related events or experiences, such as pain, surgery, legal proceedings, relocation, or other stressors.

For some patients with ASD, it is necessary to delay exposure therapy for several months into the PTSD phase for others, including patients with:

- Extreme avoidance or dissociative responses, because these presentations may indicate extreme stress responses that may be complicated by exposure.
- A primary response of anger, because anger often does not respond optimally to exposure exercises and may respond better to cognitive therapy.
- An acute grief response, because exposure therapy may complicate normal grieving.
- Borderline or psychotic features, because these people require containment and exposure may complicate their presentation.
- Significant suicidal risk, because these patients require suicide management.
- Persisting PTSD responses to childhood trauma, because addressing childhood trauma weeks after a recent trauma may be excessively difficult.

Exposure, a component of trauma-focused CBT, has been found to be more effective when provided as monotherapy than cognitive restructuring in patients with ASD [1]. The trial randomly assigned 90 individuals who experienced trauma and met diagnostic criteria for ASD to receive imaginal and in vivo exposure, cognitive restructuring, or to a waitlist control group.

After six weeks of treatment, fewer patients in the exposure group met diagnostic criteria for PTSD as compared with those in the cognitive restructuring or waitlist groups. At six months, patients in the exposure group remained less likely to meet PTSD criteria and were 2.8 times more likely to achieve remission of their initial symptoms than patients in the waitlist group.

For patients with ASD and acute, intense anxiety, agitation, or sleep disturbance in the immediate period following the traumatic event, we suggest adjunctive treatment with a benzodiazepine rather than other medications. Clinical trials have not yielded sufficient evidence to determine the efficacy of benzodiazepines in comparison with placebo or with other medications in patients who have experienced acute trauma or have been diagnosed with ASD. In our clinical experience, benzodiazepines can reduce symptoms of anxiety, agitation, and sleep disturbance. (See 'Benzodiazepines' below.)

Benzodiazepine treatment should be limited to two to four weeks. Prolonged benzodiazepine treatment may be detrimental. As an example, clonazepam 0.5 to 2 mg/day in divided doses can be used.

PSYCHOSOCIAL INTERVENTIONS

Psychosocial interventions that have been tested either to treat symptoms of acute stress disorder (ASD) and/or to prevent the development of posttraumatic stress disorder (PTSD) include trauma-focused cognitive-behavioral therapy (CBT), exposure therapy, cognitive therapy, and psychological debriefing.

Trauma-focused CBT — Trauma-focused CBT for ASD typically includes patient education, cognitive restructuring, and exposure. (See "Posttraumatic stress disorder in adults: Psychotherapy and psychosocial interventions", section on 'Cognitive-behavioral therapy'.)

- **Patient education** – Patients are educated about stressful reactions to trauma, trauma-related disorders, and treatment options. Educating patients about stressful reactions to trauma should aim to:
  - Normalize the stress response
  - Heighten expectancy of recovery
  - Explain the stress responses in terms of conditioning models that require the patient to learn that reminders are no longer dangerous

- **Cognitive restructuring** – Cognitive restructuring is used to address maladaptive or unrealistic appraisals the patient may have about the trauma, his or her response to the event, and fears of potential future harm.

- **Exposure** – Exposure therapy assists patients in confronting their feared memories and situations in a therapeutic manner. Re-experiencing the trauma through exposure allows it to be emotionally processed so that it can become less painful [2,3]. By repeatedly confronting traumatic memories or safe reminders of a traumatic experience, the individual can experience them safely, until they no longer elicit such strong emotions and can see that they are not dangerous.

  Trauma-focused CBT for ASD optimally includes both imaginal exposure and in vivo exposure. In imaginal exposure, the patient provides a detailed narrative of his or her traumatic experience in which the patient orally relives the traumatic experience with the therapist (usually for at least 30 minutes), with the intention of achieving extinction learning. By reliving the memory repeatedly, the patient can learn that the salient reminder of the trauma (eg, the memory) is no longer a threat and does not result in aversive outcomes. Based on a similar mechanism, in vivo exposure is conducted to ensure that the patient is not avoiding feared situations in their daily life, thereby consolidating the learning that reminders are no longer signaling threat.

- **Administration** – Administration of trauma-focused CBT is discussed earlier in the topic. (See 'Approach to treatment' above.)

Efficacy for acute symptoms — Trauma-focused CBT reduces symptoms of ASD. A meta-analysis of three clinical trials with 93 patients with ASD found that trauma-focused CBT reduced ASD symptoms compared with control conditions [4-8]. As an example, a clinical trial of 30 patients with acute stress disorder compared cognitive processing therapy (a variant of CBT) with supportive counseling, finding no difference in primary outcomes in [9]. This could suggest that cognitive processing therapy is not as effective as other types of trauma-focused CBT for the disorder; however, our conclusions are tempered by the trial's small sample size and the absence of head-to-head trials of cognitive processing therapy and other trauma-focused CBT interventions.

Efficacy in preventing PTSD — Trauma-focused CBT has been found to reduce the likelihood of subsequent PTSD in people with acute stress disorder [4]. A meta-analysis of five randomized clinical trials found that CBT reduced the proportion of patients meeting diagnostic criteria for PTSD at six months (relative risk 0.56, 95% CI 0.42-0.76), with continued benefit seen at three years of follow-up. A subsequent meta-analysis of 10 randomized clinical trials found that CBT

to have a moderate effect size at initial follow-up (effect size = 0.54) that was reduced to a small effect size at extended follow-up (effect size = 0.34) [10]. A meta-analysis confirmed that CBT for prevention of PTSD was the most effective of early intervention strategies, with most trials focusing on people with ASD [11].

As an example, a clinical trial randomly assigned 24 patients with ASD following civilian trauma to receive five sessions of either CBT or supportive counseling within two weeks of the traumatic event [5]. At six months following their event, a smaller proportion of patients in the CBT group met diagnostic criteria for PTSD in the CBT group compared with the supportive counseling group (17 versus 67 percent).

Exposure therapy — Clinical trials have found exposure (without the other components comprising CBT) to be effective in preventing the development of PTSD. As examples:

- A clinical trial found that prolonged exposure administered to individuals meeting PTSD symptom criteria an average of a month following exposure to a traumatic event was more effective compared with a waitlist control in reducing PTSD at five months following treatment initiation but no more effective at nine months. Two hundred and forty-two patients were randomized to receive prolonged exposure, cognitive therapy, a waitlist control, escitalopram, or pill placebo [12]. Compared with patients in the waitlist group, patients receiving prolonged exposure were less likely to have PTSD after five months (57.1 versus 21.6 percent), but no difference between groups was seen at nine months.

- A clinical trial found that an early exposure-based intervention delivered in the emergency department to persons recently exposed to a traumatic event was successful in preventing PTSD. One hundred and thirty-seven patients were randomly assigned to receive three sessions of modified prolonged exposure or an assessment-only control soon after the trauma [13]. At follow-up 4 and 12 weeks post-trauma, prolonged exposure led to a greater reduction of PTSD symptoms compared with the control intervention. No difference was seen between prolonged exposure and control groups in the proportion of patients with PTSD at four weeks (54 versus 49 percent), but a greater proportion who received prolonged exposure did not meet criteria for a PTSD diagnosis compared with controls at 12 weeks (74 versus 53 percent).

Cognitive therapy — In addition to being a component of trauma-focused CBT, cognitive therapy has also shown evidence of efficacy in preventing PTSD as a monotherapy. In a trial of 242 patients randomized to one of five treatment groups, patients assigned to receive cognitive therapy were less likely to have PTSD after five months compared with patients in the waitlist

group (18.2 versus 58.2 percent), but no difference between groups was seen at nine months [12].

Psychological debriefing — Despite extensive use following disasters and other traumatic events, psychological debriefing (also known as "critical incident stress debriefing") has not been found to be effective in reducing PTSD symptoms among individuals experiencing a traumatic event [14,15]. The intervention involves recollecting, articulating, and reworking of the traumatic event, typically in a group format. Meta-analyses of numerous clinical trials found no evidence of effectiveness for either the initial, single-session intervention [14] or for subsequent, multiple-session versions [15].

PHARMACOTHERAPY

Medications that have been tested either to treat symptoms of acute stress disorder (ASD) and/or to prevent the development of posttraumatic stress disorder (PTSD) include selective serotonin reuptake inhibitors (SSRIs) and other antidepressants, benzodiazepines, propranolol, morphine, hydrocortisone, and docosahexaenoic acid.

SSRIs — Clinical trials have not found a benefit of SSRI treatment of ASD symptoms compared with placebo. SSRIs have been found to be efficacious in reducing symptoms in patients with PTSD [16]. (See "Posttraumatic stress disorder in adults: Treatment overview".)

- A clinical trial that tested multiple treatments compared escitalopram, an SSRI, with a pill placebo in 46 individuals who met PTSD criteria an average of a month following exposure to a traumatic event found no difference in subsequent PTSD prevalence rates between the two groups (61.9 versus 55.6 percent; odds ratio 0.77, 95% CI 0.21-2.77) [12].

- A clinical trial of 31 patients with full or partial ASD were randomly assigned to receive 10 to 20 mg of escitalopram or placebo for 24 weeks, and were followed until 56 weeks [17]. Both groups experienced a decline in PTSD symptoms; the reduction was greater in the placebo group compared with the SSRI-treated group.

- A clinical trial of 60 children, age 4 to 18, with ASD symptoms following severe burns, compared fluoxetine, imipramine, and placebo [18]. Following daily treatment for one week, no difference in response rates was seen among the three groups (72 versus 60 versus 55 percent). Trauma-related and depressive symptoms typically need between two and six weeks to respond to antidepressant treatment.

Other antidepressants — Clinical trials have found mixed results for imipramine in the treatment of children with ASD following severe burns. One week is shorter than the typical trial to test antidepressants; clinical response to these drugs usually requires two to eight weeks of daily treatment.

- A trial of 25 children (age 2 to 19) who met ASD criteria following severe burns, compared treatment with imipramine to chloral hydrate [19]. After daily treatment for seven days, patients were more likely to respond in the imipramine group compared with those receiving chloral hydrate (83 versus 38 percent).

- A clinical trial of 60 children (age 4 to 18) with ASD symptoms following severe burns, found no difference in treatment response to imipramine, fluoxetine, and placebo following daily treatment for one week (60 and 72 versus 55 percent) [18].

Benzodiazepines — Small, nonrandomized trials of benzodiazepine treatment for ASD and our clinical experience suggest that benzodiazepines may be helpful for acute anxiety, agitation, or sleep disturbance in the immediate period following the traumatic event. Prolonged use may be detrimental to adaptation. (See 'Approach to treatment' above.)

- A small, uncontrolled trial described benzodiazepine treatment of four patients, within one to three weeks of trauma exposure, who had ASD symptoms that included disturbed sleep [20]. Temazepam 30 mg was administered orally at bedtime for five days, followed by two days at 15 mg. The patients experienced improved sleep and reduced ASD symptoms.

- A trial compared 13 individuals treated with a benzodiazepine within one week of trauma exposure to 13 individuals with comparable trauma exposure who did not receive a benzodiazepine; participants in the two groups were pair matched for gender and symptom severity [21]. Ten patients were treated with clonazepam (mean dose 2.7 mg/day), and three were treated with alprazolam (mean dose 2.5 mg/day). Participants in the two groups did not experience mean differences in PTSD severity ratings at one or six months. Nine participants in the benzodiazepine group compared with three controls met PTSD diagnostic criteria at six months.

Propranolol — Propranolol has been tested in the immediate aftermath of trauma exposure, with the hypothesis that reducing noradrenergic activation would result in reduced conditioning of the trauma memories and prevent the development of PTSD. Although positive results were initially seen in an uncontrolled trial [22] and in subsequent testing of fear conditioning [23], the only randomized trial examining the development of PTSD symptoms was negative [24]. Negative results were also seen for gabapentin in that trial. A meta-analysis of five retrospective

and randomized trials indicated that propranolol was not effective in preventing the development of PTSD, with relative risk of 0.92 to prevent PTSD [25].

- Forty-one patients with ASD symptoms following a traumatic event were randomly assigned a 10-day course of propranolol or placebo [23]. The results reported a trend toward limiting subsequent fear conditioning in the propranolol group.

- Forty-eight patients who experienced an acute physical injury and were admitted to a surgical trauma center were randomly assigned to receive 14 days of treatment with propranolol, gabapentin, or placebo [24]. Neither medication showed a benefit compared with placebo in PTSD or depressive symptoms at one, four, or eight months postinjury.

- Forty-one patients who experienced a qualifying traumatic event were recruited from an emergency department and randomized to 19 days of propranolol or placebo [26]. There was no difference between conditions in PTSD severity at 4- or 12-week assessments, although there was tentative support for reduced psychophysiological reactivity to trauma reminders.

Morphine — Several uncontrolled studies have noted that morphine (which reduces norepinephrine), used for pain control in the initial 48 hours after trauma exposure, is linked to reduced subsequent PTSD symptoms, although the relationship between morphine and the development of PTSD has not been tested in randomized trials. They may point to the importance of pain management in preventing PTSD [27].

- A retrospective study of 696 United States military personnel who experienced physical trauma compared the rate of morphine use during early trauma care between those who subsequently developed PTSD and those who did not. Subjects with PTSD were less likely to have received morphine (odds ratio 0.48, 95% CI 0.34-0.68) [28].

- A study of 155 consecutive hospital admissions of patients after traumatic injury showed that those who developed PTSD had received lower doses of morphine than those who had not developed PTSD [29].

- A dose-related association between morphine use and subsequent development of PTSD was observed in an earlier analysis of 24 children who received inpatient medical care for treatment of acute burns [30].

Hydrocortisone — Findings and methods of clinical trials have been insufficient to suggest use of hydrocortisone as a treatment for ASD patients to prevent PTSD. A 2014 meta-analysis of four randomized clinical trials with 165 participants found moderate quality evidence for the efficacy

of hydrocortisone in preventing the onset of PTSD (relative risk 0.17, 95% CI 0.05-0.56), with a number need to treat between 7 and 13 patients to prevent the onset of PTSD in one patient.

Docosahexaenoic acid — A randomized clinical trial failed to show evidence of efficacy of docosahexaenoic acid (an omega-3 fatty acid) in the prevention of PTSD. The trial randomly assigned 110 patients who were admitted to an intensive care unit following a traumatic injury to 12 weeks of treatment with either docosahexaenoic acid or placebo [31]. Docosahexaenoic acid did not prevent the subsequent development of PTSD compared with placebo (11.1 versus 5.5 percent) and no difference was found in the severity of PTSD symptoms between the two groups at three-month follow-up.

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Trauma-related psychiatric disorders in adults".)

SUMMARY AND RECOMMENDATIONS

- For patients with acute stress disorder (ASD), we suggest first-line treatment with trauma-focused cognitive-behavioral therapy rather than other psychotherapies or medication (**Grade 2C**). (See 'Approach to treatment' above and 'Trauma-focused CBT' above.)

- Trauma-focused cognitive-behavioral therapy typically includes patient education, cognitive restructuring, and exposure. The goals of treatment are to reduce the severity of ASD symptoms and to prevent the development of posttraumatic stress disorder. (See 'Approach to treatment' above and 'Trauma-focused CBT' above.)

- Trauma-focused cognitive-behavioral therapy is typically delivered at least two weeks after trauma exposure. Some patients may benefit from further delay in starting exposure (eg, suicidal, acutely grieving, or highly avoidant following trauma) so that more urgent problems can be addressed. (See 'Approach to treatment' above.)

- For patients with ASD and intense anxiety, agitation, or sleep disturbance in the immediate period following the traumatic event, we suggest short-term (up to four weeks) treatment with a benzodiazepine (**Grade 2C**). (See 'Approach to treatment' above and 'Benzodiazepines' above.)

- Despite extensive use following disasters and other traumatic events, psychological debriefing (also known as "critical incident stress debriefing") has **not** been found to be effective in preventing PTSD among individuals experiencing a traumatic event.

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