



Treating trauma in psychosis with EMDR: A pilot study

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ABSTRACT

Background: Initial studies have shown that posttraumatic stress disorder (PTSD) can be effectively treated in patients with a psychotic disorder. These studies however used adapted treatment protocols, avoided direct exposure to trauma related stimuli or preceded treatment with stabilizing techniques making treatment considerably longer in duration.

Method: An open trial in which adult subjects with a psychotic disorder and a comorbid PTSD ($n = 27$) received a maximum of six Eye Movement Desensitization and Reprocessing (EMDR) therapy sessions. PTSD symptoms, psychotic symptoms and additional symptoms were assessed at baseline and end-of-treatment.

Results: The dropout rate was 18.5 percent (five subjects). Only five of the twenty-two completers (22.7%) still met criteria for PTSD after treatment. PTSD symptoms, auditory verbal hallucinations, delusions, anxiety, depression, and self-esteem all improved significantly. Paranoid ideation and feelings of hopelessness did not improve significantly. Treatment did not lead to symptom exacerbation in subjects. There were no adverse events, such as suicide attempts, self-mutilation, aggressive behavior or admission to a general or psychiatric hospital.

Conclusions: This pilot study shows that a short EMDR therapy is effective and safe in the treatment of PTSD in subjects with a psychotic disorder. Treatment of PTSD has a positive effect on auditory verbal hallucinations, delusions, anxiety symptoms, depression symptoms, and self-esteem. EMDR can be applied to this group of patients without adapting the treatment protocol or delaying treatment by preceding it with stabilizing interventions.

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1. Introduction

Between 50 and 98 percent of the adults with a Severe Mental Illness (SMI) such as psychosis had at least one traumatizing experience, with an average of 3.5 traumatic incidents per person (Frueh et al., 2005; Goodman, Rosenberg, Mueser, & Drake, 1997; Mueser et al., 1998). In the general population these rates are also high and vary from 39 to 81 percent (Breslau, Davis, Andreski, & Peterson, 1991; Creamer, Burgess, & McFarlane, 2001; Darves-Bornoz et al., 2008; Frans, Rimmö, Aberg, & Fredrikson, 2005; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). A literature review of childhood trauma in people with psychosis showed that 69 percent of women and 59 percent of men with psychosis were sexually or physically abused during childhood (Read, Van Os,

Morrison, & Ross, 2005). These and later traumatic experiences cause burden in adult life. The prevalence of comorbid post-traumatic stress disorder (PTSD) in people with psychosis varies from 11 to 52 percent (Achim et al., 2011; Buckley, Miller, Lehrer, & Castle, 2009; Frueh et al., 2005; Lu et al., 2011; McGorry et al., 1991; Meyer, Taiminen, Vuori, Aejjaelae, & Helenius, 1999; Mueser et al., 1998; Shaw, McFarlane, Bookless, & Air, 2002). This is quite high when compared to the general population where the prevalence varies from .4 to 3.5 percent (Creamer et al., 2001; Darves-Bornoz et al., 2008; Davidson, Hughes, Blazer, & George, 1991; Kessler, Chiu, Demler, & Walters, 2005; Perkonig, Kessler, Storz, & Wittchen, 2000).

Trauma and comorbid PTSD negatively influence the course of psychosis (Lysaker & LaRocco, 2008; Morrison, Frame, & Larkin, 2003; Mueser, Rosenberg, Goodman, & Trumbetta, 2002; Resnick, Bond, & Mueser, 2003). There appears to be a dose-response relationship. People who are repeatedly or more severely traumatized have a greater risk of developing psychosis (Galletly, Van Hooff, & McFarlane, 2011; Janssen et al., 2004; Read et al.,

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2005; Shevlin, Houston, Dorahy, & Adamson, 2008). People with psychosis and comorbid PTSD report more severe symptoms and are hindered more in their daily functioning than those without PTSD (Mueser, Lu, Rosenberg, & Wolfe, 2010). The PTSD symptoms negatively affect arousal levels and coping styles, increase the likelihood of substance abuse and of being revictimized, and lead to a decrease of trust in self and others. All these factors increase the odds of relapse in psychosis (Mueser et al., 2002).

Despite the reported high prevalence rates, both trauma and PTSD are overlooked in the majority of people with psychosis. (Howgego et al., 2005; Lommen & Restifo, 2009; Mueser et al., 1998). After diagnosing a severe mental illness such as schizophrenia, psychologists and psychiatrists appear not to be inclined to determine whether there are additional disorders. Clinicians use “trumping rules” in establishing diagnoses (Cusack, Wells, Crubaugh, Hiers, & Frueh, 2007). Other reasons, such as more pressing issues, fear of disturbing the client or exacerbating psychiatric symptoms, and not feeling equipped to ask about and respond to trauma are also thought to be involved in the high rate of underdiagnosis. Moreover having strong biogenetical beliefs about psychosis decreases the likelihood that clinicians assess trauma history (Frueh, Cusack, Grubaugh, Sauvageot, & Wells, 2006; Young, Read, Barker-Collo, & Harrison, 2001).

In the event that a comorbid PTSD is diagnosed in people with SMI, treatment is often not provided. Fear of symptom exacerbation and a lack of competence, confidence and belief in treatment interventions are probably the most important reasons to withhold treatment from these patients (Frueh et al., 2006; Salyers, Evans, Bond, & Meyer, 2004).

People with psychotic disorders are usually excluded from scientific studies into the efficacy of PTSD treatments (Spinazzola, Blaustein, & Van der Kolk, 2005). Therefore, little is known about the usability of these treatments in people with psychosis (Bradley, Greene, Russ, Dutra, & Westen, 2005). However, not treating PTSD in people with SMI might have adverse effects on wellbeing and prognosis (Howgego et al., 2005; Lysaker & LaRocco, 2008). Not treating severe PTSD in patients without psychosis is found to have significantly more negative consequences than treating the disorder with Cognitive Behavioral Therapy (CBT) including prolonged exposure (Cloitre, Koenen, Cohen, & Han, 2002). Patients with psychosis used to be excluded from psychotherapeutic treatments. We now know however that CBT for psychosis is an efficacious and safe treatment. Symptoms decrease as a consequence of treatment (Jones, Cormac, Silveira da Mota Neto, & Campbell, 2010; NICE, 2010; Pilling et al., 2002; Tarrier, 2005). Clinically we see an increased awareness of the importance of trauma in the life of people with psychosis and increasingly patients are starting to ask for treatment for the consequences of the traumas that they experienced (e.g. Helen, 2011). Little is known however about the treatment of PTSD in people with psychotic disorders.

A Randomized Controlled Trial (RCT) on 108 people with SMI showed that a cognitive-behavioral treatment of PTSD that was predominantly based on cognitive restructuring was more effective than treatment as usual in reducing PTSD symptoms and negative trauma related cognitions (Mueser et al., 2008). There were no adverse events, e.g. suicide attempts or need for admission. However, only 15.7 percent of the subjects had schizophrenia or schizoaffective disorder in this study.

Trappler and Newville (2007) conducted a similar study. Twenty-four patients with schizophrenia and comorbid PTSD underwent a 12 week Skill Training In Affect Regulation (STAIR; Cloitre et al., 2002). These subjects were compared to a matched comparison group receiving supportive therapy. Exposure

techniques were however not incorporated in the STAIR programme, because the authors assumed that this would be counterproductive (p.322). Instead treatment focussed on topics like arousal management and safety issues. Not surprisingly, considering the absence of exposure, treatment had no effect on PTSD symptoms such as intrusions about the experienced traumas.

Experiencing psychosis and contact with the healthcare system can be a traumatic event for many individuals (McGorry et al., 1991; Shaw, McFarlane, & Bookless, 1997; Shaw et al., 2002). Jackson et al. (2009) performed an RCT ($n = 66$) in subjects that had recently experienced a first episode of psychosis. Cognitive Recovery Intervention (CRI), aimed at improving recovery after a first episode of psychosis, was compared to treatment as usual. CRI had a maximum of 26 sessions. Key components of CRI are: (a) engagement and case formulation; (b) processing (the trauma of) experiencing psychosis through cognitive therapy; and (c) changing appraisals of psychosis (Jackson & Iqbal, 2000). Subjects receiving the CRI had less postpsychotic trauma symptoms at end-of-treatment assessment than the subjects in the control condition.

Frueh et al. (2009) conducted a pilot study with prolonged exposure treatment in patients with schizophrenia or schizoaffective disorder ($n = 20$). They used an eleven week exposure based CBT programme. A maximum of eight individual two-weekly exposure sessions were preceded by fourteen two-weekly group therapy sessions which comprised psycho-education, anxiety management, social skills training, anger management and trauma issues management. The number of dropouts was high (35%). All dropouts occurred during the group therapy phase of treatment. All subjects that started the individual exposure therapy finished this part of the treatment protocol. At three month follow-up ten out of the thirteen completers no longer met criteria for PTSD. Treatment caused no adverse events. Unfortunately psychosis measures were not included in this study (Frueh et al., 2009). The treatment protocol that was used in this study, including the exposure, could be applied effectively in the subjects with psychosis without any adjustments (Long et al., 2010).

Eye Movement Desensitization and Reprocessing (EMDR) is a third psychotherapy with strong empirical support for its efficacy in treating PTSD (Bisson et al., 2007; Bradley et al., 2005; Seidler & Wagner, 2006). One study used EMDR in acutely admitted patients with schizophrenia. Kim et al. (2010) compared the efficacy of three sessions of EMDR ($n = 45$) to treatment as usual and progressive muscle relaxation. There were no differences in measures of psychosis and depression between groups at three month follow-up. However, PTSD and post traumatic stress symptoms were neither diagnosed nor specifically targeted in this study. Subjects were simply asked about stressful life events, which were then targeted and desensitised with EMDR. It is questionable whether this procedure can be expected to produce any additive effect in the treatment of acutely admitted patients.

This paper reports about an uncontrolled feasibility pilot study on the effects of EMDR in patients with a psychotic disorder and a comorbid PTSD. This feasibility study was conducted as a pilot for a large RCT that is in preparation. Unlike certain previous studies we did not adapt the treatment protocol, there is no pre-treatment stabilisation phase, and the protocol does not avoid direct exposure to trauma related stimuli. Therefore this pilot gives an indication of whether patients with lifetime psychotic disorder and comorbid PTSD can be treated effectively with routine treatments for PTSD as recommended by the guidelines. We also measured psychotic symptoms in order to examine the effect of treatment on the present psychotic symptoms.

2. Method

2.1. Study design

An open pilot trial where PTSD symptoms are treated with EMDR in subjects with a diagnosis of PTSD and a lifetime schizophrenia spectrum disorder.

2.2. Setting

Subjects were outpatients from four secondary mental health services in The Netherlands: Parnassia Psychiatric Institute, BavoEuropoort Psychiatric Institute, Pro Persona Psychiatric Institute, and GGZ Noord-Holland-Noord.

2.3. Subjects

The inclusion criteria were:

- 1) A chart diagnosis of schizophrenia spectrum disorder,
- 2) A current PTSD.

Exclusion criteria were:

- 1) Younger than eighteen years of age;
- 2) Estimated IQ below 70;
- 3) No competence of the Dutch language.

2.4. Measurement instruments

2.4.1. Primary outcome measure: posttraumatic stress disorder

The Clinician Administered PTSD Scale (CAPS; Blake et al., 1990, 1995) was used to determine current PTSD. The CAPS does not only determine whether the symptoms meet DSM-IV criteria for an actual PTSD, it also produces an interval score for the severity of the PTSD (range 0–136). Also subscale scores for re-experiencing, avoidance and arousal are available. PTSD symptoms were assessed for the trauma that caused the most distress in the previous week. The end-of-treatment administration of the CAPS was based on this same trauma. In the case of multiple traumas within the same domain and with the same perpetrator, subjects were asked to consider these as one event. The CAPS has good psychometric properties (Pupo et al., 2011; Weathers, Keane, & Davidson, 2001).

The PTSD Symptom Scale Self-Report (PSS-SR) consists of 17 items about PTSD symptoms in the previous week that are scored on a 4-point scale. The cumulative score gives an indication of the severity of the PTSD. The PSS-SR has a satisfactory validity and reliability (Foa, Riggs, Dancu, & Rothbaum, 1993).

2.4.2. Secondary outcome measures

The Psychotic Symptom Rating Scales (PSYRATS) is a measure for psychotic symptoms in the previous week and consists of two short structured interviews, the Auditory Hallucination Rating Scale (AHRS, 11 items) and the Delusion Rating Scale (DRS, 6 items). The PSYRATS has shown to be valid and reliable, and is sensitive to change (Haddock, McCarron, Tarrier, & Faragher, 1999).

The Green et al. Paranoid Thought Scale (GPTS; Green et al., 2008) is a multidimensional self-report measure of paranoia. It consists of thirty-two statements about experiences in the last month, sixteen items about ideas of persecution and sixteen items about ideas of social reference. Items are scored on a Likert scale ranging from 1 (not at all) to 5 (totally). A higher score indicates

higher levels of paranoia. The GPTS is valid, reliable and is sensitive to clinical change (Green et al., 2008).

The Beck Depression Inventory second edition (BDI-II; Beck, Steer, & Brown, 1996) consists of twenty-one items. A higher score indicates more severe depression symptoms. The psychometric properties of the BDI have been extensively tested. It is a reliable test with internal consistency and concurrent validity with other clinical ratings (Beck, Steer & Garbin, 1988). The BDI-II appears to have similar properties (Beck, Steer, Ball, & Ranieri, 1996).

The Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988) is a 21-item self-report of the severity of anxiety in psychiatric populations with a high internal consistency and acceptable test-retest reliability (Fydrich, Dowdall, & Chambless, 1992).

The Beck Hopelessness Scale (BHS; Beck, Weissman, Lester, & Trexler, 1974) is a self-report with 20 statements about the future. Items are scored as true or false. A higher score indicates a higher degree of hopelessness. The BHS has a high degree of internal consistency and concurrent validity with clinical ratings and other measures of hopelessness.

The Self-Esteem Rating Scale-Short Form (SERS-SF; Lecomte, Corbière, & Laisné, 2006) is a self-report devised for people with schizophrenia. The SERS-SF has two subscales, positive and negative self-esteem. The total SERS-SF score is calculated by subtracting the negative self-esteem score from the positive self-esteem score. The total score can therefore be either positive or negative. The scale has an adequate convergent validity, high internal consistency, and high test-retest reliability in patients with schizophrenia (Lecomte et al., 2006).

2.5. Adverse events

In every session, adverse events, such as symptom exacerbation, suicidal ideation, alcohol and drug abuse, crisis contacts with casemanagers, or admission to hospital in the previous week were monitored by the therapist. In case of the occurrence of an adverse event, the patient was asked whether he himself thought that the event was related to the treatment.

2.6. Procedures

After referral to the study, subjects were screened for PTSD with the CAPS. If PTSD was diagnosed and informed consent was obtained, the remaining baseline measurements were administered. Subjects were then treated with six weekly EMDR sessions of 90 min. After a maximum of six sessions, post-measurements were taken. During the EMDR therapy treatment as usual, i.e. case management, was continued. Therapists worked in the same team as the case managers and kept contact on a weekly basis about the progress.

In total, ten therapists participated in the study. All therapists were health and clinical psychologists with extensive experience in CBT for psychosis and at least a two-year post-doctoral clinical specialization. All the therapists received training in EMDR. EMDR competence levels varied from little (completed less than five EMDR treatments) to extensive experience (completed more than a hundred EMDR treatments). Treatment integrity checks were not made. The first author had contact with all therapists about progress and gave consultation on the protocol when required.

2.7. Eye movement desensitization and reprocessing

We used the standard eight-phase EMDR procedure as adapted into Dutch (De Jongh & Ten Broeke, 2003). See Shapiro (1995) for a detailed description of the EMDR procedure. EMDR is a treatment

procedure which is aimed at reducing the negative influence of traumatic memories or intrusions. In this procedure the patient is asked to isolate a visual representation (a single picture) of a traumatic memory. The therapist and patient determine what belief statement currently applies to that target image, e.g. “I am powerless”. The patient is then asked to form a contradictory belief statement that he would prefer, e.g. “I am now in control”. The actual desensitization then starts. Tension is built up by asking the patient to visualize the target image, pronounce the negative statement (e.g. “I am powerless”) and concentrate on the accompanying physiological experiences. The patient is then instructed to concentrate on a distractive stimulus, usually the therapists fingers. After a short period of distraction the patient is asked to briefly associate about what comes to his awareness. A new distractive stimulus is then presented. This procedure is repeated until no new associations come to the patients awareness. The patient is then asked to focus on the target image, after which Subjective Units of Disturbance (SUD) scores are asked. When the SUD score has gone down to nil, the installation phase is started. The positive contradictory statement is then ‘installed’. The patient is asked how valid the positive cognition feels at that moment (1 = completely untrue to 7 = completely true). If the validity of this cognition is not yet 7, the patient is asked to concentrate on the target image and pronounce the positive statement after which a short distractive stimulus is presented. This procedure is repeated until the positive cognition feels completely true. Some additive procedures may then be performed such as a body scan (feeling whether there is still any tension in the body) or the installation of a future template (transferring the new information to an in vivo situation that is avoided). The EMDR ends with a positive closure in which the patient is asked to verbalize the most positive thing that he has learned about himself that session in respect to the trauma that was treated.

We did not modify the treatment and did not precede it with stabilizing techniques. The EMDR was primarily focussed on the trauma that caused the current PTSD. We used the ‘first method’ approach to identify targets for desensitization (De Jongh, Ten Broeke, & Meijer, 2010). This means the starting point for the case conceptualization was an inventory of the PTSD re-experiencing symptoms that caused most of the burden in the previous weeks. The therapist and subject then decided together which re-experiencing symptom to treat first. The traumatic events related to this symptom were graphed on a timeline. The relevant memories were identified and the subject was asked to what extent a confrontation in the here and now with each memory caused distress. After this the basic protocol was applied to the most distressing target memory. After desensitization of a target, symptoms were reevaluated and a new target (if present) was chosen and desensitized.

2.8. Statistical analysis

Descriptive statistics were produced to describe the demographic characteristics and baseline variables of the total sample. Paired within samples *t*-tests for means were performed to determine the statistical significance of the changes in scores on the various measures. Effect sizes were calculated according to Cohen (Cohen, 1992). Separate completers and intention-to-treat analyses were made. In the intention-to-treat analyses we used ‘last observation carried forward’ to impute missing post-treatment data in the dropouts.

The baseline scores on the DRS, AHRS and PSYRATS were not normally distributed and therefore non-parametrically analysed using Wilcoxon Signed Rank Tests. Adverse events were determined with descriptive statistics.

3. Results

Thirty-eight patients suspected of PTSD were referred to the study. Eleven patients did not meet the criteria for participation: 7 patients did not meet PTSD criteria on the CAPS; 1 patient did not have a diagnosis in the schizophrenia spectrum; 3 patients met inclusion and diagnostic criteria but did not want to participate in the study. The first of these patients was diagnosed with cancer shortly after referral. The second patient did not want to give an informed consent, and the third did not want treatment after experiencing an increase in stress after talking about his traumas.

Twenty-seven patients enrolled in the study. Table 1 displays the demographic characteristics of these twenty-seven subjects. Five subjects (18,52%) prematurely stopped treatment. The reason for stopping were: 1 could not believe the rationale of the treatment; 1 stopped treatment after no improvement after three sessions; 1 was abroad for several months; 1 withdrew consent after a significant improvement of his PTSD symptoms after two sessions; 1 did not show up at sessions.

3.1. Efficacy

The results are shown in Table 2A (intention-to-treat) and Table 2B (completers). On average 4.72 sessions of EMDR were provided to each subject. Only five of the twenty-two completers (22.7%) still met diagnostic criteria for PTSD at end-of-treatment. The CAPS PTSD intensity score was reduced with 42.4% in the intention-to-treat analysis and with 52.6% in the completers analysis. All sub scales improved significantly. PTSD symptom scores

Table 1
Demographic characteristics of subjects.

		Frequency (n = 27)
Mean age	45.00 (SD = 9.37)	
Mean duration of psychotic symptoms	13.48 years (SD = 12.07)	
Mean duration of PTSD symptoms	13.50 years (SD = 10.25)	
Trauma causing PTSD	Sexual abuse	6
	Physical abuse or physical threatening	8
	Emotional abuse	1
	Experiences during psychosis or treatment	8
	Other causes such as accidents or experiencing war	4
Sex	Male	15
	Female	12
Living status	With partner and child(ren)	5
	With partner	1
	Alone, independently	16
	In sheltered living	4
	Homeless	1
Ethnicity	Dutch (western)	17
	Non-western immigrant	10
Psychotic disorder	Schizophrenia	6
	Schizoaffective disorder	6
	Delusional disorder	1
	Psychotic disorder not otherwise specified	14
Substance or alcohol	Abuse	2
	Dependency	3
Medication	Antipsychotic medication	25
	Antidepressant medication	8
	Lithium	3
	Benzodiazepines	10

Table 2
A: Paired samples *t*-test statistics for the mean changes between baseline and end-of- treatment. Intention-to-treat analysis with last observation carried forward ($n = 27$).

	Baseline Means (sd)	End-of-treatment Means (sd)	<i>t</i>	Sig. (2-tailed) $\alpha = .005$	Effect Size Cohen <i>d</i>
CAPS total score	72.89 (19.41)	41.96 (29.29)	6.03	.000	1.16
CAPS section B	23.70 (7.40)	11.11 (11.46)	6.07	.000	1.17
CAPS section C	27.96 (9.45)	16.30 (11.77)	5.42	.000	1.04
CAPS section D	21.22 (6.62)	14.56 (8.56)	4.48	.000	.86
PSS-SR total score	29.81 (9.57)	20.37 (11.96)	5.40	.000	1.04
BDI-II	28.30 (9.30)	22.04 (10.66)	4.41	.000	.85
BAI	48.41 (13.83)	41.74 (14.55)	4.09	.000	.79
BHS	10.41 (2.53)	8.89 (5.75)	1.32	.198	.25
SERS-SF	.46 (21.97)	5.15 (23.57)	-2.16	.041	.42
GPTS	73.04 (35.66)	67.92 (35.72)	1.51	.144	.31
B: Paired samples <i>t</i> -test statistics for the mean changes between baseline and end-of-treatment in completers ($n = 22$).					
CAPS total score	72.14 (21.23)	34.18 (26.59)	7.26	.000	1.55
CAPS section B	23.91 (8.19)	8.45 (11.05)	7.34	.000	1.56
CAPS section C	27.50 (9.51)	13.18 (10.35)	6.26	.000	1.33
CAPS section D	20.73 (6.98)	12.55 (7.99)	4.91	.000	1.05
PSS-SR total score	29.40 (9.82)	19.28 (11.95)	6.23	.000	1.33
BDI-II	28.95 (10.00)	21.27 (11.53)	4.81	.000	1.03
BAI	47.68 (14.14)	39.50 (14.14)	4.40	.000	.94
BHS	10.00 (2.07)	8.14 (5.92)	1.33	.199	.28
SERS-SF	.76 (22.07)	6.57 (23.82)	-2.20	.040	.48
GPTS	72.11 (35.07)	65.63 (34.84)	1.52	.145	.35

CAPS: Clinician Administered PTSD Scale; CAPS section B: Re-experiencing symptoms; CAPS section C: Avoidance symptoms; CAPS section D: Arousal symptoms; PSS-SR: PTSD Symptom Scale Self-Report; BDI-II: Beck Depression Inventory-second edition; BAI: Beck Anxiety Inventory; BHS: Beck Hopelessness Scale; SERS-SF: Self-Esteem Rating Scale-Short Form; GPTS: Green et al. Paranoid Thought Scale.

also significantly improved on the PSS-SR. Both the intention-to-treat analysis and de completers analysis showed comparable results. The effect-sizes are reduced in the intention-to-treat analysis, but the significant changes still have a large effect-size.

Auditory verbal hallucinations and delusions were not normally distributed in our sample because not all subjects had hallucinations or delusions at baseline. Only eight subjects experienced auditory verbal hallucinations weekly and only 5 subjects had active delusions. In stead of aggravating psychotic symptoms, treating PTSD had a positive effect on delusions and auditory verbal hallucinations in the completers. Wilcoxon Signed Rank Tests showed small but significant reductions in delusional symptoms on the DRS ($z = -2.02$, $p < .043$, $r = .30$), in auditory verbal hallucinations on the AHRS ($z = -2.17$, $p < .030$, $r = .33$), and in total PSYRATS scores ($z = -2.67$, $p < .008$, $r = .40$). The intention-to-treat analyses showed the same *Z* and *P* scores, only the effect sizes were smaller due to the larger sample size, DRS ($r = .28$), AHRS ($r = .30$), and PSYRATS ($r = .36$).

Depression symptoms (BDI-II), anxiety symptoms (BAI), and self-esteem (SERS-SF) all improved significantly from pre- to post-treatment in both the completers and the intention-to-treat analysis. In both analyses there was no significant effect on feelings of hopelessness (BHS) and paranoid ideation as measured with the GPTS.

Fig. 1 shows the auditory verbal hallucinations before and after treatment. Five patients stopped hallucinating after treatment with EMDR, while 3 patients reported persistent hallucinations. Both sub groups were comparable on the clinician administered post-traumatic stress disorder scale at baseline ($t = -1.24$; $p = .26$). The mean change in clinician administered posttraumatic stress disorder scale scores in the recovered group was 53.6 points. In the group with persistent voices the mean change was 9.7 points. This difference is statistically significant ($F(1) = 36.67$; $p = .002$).

3.2. Safety of EMDR treatment

The treatment produced stress or a temporary increase in PTSD symptoms in some subjects. Three of the 124 sessions were spent on coping skills because the subject reported exacerbation of

symptoms. In all three incidences one session was enough to help the subject regain control and maintain motivation for treatment. It happened twice that a subject contacted his case manager to discuss increased arousal. In both incidences a conversation, in which information about EMDR treatment and possible temporary side effects was given, helped to reassure the subject. One subject with a history of drug abuse had a single relapse in hard-drug use during treatment after he left the house on his own for the first time in years. There were no suicide attempts and no incidences of self-mutilation or aggression toward others. There were no admissions in general or psychiatric hospital.

4. Discussion

4.1. Outcome

The results of this open trial show that it is effective and safe to treat posttraumatic stress disorder in subjects with schizophrenia spectrum disorder using eye movement desensitization and reprocessing. Moreover treating posttraumatic stress disorder has a positive effect on other symptoms. Auditory verbal hallucinations, delusions, anxiety and depression decrease. Self-esteem also improves. Although treatment can definitely produce some stress for a small minority of the subjects, as it does in other patients with posttraumatic stress disorder, it is generally safe and does not lead to adverse events. The dropout rate was 18.5% in this study. This rate is comparable to other psychological therapies in psychosis and posttraumatic stress disorder and is quite low when compared to dropout rates in medication trials in patients with psychosis (Wahlbeck, Tuunainen, Ahokas, & Leucht, 2001). This might be due to the fact that the treatment we provided has a clear rationale and procedures, and is short in duration. Avoiding delay by not adjusting the protocol and not preceding treatment with stabilizing techniques has the effect that subjects experience that treatment can rapidly produce symptom reduction.

Some patients, who had hallucinations for many years, reported no hallucinations after treatment, while others reported persistent hallucinations despite treatment. The results of cognitive behavioral therapy in people with psychosis shows that most of the time

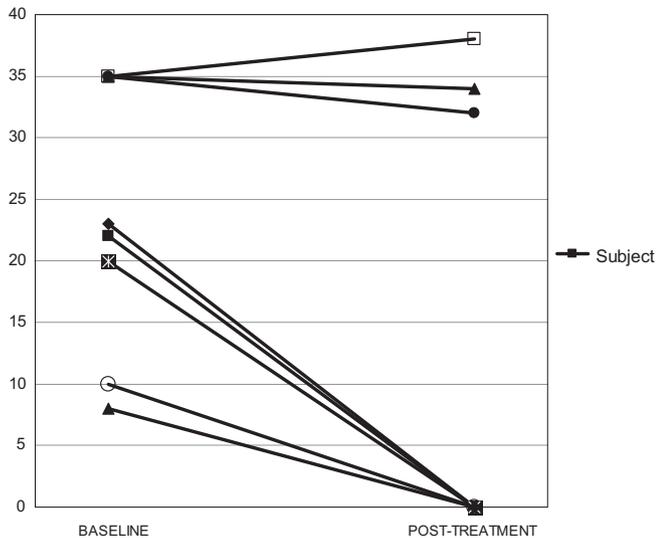


Fig. 1. Change in auditory verbal hallucinations scores on the auditory hallucination rating scale in completers experiencing auditory verbal hallucinations at baseline.

the voices continue after cognitive behavioral therapy, but that the patient experiences less distress, is less involved with the voices and has learned to be indifferent to the content of the voices. The results of this study are remarkable and suggest that auditory verbal hallucinations may have been associated with trauma or posttraumatic stress disorder in some subjects. This is in line with the fact that 70 percent of the voice hearers indicate that they started to hear voices after a traumatic or very emotional experience (Romme & Escher, 1989). It is also supported by many studies which show that traumatic experiences are a risk factor and may be causal in the etiology of psychosis (Arseneault et al., 2011; Elklit & Shevlin, 2010; Read, Bentall, & Fosse, 2009; Schreier et al., 2009). The management of psychosis should therefore include an assessment of trauma history and adequate treatment of trauma related symptoms with techniques such as prolonged exposure or eye movement desensitization and reprocessing. Future research will have to address these issues.

It is notable that not only posttraumatic stress disorder symptoms improved after eye movement desensitization and reprocessing, but also depression and anxiety diminished and when present at baseline also delusions and hallucinations improved. There were no effects on paranoid ideation and hopelessness directly after treatment. There was no general effect on psychotic symptoms, but therapy addressed specific parts of psychosis. Trauma and trauma related symptoms improved, while paranoia and associated hopelessness were persistent. This is a unique finding and is in need of replication.

4.2. Study strengths

This study shows that it is feasible to use the standard eight-phase eye movement desensitization and reprocessing procedure in patients with psychotic disorders and that this procedure is safe and effective in treating posttraumatic stress symptoms. Moreover it demonstrates that a small dose of eye movement desensitization and reprocessing already produces significant effects on a broad array of symptoms and that stabilizing interventions are not necessary. It shows that it should not be a rule of thumb to exclude patients with psychotic disorders from posttraumatic stress disorder treatment. This study is the first to show that treating posttraumatic stress disorder in patients with psychosis not only

reduces posttraumatic symptoms but also reduces auditory verbal hallucinations, delusions, depressive symptoms, and anxiety symptoms and improves self-esteem.

4.3. Study limitations

This pilot study is an open trial and has several limitations.

The most important limitation is the lack of a control group. The within-group effect is overrated compared to a between-group effect-size, because there is not controlled for placebo effects and other confounders.

A second limitation is the use of chart diagnosis to assess the lifetime psychotic disorder. Many patients had a chart diagnosis of psychotic disorder nos, which reflects typical diagnostic bias in the Dutch health care system. As the diagnosis is communicated to the patient by insurance companies, many psychiatrists are reluctant to set more stigmatised diagnoses such as schizophrenia. On the other hand, the mean duration of psychotic symptoms of more than thirteen years, suggests that in reality more subjects actually met criteria for schizophrenia while they were diagnosed with a psychotic disorder nos. It is more likely that our sample is more severely than less severely ill.

A third limitation is the fact that the therapists administered the clinician administered posttraumatic stress disorder scale, which might have led to a bias in interpretation of symptoms.

A fourth limitation is that there is no check for inter-rater reliability and routine treatment integrity checks were not performed. The therapists did fill in a form every therapy session. This form described what the content of that session had been, what targets they worked on and what the effect had been (i.e. changes in subjective units of distress and validity of positive cognitions).

A last limitation is that there is no follow-up assessment, making it impossible to determine whether treatment gain was maintained over time.

Randomized controlled trials with sufficient statistical power will need to be performed in order to confirm or refute our results. It will be valuable to include assessments of psychotic symptoms and of the relationship between psychotic and posttraumatic stress symptoms. Besides this, monitoring processes of change will give insight in what makes treatment work and test hypotheses why psychotic symptoms decrease after treatment of posttraumatic stress symptoms. Moreover, eye movement desensitization and reprocessing will have to be compared to other active treatments such as prolonged exposure.

Conflict of interest

None.

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