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Relationship between anxiety and the psychosis spectrum: A narrative review

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ABSTRACT: Anxiety symptoms and disorders are common findings throughout the psychosis spectrum, with this association affecting a multitude of aspects related to the psychotic illness. In addition, psychosis spectrum disorders may also contribute to worsen anxiety. In line with the continuum hypothesis, the comorbidity between these syndromes calls for a reframing of psychopathology. Also, anxiety may serve as a therapeutic target, with new treatment options being recently researched. The present narrative review analyzes the relationship between anxiety symptoms and disorders and the psychosis spectrum. A search was performed in six bibliographic databases for articles establishing either a direct or an indirect relationship between these entities; published from the 1st of January of 2019 to the 31st of December of 2024. The final assessment contemplated 46 studies out of 72 screened records. Here we show anxiety spectrum disorders and psychosis spectrum disorders are highly comorbid across all phases of illness. Anxiety does not affect the risk of psychosis conversion. Conflicting evidence has emerged on its influence over negative and positive symptoms. Some authors defend its beneficial role over cognitive performance. Anxiety mediates the association between the use of cannabis and psychotic experiences. Both facial expression recognition and social functioning are affected by this comorbidity. In addition, a worse outlook on the illness may affect its recurrence, ultimately leading to a reduced quality of life. Finally, a new transdiagnostic approach means patients in the psychosis spectrum should be timely assessed and treated for anxiety disorders, with new psychotherapeutic options showing promising results.

KEYWORDS: Anxiety; psychosis spectrum; psychosis comorbidity; psychosis prodrome; clinical high risk.

INTRODUCTION

Psychosis is a comprehensive term defining a cluster of symptoms related to a loss of touch with reality[1]. Throughout their lifetime, it is estimated that around 1 to 4% of the world's population will suffer from a psychotic disorder (PD), while a much larger portion – 13 to 23% - will sustain at least one psychotic symptom[2].

Anxiety symptoms and disorders are common findings throughout the psychosis spectrum. Indeed, this association may even predate the onset of psychosis itself, with close to 40% of the clinical high risk (CHR) population presenting with a comorbid anxiety disorder[3]. Current literature suggests this association is present on multiple dimensions of the illness, such as transition from prodromic states to full psycho-

sis[4,5], positive and negative symptoms[3,6,7,8,9], perception of illness and self[10,11,12,13], social functioning[6,11,14,15,16,17,18,19], quality of life (QoL)[11,14,16,20], cognitive performance[21,22], cannabis use[23,24,25], neuroanatomy[25,26], genetics[17,27,28] and facial expression recognition[19,29].

More recently, studies have found this to be a bi-directional relationship, meaning psychosis spectrum disorders may also worsen existing anxiety states or even constitute risk factors for future anxiety[22,30].

These findings are in line with the postulates of the continuum hypothesis, stating that psychotic syndromes more often than not originate from premorbid non-psychotic disorders (NPDs). The excessive comorbidity and the inter-relationship between syndromes have raised discussions on the reframing of psychopathology[17,31].

Considering the strong link between anxiety and psychosis, the former has been garnering increasing attention as a therapeutic target, mainly by its timely assessment and treatment[6,8,11,27,29,32]. Novel findings have also brought on new and interesting therapeutic avenues to explore[33,34,35].

The present narrative review aims to gather scientific evidence on the relationship between anxiety symptoms and disorders and the psychosis spectrum, from its premorbid and prodromic states to the diagnosis of a formal disorder, with special attention to the clinical implications of this comorbidity.

METHODS

A search was performed from the 1st of September of 2024 to the 31st of December of 2024 in six bibliographic databases. The inclusion criteria used for the selection process included: articles establishing either a direct or an indirect relationship between anxiety disorders and the psychosis spectrum disorders; articles published from the 1st of January of 2019 to the 31st of December of 2024. The major exclusion criterion was the following: articles either not establishing the above-mentioned relationship or studying the disorders individually (Fig. 1).

RESULTS

The final assessment contemplated 46 studies out of 72 screened records. The main reason for excluding reports was due to not being related to the topic. A review of these articles ensued.

DISCUSSION

COMORBIDITY ACROSS THE SPECTRUM

Current literature shows patients at risk are often more distressed by anxiety than by their attenuated positive symptoms (APS)[36].

A meta-analysis concluded that 75% of CHR subjects (i.e. individuals in the prodromal stages of psychosis)

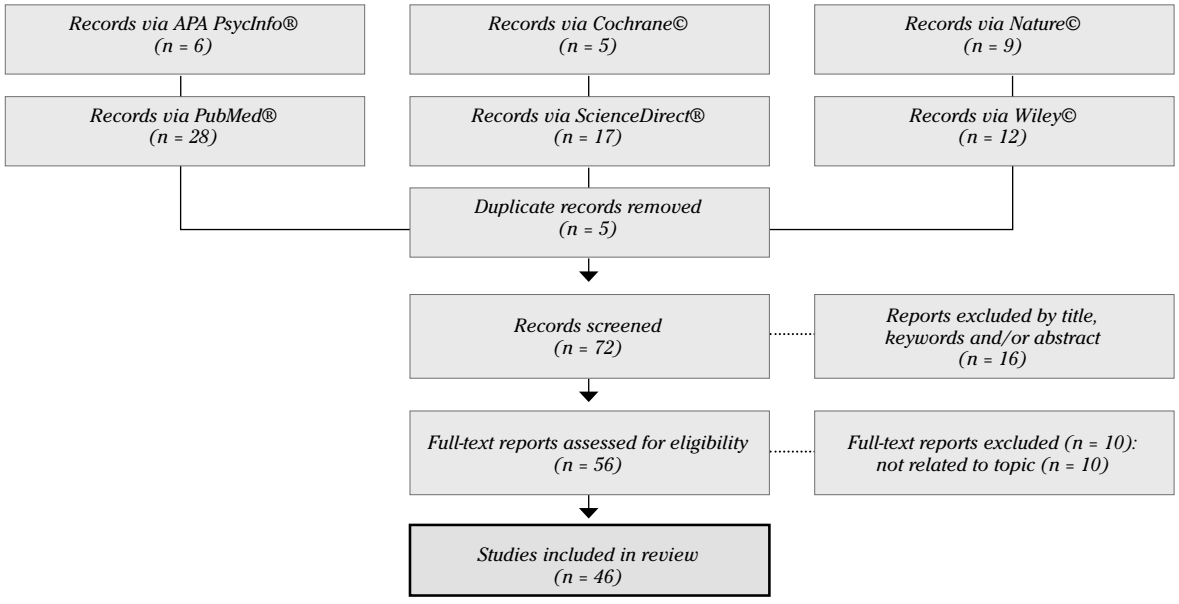

FIGURE 1. Study selection flowchart

present with a baseline comorbid mental disorder[5]. Compared to a community sample, this population showed a threefold greater risk of having a social anxiety disorder (SAD) diagnosis, with 42% of the group meeting its criteria[6]. Another study on CHR individuals found three distinct groups of participants: 62,5% of the sample showed no anxiety; 18% showed only anxiety; 19,5% showed both anxiety and a comorbid disorder[3] - a significant increase upon the rate of 15% found by a previous review[36].

Also, around 25% of the individuals with remitted first episode psychosis (FEP) referred clinically significant social anxiety (SA) symptoms after an 18-month follow-up[16]. Recently, the estimated pooled prevalence of comorbid SAD amongst individuals with a PD was 21%[11]. Interestingly, this rate was much greater for the outpatient population (25%), compared to the inpatient population (9%), likely due to the former's higher insight for SA, thus making its assessment more precise[37,38]. On the other hand, inpatients may be experiencing more psychotic symptoms, making the evaluation of anxiety symptoms a difficult challenge. In a different inpatient population, the authors found 46% of participants had a NPD upon admission, while 17% of participants presented with an anxiety or stress disorder[39]. Lastly, in a systematic review, the comorbid anxiety rate was 29% amongst a sample of FEP patients[31].

The difference in prevalence observed between the at-risk states and PD/FEP is congruent with a recent systematic review[5], probably due to a tendency for prodromic states to be more affected by mood and affective dysregulation than psychotic states.

PSYCHOSIS CONVERSION

The psychosis spectrum disorders prodrome may be defined as an early state of psychosis presenting as a deviation from an individual´s usual patterns and behaviors[40]. The most powerful predictors for this transition are the severity of positive symptoms, poorer social functioning and genetic risk for schizophrenia[41].

Recent meta-analytic studies concluded that comorbid NPDs, especially anxiety, were not significantly associated with greater risk of transition[4,5]. As a matter of fact, generalized anxiety disorder has been linked to a lower transition risk[42].

NEGATIVE AND POSITIVE SYMPTOMS

The acute setting of psychotic disorders is characterized by alertness, anxiety and sensitivity to their surroundings, while the chronic setting concurs with avoidance[43]. Also, comorbid SA samples were associated with more somatic and grandiose delusions[44]. However, new evidence on socially anxious groups has showcased no difference in terms of positive and negative symptoms[45]. On the opposite direction, suspiciousness appears to significantly predict SA[46] and both negative and positive symptoms were associated with SA[47]. Others concluded a lack of correlation between these entities[48,49].

A network analysis showed no relationship between subclinical psychotic symptoms and non-psychotic symptoms[7]. Despite SA symptoms being related to negative symptoms in CHR individuals, one study found no link with positive symptoms[6]. The authors proposed that negative symptoms could be due to SA and not a result of a prodromic state. These findings challenge the distinction between primary negative symptoms from those caused by SA. In another sample of CHR individuals at 24-months of follow-up, the anxious groups of participants noted greater negative symptoms and APS compared to their non-anxious peers[3].

According to previous studies, anxiety is related to the risk of developing auditory verbal hallucinations[50], however, a recent analysis concluded that anxiety was not associated with their negative content[8].

A cross-sectional study in an early psychosis sample concluded that SA was linked to both negative symptoms severity and ideas of reference[9].

COGNITIVE PERFORMANCE

Cognitive impairments are considered key elements of the psychosis spectrum[51,52], which manifest in the population at high risk for psychosis, becoming more severe upon the onset of a FEP[53,54]. Previous studies have inconsistently shown their association with anxiety[21], either by linking it to reduced cognitive performance[19,55], finding no correlation[55,56] or even arguing its beneficial role[56,57]. This advantageous effect may be better explained by either the optimal arousal theory [58] or the adaptation theory[59].

According to the adaptation theory, higher levels of anxiety serve as a marker of unimpaired cognitive performance[59]. Indeed, in a sample of FEP patients, their anxiety symptoms correlated positively with their cognitive functioning at baseline. In line with previous studies[60], intact cognition is linked to the emergence of affective symptoms, further reinforcing the adaptation theory[21].

On the other hand, the optimal arousal theory refers to the need for a certain level of arousal in order to improve learning capabilities[58]. As such, in individuals

with elevated levels of psychotic-like experiences (PLEs), anxiety symptoms seemed to facilitate cognitive performance. Conversely, in the group showing low levels of PLEs, high levels of anxiety symptoms affected their functioning. These findings serve as evidence for the optimal arousal theory, however, arousal and anxiety are two distinct concepts[22].

CANNABIS USE

Daily usage of cannabis increases the risk of a primary PD by up to fivefold[61]. Early use onset, weekly use and higher cumulative exposure also contributed to increased anxiety symptoms[23].

Two recent studies showcased the mediating role of anxiety on the association between cannabis use and psychotic experiences (PEs)/ PLEs[23,25], in line with a previous study[62]. On the opposite direction, substance-induced psychosis was associated with an up to sevenfold increase in the risk of developing anxiety[24].

FACIAL EXPRESSION RECOGNITION

In patients with schizophrenia, overall facial emotion recognition is severely impacted[63]. Also, patients with anxiety disorders report more negative interpretations of ambiguous expressions[64]. In addition, an early psychosis sample did not find significant differences in facial emotion recognition between the control group and the comorbid SA group[65].

A study on a small population with a diagnosis of schizophrenia concluded the patients were able to recognize positive and negative emotions, regardless of comorbid SAD. However, the SAD group made more mistakes while recognizing neutral expressions, often misinterpreting them negatively. These individuals may wrongly analyze situations as being judged or disliked, leading to social avoidance and poorer social functioning[19], in accordance with previous studies[66]. In parallel, a CHR population with SA demonstrated a bias toward angry expressions recognition, along with poorer neutral face recognition[29].

SOCIAL FUNCTIONING

Social functioning impairments are well-documented characteristics of psychosis spectrum disorders, affecting the prognosis of ultra high risk (UHR) states (i.e. individuals with high probability of developing a psychotic episode) and patients with PDs[67,68]. Anxiety is also known to exacerbate social dysfunction in these populations[69,70].

In a general population sample, the positive correlation between PLEs and social functioning was mediated by anxiety. Social functioning was affected by anxiety in the domains of leisure activities and relationships within family, while role functioning impairments were better explained by the PLEs[15]. In accordance, a cohort study evidenced that individuals with PEs and comorbid NPDs were differentiated by increased social dysfunction[17], whereas the severity of SA symptoms was related to reduced social functioning among CHR individuals[6]. In addition, patients with remitted FEP or schizophrenia accompanied by persistent SA presented with worse social functioning[11,16,19].

On the contrary, others stated higher anxiety predicted better functional outcomes in FEP patients, as it may improve illness insight, treatment-seeking behaviors and better compliance[18]. Another study failed to find a significant association between NPDs comorbidity in at risk mental state individuals and poorer psychosocial functioning[14].

PERCEPTION OF ILLNESS AND SELF

Previous literature proposed that the perceived awfulness of psychosis stands as a trigger for increased anxiety of illness recurrence[71], and actual relapse[72].

In a sample of participants in recovery from psychosis, fear of relapse and mental health anxiety (i.e. excessive and persistent preoccupation with developing a mental health illness) were elevated[10] and these constructs predict future psychosis recurrence[73]. However, fear of relapse did not predict variations in psychological distress, contrary to what was demonstrated by previous studies[74]. Mental health anxiety was the best predictor of fear of relapse, in itself a better predictor of actual psychosis relapse than early-sign surveillance.

The existing literature also suggests patients may wrongly perceive unusual experiences, which would increase feelings of anxiety[75]. In fact, individuals in recovery from psychosis negatively misinterpreted both PLEs and anxiety-related experiences, further increasing negative emotions, arousal, avoidance, delayed help-seeking and hypervigilance, worsening anomalous experiences and leading to relapse, justifying catastrophizing to a greater extent[13]. Similar results were shown in at-risk mental state patients or in those with a schizophrenia diagnosis[12], in parallel with the emotional dysregulation hypothesis[76].

A meta-analysis also concluded that individuals with a PD and comorbid SA were significantly more affected by self-statements[11].

QUALITY OF LIFE

Preceding studies on the QoL of individuals with UHR found lower scores were linked to increased anxiety[77]. Actually, the QoL of patients with a first-episode schizophrenia diagnosis was influenced by unemployment and anxiety/depressive symptoms[78].

More recently, individuals with at risk mental state suffering from anxiety demonstrated diminished QoL in both mental and physical domains[14]. Another study concluded UHR and recent-onset PD patients with more pronounced anxiety at baseline reported worse QoL at follow-up in both interpersonal relationships and environment[20]. Furthermore, in a subgroup of individuals with remitted FEP, persistent SA was associated with decreased QoL even after 18 months of treatment[16].

NEUROANATOMY

Generally, grey matter loss is linked to psychosis conversion in CHR samples[79]. Also, anxiety comorbidity contributes to grey matter loss in CHR patients[80].

Recently, a study screened CHR participants and reported that the baseline right hemisphere cortical thickness mediated the relationship between discriminatory experiences and anxiety symptoms, but not APS. The same mediation was observed for the middle temporal gyrus[26]. Secondly, authors have hypothesized hippocampal aberrations may originate delusions in those suffering from affective dysregulation[25].

GENETICS

Previous studies have showcased that isolated APS is at most weakly associated with schizophrenia polygenic risk score (PRS-SZ)[81]. However, comorbid NPDs may strengthen this correlation[82].

Recently, two cohort studies on the relationship between PRS-SZ and psychosis showed significant dependence on the presence of comorbid affective dysregulation[28]. Other authors demonstrated that, compared to participants with PEs only, those with comorbid NPDs were differentiated by an increased PRS-SZ[17]. Finally, within a subgroup of CHR patients maintaining high levels of SA at follow-up, it was possible to predict higher levels of PRS-SZ[27]. Altogether, affective dysregulation is both a predictor of PRS-SZ and a mediator of its relationship with psychosis.

ANXIETY MEDIATION ON PSYCHOSIS

The affective pathway to psychosis is currently the leading hypothesis on the mediating role of mood-related symptoms on the psychosis spectrum disorders[83]. Anxiety might lead to anticipation of threat and serve as a precursor to psychotic symptoms[32].

In a sample of early psychosis patients, anxiety and depressive symptoms significantly mediated the association between abuse and positive symptoms during the first three years of treatment[32].

A longitudinal study detected three different paths of anxiety across childhood and adolescence[84], in accordance with recent literature[85]: 70% of the sample showcased persistent low levels of anxiety; 20% demonstrated persistent moderate levels of anxiety; and, more importantly, 5% exhibited persistent high levels of anxiety. The authors concluded persistent high levels of anxiety across childhood and adolescence could be considered a good indicator of future PDs. Further, inflammatory mechanisms, measured by C-reactive protein levels at ages 9 and 15, mediated this association. The authors propose the high levels of anxiety may increase the levels of stress hormones, which would lead to a chronic low-grade inflammatory state and the subsequent downstream neurological effects would, in turn, lead to psychosis. Contrary to recent evidence[86], no mediating role was found for interleukin 6.

In a recent cross-sectional study, cumulative humiliation and state anxiety (i.e. the transient experience of anxiety) jointly predicted cognitive-perceptual disturbance, disorganization, fear of humiliation and interpersonal difficulty[87]. The authors added that the hypervigilance of state anxiety may increase the detection of ambiguous experiences, therefore increasing hallucinations[88] and leading to APS[87].

In a cohort of children with a 3-year follow-up, childhood anxiety symptoms predicted PEs in adolescence. Actually, general psychopathology at baseline also predicted PEs after three years, however, this relationship was much weaker than the one found for anxiety symptoms. Contrary to previous studies[30], PEs in childhood did not associate with the development of anxiety symptoms during adolescence[89].

PSYCHOSIS MEDIATION ON ANXIETY

Current literature states PLEs are often associated with NPDs[22], shifting the focus towards understanding their role as an antecedent to future psychiatric symptoms[90].

A cohort study concluded that an elevated incidence of PLEs during adolescence predicted anxiety symptoms[30]. Consistent with previous research[91], the

authors established that PLEs should be considered as both a marker and a precursor of future anxiety symptoms. Another study conducted in the general population found that an elevated incidence of PLEs was predictive of greater anxiety symptoms[22].

DIAGNOSTIC CONTINUUM

Taking into consideration the relationship between anxiety and the psychosis spectrum, there is a case to be made on the bidirectional nature of this relationship, creating solid groundwork for a diagnostic continuum hypothesis. The existing literature shows a great proportion of the at-risk population end up developing NPDs[92], as psychosis only represents one end to this diagnostic continuum[93].

Also, most psychotic states are caused by prior mood states, not by prior at-risk mental states[82]. A cohort study on a sample of FEP individuals concluded that psychotic syndromes tend to originate from preceding non-psychotic states. The authors observed a clear sequence of initial nonpsychotic symptoms, followed by subthreshold psychotic symptoms and later FEP[94].

However, according to previous DSM editions, NPDs do not take part in the CHR/APS diagnosis, creating a curious paradox[31]. Despite the fact the CHR/APS state should only include subthreshold psychotic symptoms not better explained by another disorder, the majority of individuals meeting the criteria for a prodromic phase present with comorbidities. These should be regarded as essential pieces in the inherently transdiagnostic and heterotypic CHR/APS construct. In fact, both anxiety and APS clusters may characterize one single syndrome, instead of pseudocomorbid conditions[17].

Previous approaches are also being increasingly criticized for providing little relevance to the biopsychosocial context surrounding each individual. New strategies are enabling retrospectively diagnosed UHR states to be prospectively approached[95].

CLINICAL IMPLICATIONS

The initial management of a psychosis syndrome is based on two major therapeutic avenues – antipsychotic therapy and psychosocial interventions, including cognitive remediation, social skills training, cognitive-behavioral therapy (CBT) and family-based interventions[2].

However, a main pillar of the illness management lays on the assessment of its comorbidities. In fact, a meta-analytic study firmly recommends the screening of SA through the 3-item Mini-Social Phobia Inventory or the following two questions: Do you find yourself avoiding social situations or activities? If so, is it because you are embarrassed or fearful of negative evaluation in such social situations?[11] Targeting the reduction of anxiety may potentially reduce critical, threatening and self-harm ideas in affective psychosis[8]. At sub-threshold levels of psychosis, tackling anxiety symptoms would be beneficial for social functioning[6], QoL[20] and positive symptoms[32].

Interventions targeting the perception of illness may reduce relapse rates, however, self-monitoring may be harmful, especially when hypervigilant states lead to catastrophizing of symptoms. CBT may prove efficient in dealing with catastrophizing, especially when based on psychoeducation about the illness cognitive model, alerting for its problematic hypervigilance, while also fighting self-stigma and providing the patients with better coping skills[96].

Mental health anxiety may also be covered by the existing health anxiety CBT[10]. Others suggest interventions targeting the most distress-inducing beliefs[12], such as psychoeducation, sharing of stories of recovery, behavioral experiments and verbal reattribution techniques.

CBT for anxiety is less effective in psychosis when compared to non-psychotic controls and bears a small effect on psychotic symptoms[97]. Others concluded patients with comorbid FEP and SA demonstrated significant improvements on SA when using both CBT for SA and cognitive remediation (i.e. a form of therapy aimed at reducing cognitive deficits). The effect on negative symptoms, functioning and self-stigmatization was more relevant on the CBT group[98].

Other forms of psychotherapy, such as family focused therapy, would reduce anxiety and improve family connection in CHR populations[99]. In addition, a brief 3-minute mindfulness exercise significantly reduced state anxiety for early psychosis patients[34]. Also, an early psychosis population showed a significant reduction in state anxiety after a single session of yoga[33].

One common issue of the management of psychosis is treatment attrition. A study on cognitive remediation showed high attrition associated to a group intervention, meaning that only 44% of participants concluded all the proposed sessions, compared to 59% in the CBT sample[100]. Another study found that individuals with increased SA showed better compliance. However, anxiety induced by the group setting was the chief reason for quitting the program[101]. Some patients would benefit from individual interventions. To that avail, an online intervention consisting of therapeutic comics and social

networking features reported an increased feeling of control over SA symptoms in FEP patients[35].

Finally, regarding the use of cannabis, reducing anxiety symptoms in cannabis users may prevent or delay the development of PLEs, making anxiety symptoms an important target for reducing psychosis risk[23]. Also, a more comprehensive debate on the legalization of cannabis is much needed[24].

CONCLUSION

In conclusion, anxiety and psychosis spectrum disorders are highly comorbid across all phases of illness, affecting a multitude of areas. While the risk of psychosis conversion from its prodromic stages is not affected by anxiety, conflicting evidence has emerged on its influence over negative and positive symptoms. In addition, recent studies have reinforced both the adaptation theory and the optimal arousal theory, providing anxiety with a beneficial role in PDs. Anxiety also mediates the association between the use of cannabis and PEs, while substance-induced psychosis predicts future anxiety. The functionality of an individual is also severely impaired by this comorbidity, affecting facial expression recognition and social functioning. Regarding their perception of illness, patients fall in a cycle of hypervigilance, followed by anxiety and illness recurrence. All these factors ultimately contribute to a very diminished QoL. A biological background may be one of the culprits, as both the PRS-SZ and the grey matter volume are negatively affected by this comorbidity. As such, a diagnostic continuum hypothesis is garnering increasing attention. Patients in the psychosis spectrum should be timely assessed and treated for anxiety disorders. While traditional forms of psychotherapy should be improved, new therapeutic avenues also warrant further investigation.

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