Puerperal Psychosis

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9.1 Introduction

The perinatal period is a time when women are at the highest risk of mental health difficulties [1], with suicide remaining a leading cause of maternal death within a year of childbirth [2, 3]. The most severe perinatal mental health disorder is postpartum psychosis. Postpartum psychosis is rare, affecting around 1–2 in 1000 deliveries; however, despite this very low incidence, the postpartum represents a period of incredibly high risk of developing such severe psychopathology—23 times higher than any other period in women's lives [4].

Characterised by a rich variation in symptomology, this rare disorder is not currently included in current diagnostic systems such as the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) or International Classification of Diseases (ICD) [2, 4, 5]. It exists only as an umbrella term, encompassing a number of severe psychiatric presentations affecting women after childbirth [2, 4].

Women with postpartum psychosis will frequently present with severe affective episodes; for some this may be mania or psychotic depression, whilst others develop mixed episodes of high and low mood [2]. Psychotic features—such as paranoia,

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hallucinations and delusions—are common, estimated to be present in over 70% of cases [6, 7]. Some also report neurological symptoms, including decreased consciousness and motor difficulties [4, 5]. Often, symptom onset will occur abruptly and dramatically, typically within 4–6 weeks after childbirth [2, 8].

For the majority of these women, this severe postpartum episode will be their first experience of any acute psychiatric history, hereafter described as first-onset postpartum psychosis [5]. Nevertheless, one of the strongest predictors of postpartum psychosis onset is a previous diagnosis of bipolar disorder [9, 10]. Postpartum mood episodes across the mood disorder spectrum are highly prevalent, but the relationship between postpartum psychosis and bipolar disorder has been evidenced most consistently [11–13]. Indeed, early research considered postpartum psychosis to be a specific presentation of bipolar disorder, triggered by parturition [3, 14, 15]. It is likely that women with bipolar disorder are particularly susceptible to relapse during this period compared to other psychiatric disorders such as schizophrenia [16].

This chapter will introduce the specific course and risk factors involved in postpartum psychosis. Based on clinical and academic research across the world, it will establish this severe disorder as a psychiatric emergency. Finally, it will highlight the necessary factors that should be considered when dealing with the treatment and management of these cases in order to minimise harm to the mother, child and their outer network.

9.2 Epidemiology

9.2.1 Burden of Disease

Estimating the global prevalence of a rare disorder such as postpartum psychosis poses several challenges. Incidence rates—which vary between 1 and 2 per 1000 childbirths—are typically based on inpatient admissions in Western societies [1, 2, 4]. This results in an under-representation which limits understanding of global and, in particular, non-Western outpatient cases. A recent meta-analysis conducted by the World Health Organization found incidence rates in lower-income countries to be relatively comparable to Western estimates, despite Nigeria reporting the highest incidence at 2.6 in 1000 childbirths [1]. These findings must be considered alongside the caveat that postpartum psychosis is a highly heterogenous term, due to inadequate diagnostic systems and poorly understood cultural differences. Contact with services may present another opportunity to estimate prevalence, being that hospital admissions are frequently necessary following a postpartum episode [2]. One population-based study estimated the risk of being admitted during the first 30 days postpartum with a first-onset postpartum bipolar disorder diagnosis to be 23.33% [17]. Although a valuable starting point, this estimate does not take into consideration the considerable likelihood of being misdiagnosed [18].

Despite the low prevalence, women are far more likely to develop a first-onset affective psychosis during the postpartum period than any other time point in their

lives [4]. For women with bipolar type I disorder, incidence sharply increases to 1 in 5 [19]. Subsequent pregnancies for women with a history of postpartum psychosis confer greater risk still, at one in two childbirths [2, 20]. The true incidence is likely to be much higher given the need for additional data.

It is unsurprising that the financial burden of such a severe mental illness is steep. In a 2014 report by the Centre for Mental Health in the UK, postpartum psychosis was estimated to cost the economy £53,000—roughly $68,800 in US dollar—*per case* [21]. These costs—based on prolonged service use and losses to productivity and quality-adjusted life years—are likely to be much higher due to insufficient data on adverse effects on the child. The economic costs relating to the mother alone are double that of perinatal depression or anxiety, with costs to the public sector being at least 20 times higher for postpartum psychosis. These findings demonstrate the need to address what can only be described as a psychiatric emergency.

9.3 Aetiology

The relationship between postpartum psychosis and the childbirth trigger is indisputable when considering the highly specific risk profile. A meta-analysis found that over 40% of women with first-onset postpartum psychosis experienced affective psychosis isolated to the postpartum period [22], with others reporting that this figure could be up to 50% [4]. Based on population studies, where increased risk for postpartum depression persisted up to 5 months postpartum, the risk for bipolar-type disorders remained increased for only 2 months [17]. Postpartum disorders classified as schizophrenia-type were even more specific still, with increased risk persisting for just 30 days following childbirth. Pregnancy seemed to be protective against accessing services, and overall, the window of highest risk fell between 10 and 19 days postpartum for first-onset and readmission risk [17, 23]. Childbirth may be the key instigator in the onset of postpartum psychosis, yet it is likely that a complex interaction between a range of biological and psychological risk factors is also at play.

9.3.1 Bipolar Disorder

In the DSM, most postpartum psychosis cases fall under the mood disorder nosology, making it almost impossible to distinguish from bipolar disorder with a postpartum recurrence. This is understandable in view of the significant overlap between symptoms, risk factors and prognosis. For women with a first-time onset of psychiatric disorder within 2 weeks of delivery, the likelihood of converting to a bipolar disorder diagnosis has been reported to be over three times higher than for women who present outside of the perinatal period [18]. Similarly, women with bipolar disorder are at significant risk of a postpartum psychosis diagnosis [23], with a history of bipolar disorder—familial or individual—remaining one of the strongest risk factors for postpartum psychosis [9, 14, 15].

9.3.2 Risk Factors

9.3.2.1 Obstetrics

Based on the specificity of the puerperal trigger, it is reasonable to presume that obstetric factors play a role, the most cited of which is primiparity. In one study, 35% of women with bipolar type I disorder reported psychosis or mania following their first child. This dropped to 20.5% for a second pregnancy and reached just over 14% for subsequent pregnancies [19]. This was the case despite excluding women with only one pregnancy from the analyses as a way of controlling for the possibility that women chose not to have further pregnancies following their first. These findings therefore suggest possible differences between the first and subsequent pregnancies that impress greater risk for affective psychosis.

Postpartum psychosis is not the only condition that is more common following a first pregnancy. Pre-eclampsia—which has been associated with primiparity—has been shown to increase risk of psychiatric postpartum episodes [2, 24]. Given the similar risk profiles of postpartum psychosis and pre-eclampsia, it seems possible that they share common disease mechanisms. Notably, pre-eclampsia begins during pregnancy, signalling a marked difference to postpartum psychosis. It is suspected that pre-eclampsia induces a hyperinflammatory state which could promote changes in brain function due to disruptions to the blood-brain barrier [24]. Similar comorbidities may also aid understanding of the disease mechanisms underpinning postpartum psychosis.

Research has also shown that affective psychosis is more likely following a full-term live or stillbirth delivery compared to termination or miscarriage [25]. This provides further evidence for the significance of the childbirth trigger. Other obstetric factors, including delivery complications, Caesarean section and having a female baby, have been previously cited as increasing risk of postpartum psychosis; however, a retrospective study found no robust evidence for these [26].

9.3.2.2 Reproductive Hormones

Hormones offer an obvious candidate for the aetiology of postpartum psychosis due to the array of hormonal changes that occur during the postpartum period. Research in the past has implicated the typical drop in gonadal steroids, such as oestrogen, following childbirth with psychosis onset [27]. Others cite improvements in psychotic symptoms following oestradiol treatment [28]. Yet many of these studies remain largely outdated and irreplicable [4]. This lack of reliable evidence for the role of hormones in postpartum psychosis is also true for progesterone and oxytocin.

Nonetheless, it is important to remember that these hormones do not occur in isolation but interact in a complex system of neurotransmitters and other hormones. Consequently, the differential sensitivity to hormone shifts during the postpartum period—which is exhibited by women with a history of postpartum episodes—is likely mediated by other factors linked to this system. Evidence for this can be inferred from a study of women with postpartum depression, where perinatal hormone conditions were pharmacologically mimicked [29]. The abrupt withdrawal of increased oestradiol and progesterone (a hallmark of parturition) marked a peak in

depressive symptoms in women with a history of postpartum depression. These findings illustrate the possibility that gonadal steroids may play a role in the aetiology of postpartum psychiatric episodes, despite the overall levels of these hormones remaining largely the same as for women without a similar history [7, 29].

One potential mechanism through which these hormones may contribute to postpartum psychosis is the dopaminergic system. Dopamine, along with other neurotransmitters such as noradrenaline and serotonin, is often implicated in the aetiology of nonpuerperal affective or psychotic disorders [7], suggesting they may have a potential role in postpartum psychosis. Further evidence for the role of dopamine can be found in case studies of women outside of the postpartum period where administration of dopamine agonists induced mania or psychosis; however, this phenomenon seems to be rare [30, 31].

9.3.2.3 Immunological Disease

Psychosis has long been linked to immune system function—from infectious agents leading to psychosis to several autoimmune disorders such as multiple sclerosis including psychosis as a symptom [32]. A comprehensive review of this relationship is beyond the scope of this chapter; nevertheless, several eminent findings are worth mentioning.

Microglia—the brain-based counterpart of macrophages—are heavily involved in immune response as well as neurodevelopment and synaptic functioning. Due to the difficulty in studying microglia in vivo, most research has opted to indirectly measure their functioning through their production of inflammatory cytokines which promote immune response. In a meta-analysis of 30 studies, inflammatory cytokines were aberrant in individuals with bipolar disorder compared to healthy controls [33]. Further evidence demonstrates that levels of inflammatory cytokines are positively associated with the acute phases of bipolar disorder—in particular mania—yet testimony of their relationship with depressive episodes remains inconsistent [34, 35]. Notably, these same markers were not associated with any states or symptoms of non-affective psychosis (schizophrenia), suggestive of an immune mechanism aetiology specific to affective psychosis [34].

Natural killer cells may also be implicated due to their role in cytokine production and cytotoxic functions. A recent study found that natural killer cell activation was much higher in individuals with first-onset psychosis who were subsequently diagnosed with bipolar disorder [36]. The authors cited inappropriate NKG2C expression (a cell-surface receptor) as the possible mechanism, as this has been linked to abnormal cytokine production, toxicity and severity of psychotic or manic symptoms. Circulating monocytes (part of the same developmental lineage as microglia) and T cells (which interact with monocytes and microglia) are white blood cells that also exhibit abnormal levels in individuals with bipolar disorder [32, 37]. Finally, not only do bipolar disorder patients show high prevalence for autoantibodies—a marker of an autoimmune response—but so do their first-degree relatives, implying a potential familial immune abnormality [32].

Given the close relationship between bipolar disorder and postpartum psychosis, concurrent findings are to be expected for the latter group. Research into immune

dysfunction and postpartum psychosis is much more sparse, with most evidence originating from centres in the Netherlands [24, 37, 38] and India [39]. There is some variation in these studies, with one reporting robust increases in cytokines and monocytes in women with postpartum psychosis [37] and another a decrease [39]. Similarly, one reports an overall decrease in T cell activation in women with postpartum psychosis compared to controls [37], whilst another reports a specific decrease in CD4 helper and CD8 cytotoxic T cells alongside an increase in natural killer cells and regulatory T cells [39]. Still, the consistent finding of immune system abnormality across two very different cultural contexts advocates a strong link between postpartum psychosis and immune response.

The increase in prevalence of both affective psychosis and autoimmune disease during the postpartum period further supports a common underlying immunological mechanism. Postpartum psychosis has been reported to co-occur with or exacerbate symptoms of autoimmune thyroiditis, multiple sclerosis and rheumatoid arthritis [4, 32, 40], indicative of an immune dysfunction mechanism underpinning these conditions. This is further supported by research that has shown central nervous system antibodies in a subset of women with postpartum psychosis, suggesting comorbid autoimmune encephalitis [38]. These immune dysregulations—and the vast number of symptoms related to them—likely reflect an interaction between a number of genetic, epigenetic and environmental traits.

9.3.2.4 Genetic Factors

There exists a strong body of evidence for a genetic component in postpartum psychosis. Mood disorders including bipolar disorder have consistently been found to aggregate in families [14, 15]. A family history of postpartum psychosis has been reported to increase risk of relapse by over twofold in women with bipolar disorder [15]. Further exploration of this familial component in a seminal linkage analysis led to the discovery of one genome-wide significant loci on chromosome 16p13 and, to a lesser extent, 8q24 [41]. Specific genes associated with increased postpartum psychosis risk, however, remain elusive.

Previous research has focused on candidate genes relating to serotoninergic, hormonal and inflammatory pathways. Serotonin-related genes offer interesting candidates as they are modulated by oestrogen. Whilst no evidence has been found linking postpartum psychosis with the 5HT2A serotonin receptor [42, 43], there has been some evidence for an association with two loci within the serotonin reuptake transporter (SERT/5-HTT) gene [42]. Exploration into hormonal pathways has found no relationship between postpartum psychosis and the oestrogen receptor alpha (ESR1) gene [44, 45]. Nonetheless, one study reported an association with the methyltransferase-like 13 (METTL13) gene—which regulates oestrogen receptor-related gene transcription [45]. Genes encoding inflammatory cytokines such as tumour necrosis factor alpha (TNFα) are also found to be regulated by oestrogen yet do not seem to be associated with postpartum psychosis risk [46]. In spite of this, it is likely that immunological genes may contribute a peripheral role. Supportive of this is the upregulation of immune-related gene expression in the monocytes of women with postpartum psychosis [37] which coincides with the downregulation of microRNA that moderates these immune-related genes [47].

More recently, researchers in the UK have used a case-control approach to compare genetic risk for different mental health conditions in women with first-onset postpartum psychosis, women with bipolar disorder (with and without history of postpartum relapse) and control women [48]. When looking at schizophrenia and bipolar disorder genetic risk, women with first-onset postpartum psychosis and women with bipolar disorder had very similar levels of risk - higher than that of control women. Looking at genetic risk for major depression however, woment with first-onset postpartum psychosis and women with bipolar disorder differed. Women with first-onset postpartum psychosis had risk levels similar to that of controls whilst women with bipolar disorder had higher levels of genetic risk for major depression. This research is the first to suggest that postpartum psychosis may be partially genetically distinct from bipolar disorder, supporting its recognition as its own disease entity within the bipolar disorder spectrum.

These collective findings, though promising, do not conclusively elucidate the genetic risk associated with postpartum psychosis. Replication is required, along with unbiased, genome-wide association approaches with large sample sizes to reach sufficient statistical power. Given the rarity of the disorder and the robust sample sizes required for such complex disorders—tens of thousands at least—these studies will likely require large-scale, international consortia similar to those established for bipolar disorder and schizophrenia [49, 50].

9.3.2.5 Medication Changes

Women with an established bipolar disorder are faced with the difficult decision of whether to continue their medication during pregnancy. Previous research has shown that discontinuing lithium treatment during pregnancy did not increase risk of recurrence compared to women who were not pregnant [51]. Following childbirth however, risk of relapse rose drastically for women having discontinued treatment. More recent research also reported a twofold increase in risk of recurrence during pregnancy after mood stabiliser discontinuation [52]. These findings demonstrate the need for improved treatment planning that considers risk to both mother and baby.

9.3.2.6 Sleep Deprivation

For some individuals with bipolar disorder, sleep deprivation has been linked to increased risk of manic episodes, particularly for women [53, 54]. This relationship has been shown to exacerbate risk during the postpartum period, a time of significant sleep loss. Women with bipolar disorder who described sleep loss as a trigger for manic episodes were twice as likely to experience postpartum psychosis than those whose episodes were not triggered by sleep loss [53].

The evidence relating sleep loss and postpartum psychosis remains sparse but compelling. Firstly, disruptions to the circadian rhythm are commonly reported in postpartum psychosis, often preceding the postpartum episode [4]. Second, indirect evidence on the importance of sleep comes from the observation that targeting sleep loss alone can lead to remission in about 6% of cases [55]. Finally, neurotransmitters—already discussed as a mechanism in the aetiology of postpartum psychosis—are also likely to play a role in sleep [7]. Sleep disturbance therefore offers an

important and easily monitored risk factor when considering disorder prevention for women at high risk.

9.3.2.7 No Association with Personality or Adverse Childhood Events

Bipolar disorder has been associated with a number of personality traits, including impulsivity, neuroticism and low extraversion [56, 57]. In light of this, it is easy to assume that postpartum psychosis would follow a similar pattern. This is not the case. Neither personality traits, cognitive styles nor affective temperaments were found to differentiate women with postpartum psychosis and women with bipolar disorder [56]. Similarly, rates of adverse childhood events, including abuse, deaths, parental separation and serious illness, seem to be no different [58], demonstrating that there is little evidence of a link between psychosocial factors and postpartum psychosis.

First-onset postpartum psychosis

The number of women who develop postpartum psychosis yet have no family or individual history that would predispose them to it is estimated to be 50% or higher [2]. A subset of these women go on to be diagnosed with bipolar disorder and will continue to experience episodes outside of the postpartum period [13]. For these women, it is possible that childbirth triggers a bipolar diathesis that was undetectable up to that point [13].

Generally those with affective psychosis confined to the postpartum period will have a more favourable disease course than women with postpartum psychosis within the context of an existing bipolar disorder [22, 59]. One study found that women with first-onset postpartum psychosis remained stable during pregnancy and were less likely to relapse during the postpartum period—provided they began treatment with lithium prophylaxis following childbirth [59]. Furthermore, researchers have shown that women with first-onset postpartum psychosis do not show significant enrichment of obstetric risk factors [60]. These women also exhibited delayed symptom onset and mood incongruent psychotic symptoms, which stands in contrast to bipolar disorder. These differences have led researchers to consider first-onset postpartum psychosis to be its own distinct category, making management yet more complex [22, 55]

9.4 Management

9.4.1 Diagnosis

Despite difficulties with diagnosis due to poor stratification in diagnostic manuals, the temporal link to childbirth and severity of symptoms facilitates identification. Clinical presentation is rapid, with over half of symptom onsets taking place by postpartum day three [2]. Researchers have identified three overarching symptom profiles—manic, depressive and atypical—the latter of which is characterised by disorientation and delirium-like symptoms [61]. The majority of women in this sample presented with the depressive profile, and the most prevalent symptoms were irritability, abnormal thoughts and anxiety. It is important to note that women who presented with a depressive profile also demonstrated psychotic or manic symptoms; however, depressive symptoms were most prevalent. Data from South

India has shown that over half of women reported infant-related delusions which could affect mother-child interactions [62].

One major setback for diagnosis of postpartum psychosis is the lack of recognition in the DSM and ICD. As a result, diagnosis relies on women meeting the criteria for a severe mood episode as well as the post-natal onset specifier [2]. Women are therefore often misdiagnosed, which can contribute to inappropriate management plans and unwarranted stigma. Symptoms can fluctuate over time, and intrusive thoughts of harming their children are common for new mothers even within the general population, creating additional complications with diagnosis [63]. This may be further exacerbated by comorbidities such as autoimmune encephalitis which may be the root of psychotic symptoms. As such, extensive examinations and laboratory tests are necessary to determine whether an organic cause of symptoms is present [2, 55].

9.4.2 Primary Prevention

Women at high risk first need to be identified based on the described risk factors. They will require a prebirth plan, involving close family, friends and health care professionals [63]. This is necessary to reduce stress, increase support and coordinate all those involved [4]. Women should be advised to prioritise sufficient rest during the postpartum period, and many recommend beginning lithium prophylaxis immediately following childbirth [4, 55, 63]. Rates of recurrence are much higher for women with a history of postpartum psychosis should they decline prophylaxis [9, 16, 55]. Some research reports that response to lithium prophylaxis tends to cluster in families [64]; therefore, if the woman in question has a lithium-responsive family member, prescribing lithium is likely to be an appropriate option.

Other preventative measures that have been explored include antipsychotics and hormone therapy [9]; however there exists very little reliable evidence for the efficacy of these. Furthermore, for women with first-onset postpartum psychosis, preventative measures are unlikely to have been taken, meaning that treatment is the only option.

9.4.3 Treatment

To date, there have been no randomised controlled trials for the treatment of postpartum psychosis. This has contributed to a lack of internationally recognised, specific guidelines for treatment [9, 55]. Despite this, antipsychotics are typically used globally as the first-line treatment [55].

Most research has focused on lithium treatment, perhaps due to its success for patients with bipolar disorder. One specific treatment plan of note was developed at the Erasmus Medical Centre in the Netherlands. This consisted of a four-step additive method, utilising first benzodiazepines, antipsychotics, mood stabilisers and finally electroconvulsive therapy (ECT) [55]. Whether women required the next

level of treatment was dependent on whether their symptoms persisted. Using this method, the majority of women remitted within 2 months, which is consistent with other evidence for the efficacy of pharmacotherapy [63]. In fact, all women included in the study ($N = 64$), except for one, achieved full remission by step three of the method, suggesting that ECT is not necessary in the majority of cases. The most effective treatment was lithium and antipsychotic adjunctive therapy; however, the performance of these treatments alone was not tested and therefore cannot be supported based on this research [55].

ECT has been predominantly recommended for treatment of postpartum psychosis with depressive or catatonic features due to its longer disease duration than postpartum mania [4, 55]. The little evidence published suggests that ECT is highly effective, particularly for those with treatment refractory postpartum psychosis; however, this is based on dated research [3, 4]. More recently, some have advocated ECT as a first-line treatment option [65, 66]. ECT has also been credited with improving mortality rates associated with postpartum psychosis, after being more widely prescribed in the 1940s and inducing rapid symptom resolution [3]. Nonetheless, due to the efficacy of pharmacotherapy in many cases, ECT is often deemed unnecessary [55]. Women who are concerned about drug treatment and breastfeeding may choose ECT as this is reported to have minimal impact on the baby through breastfeeding [66]. The decision on whether individuals should use ECT or pharmacotherapy should ultimately be discussed with the patient and their family, with consideration of their specific symptoms and needs.

Women who require drug treatment are not expected to continue pharmacological maintenance therapy indefinitely. Particularly for those with episodes isolated to the postpartum period, gradual tapering off of lithium maintenance therapy is advised [4, 55]. This is providing that women remain in full remission for a significant period postpartum.

9.4.4 Prognosis

Following appropriate treatment, symptom remission can occur within several weeks [6, 55, 63]. Some reports show that women who exhibit manic features have less than half the duration of illness compared to women who exhibit mixed or depressed symptoms [4, 60]. The short-term prognosis for postpartum psychosis is therefore relatively positive; however, this is based on treatment compliance. Women who do not receive appropriate treatment suffer a much longer course of illness, around 8 months on average [55]. In addition, it has been reported that more than half of women require a year or more to achieve complete symptom resolution [13].

The prognosis tends to remain overall positive in the months following remission. One prospective follow-up study found that the majority of women reported good functioning, in terms of work and interpersonal relationships, 9 months postpartum [67]. Yet these women also reported higher rates of depression and anxiety, and the small subset of women who relapsed reported substantially worse functioning.

In the long term, the recurrence rate of postpartum psychosis has been reported to be high, although estimates are variable depending on a woman's personal and familial psychiatric history. Considering overall relapse rates for women with any psychiatric history of postpartum psychosis, bipolar disorder or both, one retrospective cohort study reported 52.2% in any subsequent delivery [13], whilst a meta-analysis which included this study reported an overall recurrence rate of 35% [16]. Similarly, a recent report suggests that women with a history of both bipolar disorder and postpartum psychosis have a 43% risk of severe relapse related to a subsequent pregnancy [7]. Whilst women with bipolar disorder have a postpartum relapse risk of around 35% following their first child, this risk decreases with subsequent pregnancies [16, 19]. For women with first-onset postpartum psychosis, the relapse risk for subsequent pregnancies is 31% [4, 16]. One review suggested that postpartum relapse is more severe for women with a history of postpartum psychosis compared to bipolar disorder [16]. Despite this, women with bipolar disorder are more likely to relapse during the postpartum period even with the use of lithium prophylaxis [59]. For women with first-onset postpartum psychosis, the risk of experiencing non-puerperal episodes after the incipient episode is high [4, 13, 16]. A recent meta-analysis using longitudinal data that spanned up to 26 years showed that women with first-onset postpartum psychosis (i.e. without co-existing bipolar disorder) have a 43.5% risk of recurrence outside of the postpartum period [22]. These episodes will typically fall within the bipolar disorder spectrum [4]. Indeed, the presence of any type of psychiatric episode during the postpartum period is expected to substantially increase a women's risk of developing bipolar disorder in the years following childbirth [18]. In spite of this, there are a number of women who only experience symptoms during the postpartum period [22].

9.4.4.1 Complications

An ongoing drawback in treatment and management is the lack of appropriate services. Within the UK, around half of women cannot access specialist services for perinatal mental health [21]. This figure is estimated by the World Health Organization to be much higher for women living in low- and lower middle-income countries [68]. The rarity of the disorder feeds into a lack of understanding within society, leading to women often being isolated socially as well. It has been estimated that one out of five marriages ends due to postpartum psychosis [69].

This absence of support can have severe consequences. Women are at 70% increased risk for suicide during the postpartum period compared to any other period in their lives; and for every 1000 women who experience postpartum psychosis, 2 complete suicide [3]. There have also been reports of a slight increase in risk of women with postpartum psychosis committing infanticide [28]; however, the data on this is minimal. These reports are already devastating, yet these cases are often exaggerated in mainstream media, contributing to the overall stigma endured by these women [63].

The relationship between mother and child can be adversely affected by a mother's mental state. Women with postpartum psychosis are less likely to have impaired mother-to-infant bonding compared to women with postpartum depression [70].

Although bonding can improve following treatment, some women will continue to experience impaired bonding which could impact the long-term development of their child. Women who have delusions about someone wanting to harm their child can remain affectionate and bond well with them, whilst women who believe their baby is evil may be more likely to be unable to care for its needs or harm them [62].

It is also important to consider the fact that cultural differences in the presentation and understanding of this disorder may be at play. The global prevalence of postpartum psychosis is relatively consistent [1]; however, it is possible that the concept of postpartum psychosis is incongruent between cultures. The types of symptoms that are expressed by women with postpartum depression have been found to differ even between Western countries that are considered comparable such as the USA and the UK [71]. Research from India has demonstrated that only 1/3 of women and their carers believed their symptoms had biomedical origins, with others citing psychosocial stressors or supernatural causes [72]. Psychoeducation may be beneficial to improve understanding and social support for women and the people around them.

9.5 Conclusion

Whilst childbirth can be a powerful trigger for many psychiatric disorders, few are as severe as postpartum psychosis. For some women, this may be the incipient episode of a bipolar-type disorder. For others, their symptoms will be limited to the postpartum period. The unique specificity of this disorder—isolated to an especially small window of time—allows for an unprecedented opportunity to study its aetiology. Research in this area has identified obstetric, hormonal, immunological and genetic risk factors. An understanding of these risk factors will effectively improve management. Fortunately, with appropriate care and planning, overall prognosis can be good, with women typically responding well to treatment. Several limitations of prevailing research within this area remain, illustrating the need for larger and more diverse samples. The potential benefits of this approach are immeasurable. Utilising international datasets will improve understanding of this disorder, which will lead to more informed nosology and homogeneity in diagnosis. This will, in turn, provide further insight into clinical management and treatment.

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