

Is postnatal depression a risk factor for sudden infant death?

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SUMMARY

Background: In New Zealand, an association has been shown between postnatal depression and sudden infant death syndrome (SIDS).

Aim: To replicate the New Zealand study.

Design of study: Case-control study.

Setting: The city of Sheffield, UK.

Method: The database of the Sheffield Child Development Study was used. Demographic and obstetric data were collected and at one month postpartum the Edinburgh Postnatal Depression Scale (EPDS) was administered. Detailed information on the cause of all infant deaths was available.

Results: There were 32 984 live births during the study period (from the year 1988 to 1993) and 42 babies died with the cause registered as SIDS. Multivariate analysis showed that smoking was the most important risk factor for SIDS (odds ratio [OR] = 7.24, 95% confidence interval [95% CI] = 2.76 to 19.01), followed by a high EPDS (OR = 3.20, 95% CI = 1.46 to 6.99) and residence in an area of poverty (OR = 2.33, 95% CI = 1.06 to 5.11).

Conclusions: The Sheffield data confirm the New Zealand findings. A high EPDS score and, by implication, postnatal depression, may be risk factors for SIDS; however, there are many possible explanations for the association.

Keywords: sudden infant death; postnatal depression.

Introduction

THE term 'sudden unexpected death in infancy' (SUDI) encompasses both the sudden infant death syndrome (SIDS) group, in whom no satisfactory cause for death is found and also babies who die unexpectedly, but in whom a sufficient cause of death is found at post mortem. It has been known for many years that SUDI is not distributed randomly in the population and that some of the risk factors, such as sleeping position and parental smoking, are potentially modifiable.¹ Parental unemployment and social disadvantage are also important factors,^{2,3} but how and why they are linked to infant death is still uncertain.

The Sheffield Child Development Study found an association between SUDI and parental stress, emotional or psychiatric illness, including suicide attempt and consideration of pregnancy termination.⁴ Confidential enquiries into families with more than one unexpected infant death showed a high rate of psychiatric illness.⁵ These observations suggested that parental psychological factors might play a part in some infant deaths.

The possibility that SUDI might be related to postnatal depression (PND) was raised by a study in New Zealand,⁶ after controlling for possible confounding factors, the rate of SIDS was found to be some three times higher in the infants of depressed mothers when compared with those of non-depressed controls.

We report here the results of a study to test the hypothesis that the incidence of SIDS is increased in the babies of women at risk of postnatal depression in a UK population and, if so, to examine possible reasons.

Method

This project was part of the Sheffield Child Development Study, which was established in the 1970s to provide city-wide data about factors related to sudden infant death⁷ and to evaluate the effect of increased health visitor care for high-risk infants (risk-related intervention).^{8,9} The study reported here used prospectively collected data about all births registered in Sheffield during the five-year period from 1 June 1988 to 30 May 1993.

The 'Back to Sleep' campaign, which was designed to encourage the supine sleeping position, was introduced during the study, but we have not included this in our analysis for two reasons. First, numbers of SIDS and SUDI cases before and after the campaign were too small for meaningful analysis; secondly, the method of data recording for this variable changed during the study period.

Data collection

Birth data comprised obstetric, neonatal, and socio-demographic data extracted from the birth notification form. At

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HOW THIS FITS IN*What do we know?*

Sudden unexpected death in infancy (SUDI) is known to have strong associations with poverty and deprivation. Postnatal depression (PND) is common and often undiagnosed.

What does this paper add?

One previous report from New Zealand found an association between SUDI and PND and this study confirms the association. There are probably multiple mechanisms.



one month of age, all infants were visited at home by their health visitor. A questionnaire was completed which covered a range of medical and social factors. 'Smoking' refers to mothers and is based on self reports.

Edinburgh Postnatal Depression Scale

The Edinburgh Postnatal Depression Scale (EPDS) was completed by the mother during this visit. The EPDS is a ten-item self-report screening instrument for postnatal depression. Scores above the cut-off level (12 or more) are referred to as high scores and those below this level as low scores. Women with high scores have a high probability of being depressed.¹⁰ Cox *et al*, who devised the EPDS, reported a sensitivity of 86%. Murray and Carothers¹¹ derived data from a larger ($n = 646$) community-based representative population at six weeks postpartum. They reported that the cut-off of 12.5 gave a specificity of 95.7%, a sensitivity of 67.7%, and a positive predictive value of 66.7%, using the Standardised Psychiatric Interview (SPI) as the reference test.¹² The corresponding values for a cut-off of 11.5 were 92.5%, 76.7% and 56.8%.

For reasons that we could not determine, a previous researcher in Sheffield had altered the wording of two of the ten items of the standard version of the EPDS. These items were questions three ('I have blamed myself unnecessarily when things went wrong') and seven ('I have been so unhappy that I have difficulty sleeping'). After initial analyses using the full scale, these two items were excluded from subsequent analysis, which was therefore based on an eight-item screening test with a cut-off score of 9 or more.

It was necessary to establish that these modifications to the scale, minor though they were, did not alter the validity of the EPDS. Their effect was examined using the original dataset collected by Murray and Carothers,¹¹ with the permission and help of the authors. The analysis included 229 women with known EPDS scores who had completed the SPI. On the basis of the SPI, women were divided into two groups — depressed ($n = 61$) and not depressed ($n = 168$). The cut-off score for the eight-question scale that corresponded most closely to the one used in the ten-item scale was 9 or more. We found that the eight-question scale had the same sensitivity and specificity as the standard ten-item scale in predicting clinical depression as diagnosed by the SPI. Interestingly, a combination of just four questions (numbers 2, 7, 8, and 10) categorised depressed and non-depressed women as well as the ten-item scale.

Commentary

This paper confirms an association between high scores on the Edinburgh Postnatal Depression Scale (and thus, very probably, depression in the late puerperium) and SIDS. The nature of the association is unexplained but several hypotheses are advanced, of varying plausibility.

Smoking is a higher risk factor for SIDS than depression and it might be supposed that stressed and depressed mothers would be more likely to be smokers, but the paper does not, I think, show this to be so. There is a lack of data about male partners; it is possible that a certain type (or types) of man and relationship could contribute to maternal depression and SIDS.

The suggestion that a puny baby might be more likely to suffer SIDS and to increase the mother's risk of getting depressed is persuasive, and pre-term birth was another risk factor for SIDS; though it was not, I think, shown to be associated with depression.

Turning to how depression could cause SIDS, it is true that depressed mothers may have thoughts of harming their babies, though these thoughts are far more often fears than wishes. Infanticide by women with non-psychotic depression is extremely rare. Depressed mothers have difficulty in playing with their children, and it is possible that a disorder which makes mothering such a burden could impair maternal care.

The take-home message (apart from the inevitable need for further research) that depressed women in the postpartum period require better identification and care is unexceptionable. However, the issue of an increased risk of SIDS needs to be handled with considerable sensitivity and discretion. The paper cites evidence that some women already falsify their responses to the EPDS for fear that their babies will be taken into care. The last thing that depressed mothers need is exacerbation of their guilt and fears.

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Effect of deprivation

The postcode of each mother was matched with demographic data to allocate an 'area of poverty' index (scored 1 to 4), which uses City Council data on the percentage of families in each postcode who receive housing benefit and/or income support.

Case definition

A case was defined as the death of an infant in the period between one week and one year of age, for which the registered cause of death following post mortem examination was given as unexpected death in infancy. This is synonymous with SIDS. It excludes deaths, which, though unexpected, were shown at post mortem to have a sufficient cause for death which was shown on the death certificate. The control group comprised all births where the infant was alive at one year of age.

Data handling and statistical methods

All data were stored on a computer using a unique identifier number to ensure anonymity. Two binary outcome variables were under investigation; a mother having an infant who died as a result of SIDS or a live infant, and having a high or a low EPDS score. The objective was to examine the relationship between each of these outcome variables and a

number of explanatory variables. The interaction between the outcome variables was also considered. Because of the inclusion of several categorical variables, the use of logistic regression was considered to be most appropriate. The stepwise logistic regression model was fitted by the method of maximum likelihood. Analysis was carried out using the statistical software package SAS®. Odds ratios (ORs) are quoted where relevant together with confidence intervals (95% CIs). An arbitrary 5% level of significance was adopted throughout.

Results

Descriptive data

There were 32 984 live births in Sheffield during the study period, 42 unexpected infant deaths registered as SIDS, and 92 post perinatal deaths from other causes (post perinatal mortality rate of 4.05/1000 live births). There were 4333 women with high EPDS scores and 24 583 with low scores; thus, 15% of those women for whom a score was recorded had high scores. No score was recorded in 4068.

EPDS data were available for 32 of the 42 women whose baby had died of SIDS. Of the other ten, four SIDS deaths occurred before one month of age and therefore no EPDS was available. Six SIDS deaths beyond the age of one month were infants of mothers for whom no EPDS score was recorded.

Univariate analysis

Of the 42 SIDS cases, 13 occurred in infants of the women with high EPDS scores, a rate of 3.0/1000 live births; and 19 SIDS deaths were in the infants of women with low EPDS scores, a rate of 0.77 per 1000 live births (univariate OR = 3.9; 95% CI = 1.9 to 7.9).

Missing EPDS data

To apply the most rigorous test to the hypothesis that there is an association between high EPDS scores and SIDS, we carried out a sensitivity analysis using two conservative assumptions with regard to the missing data. The first relates to the six SIDS cases after one month of age, among the babies of the 4068 women for whom no EPDS score was recorded. If it is assumed that all of these women had low scores, then the total SIDS rate would be 25 cases in 28 651 women with low scores, compared with 13 in 4333 women with high EPDS scores (OR = 3.5; 95% CI = 1.8 to 6.8). If, in addition, it is assumed that none of the mothers of the four babies who died before one month of age had a high score, then the rate would be 29 cases in 28 651 (OR = 3.0; 95% CI = 1.5 to 5.7).

Multivariate analysis

Table 1 summarises the findings in cases and controls for which data were complete. In addition to mothers' smoking status, residence in an area of poverty and pre-term birth, a high EPDS score also independently predicted an increased risk of SIDS (OR = 3.20; 95% CI = 1.46 to 6.99).

The following variables did not reach statistical significance within the model: maternal age; number of previous pregnancies; birth weight; number born (multiple births);

maternal psychiatric history; year of birth (within study); month or season of birth; number of health visitor visits; baby's sex; mother's feeding intention (breast or bottle); mode of feeding at one month (breast or bottle) or maternal satisfaction with the infant's feeding.

High EPDS scores may reflect a range of other influences on mental state. Table 2 shows the factors that were significantly associated with a high EPDS score. The single best predictor of a high score on the EPDS was the mother's dissatisfaction with the infant's feeding pattern at one month postpartum. An odds ratio of less than 1 indicates a significant association between PND and babies who were small for dates as well as those who were premature. There is an association between successful exclusive breastfeeding at one month and low EPDS scores.

We examined the effect of time on the observed variables. Maternal smoking showed a small but significant reduction with a linear trend evident over the five-year period. None of the other variables showed a significant change. There was no apparent seasonality effect that linked month of birth with likelihood of high EPDS score.

Interactions

The model was examined for any interaction between smoking and depression. None was found.

Discussion

This study, like that of Mitchell *et al* in New Zealand,⁶ had several limitations. Both studies suffered from a high rate of missing data, though in neither case was it likely that this would invalidate the conclusions. We found that even with the extreme assumption that all cases with missing EPDS data had normal EPDS scores, there was still an excess of SIDS in the babies of women with high scores. Secondly, the EPDS is a screening test and high scores are not diagnostic of depression, but it was not possible to validate the EPDS

Table 1. Factors predicting sudden infant death. This analysis included mothers of 26 babies who died with a diagnosis of SIDS and mothers of 24 750 survivors.

Variable	Odds ratio	Confidence interval
Maternal smoking (yes/no)	7.24	2.76–19.01
High EPDS score	3.20	1.46–6.99
Area of poverty (yes/no)	2.33	1.06–5.11
Gestation (weeks)	0.80	0.78–0.82

Table 2. Factors predicting EPDS score. This analysis included 3419 mothers with high EPDS scores and 20 181 with low scores.

Variable	Odds ratio	Confidence interval
Dissatisfaction with infant feeding	2.87	2.55–3.23
Previous psychiatric history	2.28	1.94–2.69
Maternal smoking	1.33	1.23–1.45
Number of health visitor visits	1.27	1.20–1.35
Area of poverty	1.14	1.04–1.26
Intention to breast feed	1.17	1.09–1.26
Previous pregnancies	1.08	1.05–1.11
Birth weight	0.93	0.87–0.99
Breast feeding at one month	0.63	0.58–0.69

results with data from psychiatric interview. The implications of this are discussed below. Thirdly, the EPDS used in Sheffield had been modified; however, we were able to show that this did not affect the validity of the scale. Fourthly, the EPDS was administered in our study and in that of Mitchell *et al* at four weeks postpartum, whereas the standardisation data were gathered at six weeks. EPDS scores can change, and postnatal depression may become more obvious over time; this suggests that, if anything, data collected at four weeks might underestimate the true prevalence of depression.

Depression is a common and important mental health problem for mothers of young children, but is frequently unrecognised by health professionals and sometimes by the mother herself.¹³ Postnatal depression has important implications for the baby as well as the parents, since the disturbances in mother–infant interactions associated with PND have long-lasting effects on infant development.¹⁴ However, the hypothesis that PND might be linked with SIDS is at first sight somewhat less plausible.

Our results show that a high EPDS score is a risk factor for SIDS and this remains significant after controlling for other possibly confounding variables; in particular, poverty and maternal smoking. This supports the findings of Mitchell and colleagues in New Zealand,¹ and the odds ratios were also similar (3.20 in our study and 3.27 in theirs).

There are several possible explanations for the association between a high EPDS score and SIDS. First, the EPDS is a screening test for women at high risk of depression — it does not confirm a diagnosis of clinical depression. Although the EPDS has been shown to have acceptable sensitivity and specificity as a screening test for clinical depression, the sensitivity at six weeks of age, using a cut-off of 12 or more, is only around 70%; in other words, almost one-third of cases are missed.¹¹

A systematic bias determining which cases were identified and which were missed might, however, produce misleading results, unless the EPDS is directly validated on a large sample of the population being studied. In another study in Sheffield (Cubison, personal communication, 1999), some women attending a group treatment programme for severe postnatal depression explained that they had deliberately provided incorrect answers to the EPDS for fear that their infant might be taken into care. We do not know how common this phenomenon might be but, although it seems unlikely that this can explain our findings, the possibility cannot be entirely excluded.

Secondly, the high score may be a proxy for other factors that are more directly linked with infant death. Although the effect remains after taking account of the two most obvious candidates — residence in an area of poverty and maternal smoking — the high EPDS score was itself correlated with a number of other social, environmental or personality variables.¹⁵ Our dataset did not include all the factors that might be important; for instance, there were no data about the male partners of these women.

Thirdly, women are told that smoking is bad for babies,¹⁶ but those who cannot stop are advised not to smoke or to permit smoking near the baby. Anecdotal evidence suggests that most women do try to heed this advice, but per-

haps those who are depressed have more difficulty in controlling their infant's smoke exposure.¹⁷ The study did not have sufficient power to investigate this adequately but, in view of the major significance of smoking in relation to SIDS, it is a plausible explanation.

The fourth possibility is that women who are depressed may be less able to care for their infants. This could be mediated in several ways. There is a relationship between stopping breastfeeding and depression,¹⁸ but although breastfeeding protects against some infections there is little or no difference in the SIDS rates between breast and bottle-fed infants in the UK,¹⁹ so this is unlikely to be the explanation. The link between dissatisfaction with the infant's feeding, depression, and SIDS might be mediated by poor weight gain and failure to thrive. Depression might reduce the mother's ability or willingness to identify the signs of illness in her infant, resulting in a delay in obtaining medical care. We were unable to determine whether there was a link between a high EPDS and death from a diagnosable or treatable cause, since the numbers in this latter group were too small. Disturbed mother–baby interaction might in theory affect neurological functioning; for instance, by affecting the baby's sleep patterns,²⁰ but there is no evidence that this could result in infant death.

A fifth possibility is that infants who are less physiologically stable or mature might be more likely to induce depression in their mothers and such babies might also be more likely to suffer SIDS. Murray and colleagues found that 'poor infant motor scores and high irritability were strongly predictive of the onset of maternal depression by eight weeks postpartum. These effects, obtained after taking account of both maternal mood in the neonatal period and maternal perceptions of infant temperament'.²¹

Finally, painful though it is, we cannot ignore the possibility that some infant deaths may not be accidental.²² Post mortem findings are often non-specific and it may be impossible to distinguish between genuine SIDS and death due to imposed airway obstruction.^{23,24} Reports of infanticide and obsessional thoughts about infanticide by women with severe depression or psychotic illnesses,^{25,26} and interviews with mothers whose infants have died of supposed SIDS, indicate that not all deaths are accidental or inexplicable, but the proportion of all SIDS that might be accounted for in this way is unknown.²⁷

In summary, our study supports that of Mitchell and colleagues in New Zealand, suggesting that postnatal depression may be an independent risk factor for SIDS. Although the nature of the association is unknown, and needs further study, our findings strengthen the case for better identification and care of depressed women in the postnatal period and health professionals should be aware that infants of depressed mothers may be at increased risk of SIDS.

Conclusions

The study had three main limitations: EPDS data were not recorded on a significant proportion of subjects; the women with high EPDS scores did not undergo a standardised diagnostic assessment; the possibility is raised that some women's answers to the EPDS may not reflect their mental state.

The results suggest that postnatal depression may be associated with an increased risk of unexpected death in infancy.

Intervention programmes to identify and treat depression should recognise the probable multifactorial nature of the associations.

The identification and treatment of postnatal depression may be important for the safety and wellbeing of the infant as well as that of the mother.

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