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Mania Following Bereavement

SIR: I read with interest Rosenman and Taylor's case report of mania following bereavement (*Journal*, April 1986, **148**, 468–70). The authors state that such reports showing this association are uncommon. I report two further cases.

Case reports: (1) A lady who had no previous psychiatric history, first presented aged 49 years, two months after the sudden death of her husband. He had taken his own life whilst she was at work. At first, she grieved appropriately but six weeks after her bereavement she became restless, irritable and garrulous. She returned to normal mood within one month following treatment with neuroleptic medication and ECT. Four years later her mother died of carcinoma. She grieved initially but soon became cheerful. By the time of presentation, one month after her bereavement, she was restless, overtalkative, sexually disinhibited, giggly and expressed paranoid ideas with regard to her neighbours and sons. She said that nursing her mother for eight years had imposed a great strain on her and that her behaviour was a reaction to the lifting of this strain. She became euthymic within two months on treatment with haloperidol but 18 months later presented with a further manic episode. This occurred three weeks after a celebration in her husband's family to which she had not been invited and she had been initially very upset. She has been well in the nine years since the last affective episode.

(2) A 58 year old lady with a previous history of bipolar affective disorder presented the day after the funeral of her husband who had died suddenly of a myocardial infarct one week previously. Within 24 hours of his death she became restless, overtalkative and insomniac. On admission she talked incessantly and maintained that she felt "hilarious" in spite of occasional tearfulness. She believed she had special powers of healing people and that the television was telling her what to do. She could hear her husband talking to her. Her mood gradually stabilised on treatment with haloperidol, but one month after the bereavement she became depressed. She was then successfully treated with an antidepressant and discharged. One year later she presented with depression requiring treatment with ECT. Her mood stabilised but after a visit to her husband's grave three months later, she became manic with mixed affect. This resolved and she has been well for the last six months.

Rosenman and Taylor discuss the mechanism of manic response to bereavement. They cite the Post *et al* (1981) finding that a previous history of affective disorder predisposes to a rapid onset of mania. These two cases support this: (1) with no previous history of affective disorder did not develop mania until six weeks after her husband's death and (2) with a well established bipolar affective disorder developed mania within 24 hours of her bereavement. That repeated episodes of illness establish a facilitated pathway by which rapid changes of mood could occur, may be further supported by the recurrence of mania following case 2's visit to her husband's grave 15 months after his death, and following case 1's perceived rejection by her in-laws.

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SIR: The case report by Rosenman and Taylor (*Journal*, April 1986, **148**, 468–470) of mania following bereavement was of considerable interest. I report another two cases.

Case reports: (1) A 46 year old divorced engineer was admitted as an emergency in a hypomanic state on the evening of his mother's funeral. Instead of returning home from the funeral he had gone to his place of work where his behaviour had caused concern, the work's medical officer had arranged admission. On admission he was dressed in a

morning suit and was constantly pacing the floor, his mood elevated, he was overbearing and slightly aggressive in manner. There was marked pressure of speech and flight of ideas. This state persisted for six weeks and was followed by a period of depression. There is no evidence that he was hypomanic prior to his mother's death. There had been a hypomanic episode some two months beforehand but this lasted less than a month and there is well documented evidence that his mood remained stable up to the time he had leave from work after his mother's death.

Almost two years later his father died and five days afterwards he was again admitted in a similar state. Again there was no evidence to suggest that his mood was elevated before the event.

This patient had an established manic-depressive disorder and had been subject to mood swings with no apparent precipitant. On these two occasions and on a possible third occasion when a hypomanic episode followed his wife leaving him, there was a clear precipitating event.

(2) A 24 year old single woman was admitted from the police station in a manic state. She had been found wandering naked and was grossly overactive, her mood alternated between elation and aggression, her speech was constant, with flight of ideas, punning and clang associations. Three weeks previously her adoptive father had committed suicide. She had felt pressure from the family as there had been a suggestion that she had been responsible for her father's depressive illness. During the three weeks after his death she had become progressively more disinhibited, over-active and promiscuous. Again, there is no suggestion that her mood was different before the death.

She had no previous history of affective disorder. However, a year before this episode she had been diagnosed as having a personality disorder. It is possible that her behaviour at this time indicated a mild hypomanic episode. Since the reported incident she has had one depressive swing starting three days after she gave birth. This depressive swing was followed by a period of mild hypomania.

I am grateful to Dr F G Spear, Consultant Psychiatrist, for permission to report these cases.

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Lithium Carbonate and Piroxicam

SIR: The report by Walbridge and Bazire (*Journal*, August 1985, **147**, 206–207) of an interaction between lithium carbonate and piroxicam prompts us to report details of a patient who also experienced lithium toxicity, probably attributable to this combination.

Case report: A 62 year old female with a 20 year history of manic depressive illness was started on lithium in March 1984 during her twelfth admission to hospital. Initially her renal function was checked and showed a plasma creatinine of 80 μmol/L. Her plasma lithium was stabilised at 0.6–0.75 mmol/L (checked monthly) with a dose of 800 mg of lithium carbonate at night. Ten months later, on 24.1.85, piroxicam 20 mg daily was prescribed for osteoarthritis of her right knee. Concurrent medication was as follows: amitriptyline 150 mg at night from 16.1.85 (previously on dothiepin 75 mg at night started before 1984), chlorpromazine 175 mg daily (from before 1984), procyclidine 5 mg tds from 14.1.85, and temazepam 20 mg at night from 11.1.85. Her January lithium level (taken on 8.1.85) was slightly raised at 0.85 mmol/L, possibly due to a recent loss of weight. At the end of January, she became noticeably agitated and tremulous: and during the next few days developed ataxia. During this period she was eating and drinking very little. A lithium level, done on 7.2.85 was 2.85 mmol/L with a raised plasma urea of 12 mmol/L and plasma creatinine of 142 μmol/L. All drugs were stopped and i/v fluid replacement started. Plasma lithium fell in four days to 0.35 mmol/L and plasma creatinine was inside the normal range (60–120 μmol/L) within 48 hours. Renal function tests continue to show mid-range values at the present time. She was also treated for an E. coli urinary tract infection with Amoxycillin starting 8.2.85. The lithium toxicity in this patient was probably exacerbated by dehydration and a urinary tract infection, but we consider that a piroxicam/lithium interaction could have contributed significantly.

Although the lithium/non-steroidal anti-inflammatory drug (NSAID) interaction has been well established, the mechanism is not completely understood. One mechanism, that of suppression of renal prostaglandin synthesis by NSAIDs leading to a fall in GFR and thus a reduced clearance of lithium has been clearly implicated in some cases (Nadarajah & Stein, 1985). This is reversible but the risk may be greater with NSAIDs with a long half-life (Adams *et al*, 1986). Walbridge and Bazire suggest using an NSAID with a short half-life, e.g., ibuprofen, to reduce the extent of the interaction; however reports have shown marked increases in plasma lithium concentration within a short time of initiation of ibuprofen therapy (Ayd, 1985; Leftwich *et al*, 1978).

In the light of this, it has been of concern to us that ibuprofen is now a non-prescription drug in the UK and can be bought freely over the counter. With this in mind, we now counsel our lithium patients on various aspects of their therapy with the use of information cards which specifically refer to the possibility of an interaction with ibuprofen. We ask our patients to show their card to any chemist they buy medicines from.

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