was at my local accident and emergency department, where I was told that I was a “waste of time” because I was worried that my lithium level was too high. I also recall, on a separate occasion, being asked to leave the hospital canteen because I was a psychiatric patient. It is because of these and other instances that I believe that one of the biggest harbourers of stigma is general medicine in hospitals. To the patient being told to “Pull yourself together” after a suicide attempt. That isn’t going to help anyone, and it is crucial for those in attendance to be willing to listen to the patient and act responsibly in a caring and supportive manner. Staff in accident and emergency departments especially must learn how to deal effectively with psychiatric patients as they are often the first port of call for those in acute mental distress with the same being applicable to police forces and general practitioners.

Finding some shoes

Do I have mental health problems? Hmm, yes, I suppose so—but I prefer to bring the terminology into the 21st century and call them mental health

Further information

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challenges. Now that I seem to be on the correct medication, I am, for the most part, able to lead a full and varied life. I play hockey, I’m the lead guitarist in a rock band, and I write and advise on mental health issues. My life, albeit as a person with bipolar affective disorder, is far from over. I’m certainly not giving up on myself, and even when I’m ill I gain reassurance from those around me that this is not the end and that I will regain the life that seems to have shattered into a million pieces during this particular crisis. Life is like balls being thrown at you from all sides of a tennis court. I’m still working on my backhand. How good is yours?

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Commentary: Bipolar disorder—services need to catch up with rapidly developing evidence base

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The article above powerfully describes the misery and anguish that bipolar disorder causes.¹ Bipolar disorder is characterised by disabling swings of mood between mania (and mixed mania and depression) and depression. In the Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV), it is subdivided depending on the severity of the manic symptoms: bipolar I disorder is diagnosed when episodes of mania or mixed episodes occur with or without episodes of depressive illness, and bipolar II disorder is diagnosed when depression occurs with less severe episodes of elevated mood (hypomania) that do not lead to dysfunction or disability.^{w1} The lifetime prevalence of bipolar I disorder is around 0.5%; including bipolar II and less severe bipolar spectrum disorder increases this to around 5%.² The aetiology of bipolar disorder remain uncertain. It is strongly familial, although the precise genetic causes remain elusive.³ After the first diagnosis of mania, most patients achieve symptomatic recovery, but relapse is common, and after four years only about one third recover to premorbid function.³ Bipolar disorder is now recognised to be one of the commonest causes of worldwide disability especially within the 15–44 age group.³ Despite that, the needs of patients with bipolar disorder are almost completely ignored by UK policymakers, which makes service development very difficult.^{w2} Compared with schizophrenia, bipolar disorder is under-researched.^{w3}

Management of bipolar disorder includes specific treatment for the acute manic or mixed and depressive phases of the illness and long term strategies for relapse prevention. Historically, most effort has been put into researching pharmacological

treatments for acute mania. Antipsychotics and other drugs, including lithium and valproate, are effective in the acute phase of mania.³ There is very little evidence that psychosocial interventions are effective in mania. There is still limited evidence for interventions in the depressive phase of the illness, despite the fact that this accounts for the most substantial disability associated with the disorder.^{w4 w5} although this situation is rapidly improving. Antidepressants such as selective serotonin reuptake inhibitors seem to alleviate symptoms of depression, although trial data are scarce,^{w6} and the risk of precipitating manic or mixed episodes and mood destabilisation means that combination therapy with an antimanic drug is advised when there is a history of mania (such as bipolar I disorder).² For that reason, other drugs such as mood stabilisers (lithium, valproate, lamotrigine) and, increasingly, atypical antipsychotics, for example, quetiapine, are often recommended as first line therapies.^{2 4 w7} The current draft NICE guideline amply demonstrates the substantial uncertainty that currently prevails about the most appropriate first line treatment for bipolar depressive episodes,⁴ and the BALANCE 2 trial currently in development seeks to address this (<http://cebmh.warne.ox.ac.uk/balance/balance2/index.html>). Very little evidence currently supports the use of psychosocial interventions in acute depressive episodes in bipolar disorder.³

Bipolar disorder is often a relapsing illness, and many patients will need long term chronic disease

See web references w1–w9 on bmj.com

management to prevent relapse and optimise functioning. Lithium carbonate has long been the main long-term drug and there is reasonable evidence that it is effective, particularly in preventing manic episodes.⁵ Lithium also appears to have an antisuicidal effect in mood disorders.^{6,8} More recently, fashion and convenience have led to an increase in the prescribing of valproate, and the evidence for this drug is now accumulating.⁴ The BALANCE trial comparing lithium, valproate, and combination therapy with lithium plus valproate is currently under way (<http://cebmb.warne.ox.ac.uk/balance/balance1/index.html>). In general the long term drug treatment of bipolar disorder is under-researched, and many drugs, particularly antiepileptics and unproved combinations, are used off-licence and with little evidence of efficacy. Psychosocial interventions such as family psychoeducation, cognitive behaviour therapy, and symptom recognition training are promising, although the evidence is still fairly limited.³

The optimal long term management of bipolar disorder remains a specialist activity entailing expert psychopharmacological management, psychosocial interventions, and self management. Evidence is emerging that specific collaborative disease management programmes may improve outcomes.^{6,9}

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Interactive case report

An alcoholic patient who continues to drink: case presentation

Stuart McPherson, Colin John Rees

Mr Bond is a 42 year old man with alcoholic cirrhosis who was admitted to our unit with haematemesis. He had had three previous admissions with alcohol related problems and had twice bled from oesophageal varices. The varices had been treated by sclerotherapy and banding, and he was taking propranolol 40 mg twice daily as secondary prophylaxis. His most recent endoscopy had shown three, barely noticeable oesophageal varices.

Mr Bond has a history of excessive alcohol intake over five years, with no periods of abstinence. He was drinking 3-4 litres of strong cider a day up until the day of admission. A consultant psychiatrist had previously diagnosed depression and alcohol dependence syndrome. Although Mr Bond had been treated with paroxetine, he discontinued it as he thought it was unhelpful. He had also seen the drug and alcohol team several times. His attendance at appointments with the psychiatrist and drug and alcohol team was sporadic as he felt that these were of little benefit to him. He complied with medical drug treatment.

Results of investigations at presentation

Investigation	Result (normal range)
Haemoglobin (g/l)	114 (130 to 180)
Platelets ($\times 10^9/l$)	53 (150 to 450)
Mean corpuscular volume (fl)	90 (76 to 96)
Prothrombin time (seconds)	19 (10 to 15)
Serum urea (mmol/l)	2.9 (2.5 to 7)
Total bilirubin ($\mu\text{mol/l}$)	82 (0 to 17)
Alanine aminotransferase (U/l)	63 (5 to 40)
Alkaline phosphatase (U/l)	123 (30 to 125)
Albumin (g/l)	30 (35 to 50)

Questions

1 What are the likely causes of Mr Bond's bleeding?

2 How would you rate the severity of this bleed?

3 What initial steps should be taken in his management?

4 Given Mr Bond's continued alcohol consumption, how far should we go with treatment?

Please respond through bmj.com, remembering that Mr Bond is a real patient and that he and his carers will read your response

Mr Bond lives with a partner and has three children from a previous marriage, whom he rarely sees. He worked for the local council for 22 years but stopped working several years ago because of his alcohol dependence and depression. His marriage break-up a few years earlier had precipitated particularly heavy drinking. Mr Bond describes himself as a worrier and turns to alcohol when things go wrong. His mother died at the age of 60 from alcoholic liver disease.

On presentation with gastrointestinal bleeding he was jaundiced and showed signs of chronic liver disease (table). He was not encephalopathic. His pulse was 140/min and blood pressure 118/67 mm Hg with no appreciable postural drop. He had mild ascites but no hepatosplenomegaly. Per rectal examination showed melaena.

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This is the first of a three part case report where we invite readers to take part in considering the diagnosis and ethical issues surrounding the management of a case using the rapid response feature on bmj.com. Next week we will report the case progression, and in four weeks' time we will report the outcome and summarise the responses