Dr B, General Practitioner

A Report by the

Health and Disability Commissioner

(Case 13HDC00048)

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Executive summary

Background

1. In 1996 Mr A, then in his teens, was diagnosed with depression and bipolar affective disorder.[1] Mr A's psychiatrist prescribed him lithium carbonate (lithium) as a mood stabiliser.

2. On 11 October 2002 Mr A was transferred to the care of general practitioner (GP), Dr B,[2] at a medical centre. Dr B continued to prescribe lithium for Mr A. Dr B did not inform Mr A of the risks of lithium treatment at any stage.

3. Between 2002 and 2007 Mr A experienced a range of symptoms including severe constipation, faecal impaction, vomiting, anorexia, weight loss, dehydration and episodes of polyuria.[3]

4. Between 2002 and 2007 Dr B monitored Mr A's lithium levels inconsistently, on one occasion not ordering any tests for two years. Dr B monitored Mr A's renal function at inconsistent intervals of between one and eleven months. Initially Mr A's blood tests for renal function were normal, however, from July 2005 they began showing abnormalities, including elevated creatinine levels.

5. Between 2002 and 2007 Dr B referred Mr A to a number of different health providers including Community Mental Health, the Gastroenterology Outpatient Department at the DHB, and private gastroenterologist Dr C.

6. On 6 March 2006 Dr C wrote to Dr B advising him that Mr A had a raised creatinine level and his lithium level was "outside the normal range" indicating evidence of mild renal impairment. Dr C recommended a reduction in lithium dose.

7. In early 2007 Dr B referred Mr A to psychiatrist Dr E. Dr E advised Dr B to discontinue Mr A's lithium treatment owing to renal impairment evident on blood tests. Following Dr E's advice, Dr B ceased Mr A's lithium treatment in June 2007.

8. Dr B tested Mr A's renal function only once a year in 2008, 2009 and 2010. The 2009 and 2010 test results were abnormal.

9. There is no record that Dr B advised Mr A of his test results at any time.

10. In October 2010 Mr A was diagnosed with chronic kidney disease.

Findings

11. As Mr A's GP, Dr B had the responsibility to monitor Mr A's renal function and the levels of lithium in his blood, and to act appropriately on any abnormal results. The

[1] Individuals with bipolar disorder experience episodes of an elevated or agitated mood known as mania (or hypermania, depending on the severity) alternating with episodes of depression.
[2] Dr B is a vocationally registered GP. Dr B obtained vocational registration in July 2012.
[3] Excretion of abnormally large amounts of urine.

failure by Dr B to monitor Mr A's treatment adequately was a breach of Right 4(1)[4] of the Code of Health and Disability Services Consumers' Rights (the Code).

12. In addition, the Commissioner found that Dr B failed to inform Mr A of his abnormal test results and, consequently, Mr A was unable to make informed choices about his treatment. In doing so, Dr B breached Rights 6(1)(f)[5] and 7(1) of the Code.[6]

13. The Commissioner also found that Dr B was aware of the need to monitor Mr A and as such Mr A should have been made aware of the ongoing importance of monitoring. By doing so, Dr B would have further empowered Mr A in participating in his care.

14. The Commissioner found that it was appropriate for Dr B to continue lithium treatment until he was advised by an appropriate specialist to discontinue the treatment.

Complaint and investigation

15. The Commissioner received a complaint from Mr A about the services provided to him by Dr B. The following issues were identified for investigation:

- *Whether Dr B provided an appropriate standard of care to Mr A between October 2002 and October 2010.*

16. An investigation was commenced on 11 June 2013.

17. The parties directly involved in the investigation were:

Mr A	Consumer/complainant
Dr B	General practitioner
Also mentioned in this report	
Dr C	Gastroenterologist
Dr D	Psychiatrist
Dr E	Psychiatrist
Dr F	General practitioner
Dr G	General practitioner
Dr H	General practitioner
Dr I	General practitioner

[4] Right 4(1) of the Code states: "Every consumer has the right to have services provided with reasonable care and skill."
[5] Right (6)(1) of the Code states: "Every consumer has the right to the information that a reasonable consumer, in that consumer's circumstances, would expect to receive, including … (f) the results of tests."
[6] Right 7(1) of the Code states: "Services may be provided to a consumer only if that consumer makes an informed choice and gives informed consent, except where any enactment, or the common law, or any other provision of the Code provides otherwise."

18. Information was also reviewed from ACC and the district health board.

19. Independent expert advice was obtained from in-house clinical advisor Dr David Maplesden (**Appendix A)**.

Information gathered during investigation

Background

20. In 1996 Mr A, then in his teens, was diagnosed with depression and bipolar affective disorder. On 26 September 1996 Mr A's psychiatrist prescribed him lithium carbonate 250mg daily as a mood stabiliser. Between 1997 and 2002 Mr A was on an increased dosage of 400mg twice daily.

Effects of lithium treatment and appropriate monitoring

21. Lithium is used most commonly as a mood stabilising drug in the treatment of bipolar disorder. Potential adverse effects of lithium treatment include mild gastrointestinal effects (such as nausea, vomiting and diahorrea), fine hand tremours, and polyuria. Skin conditions such as rashes and leg ulcers may be aggravated by treatment. Less common, but potentially more serious effects include impaired renal function caused by dehydration, nephrotoxicity,[7] thyroid disorders, nephrogenic diabetes insipidus,[8] and hyperparathyroidism.[9]

22. With respect to nephrotoxicity, the potential risks of lithium treatment has become increasingly well known since the mid 2000s. Prior to this, the risks of lithium treatment were less clear, although it was accepted that the medication had a narrow therapeutic range and that patients on lithium treatment should be closely monitored.

23. It is important to check for existing renal impairment from unrelated causes, and to monitor renal function in patients on lithium treatment because of the risk of nephrotoxicity caused by lithium treatment, or the need to adjust the dose, or stop therapy in patients with existing renal impairment from unrelated causes. It is also important to test the level of lithium in a patient's blood, especially in the event that renal function is found to be abnormal, in order to ascertain whether impaired renal function could lead to lithium toxicity.

24. Best practice suggests monitoring of both lithium levels and renal function to be three-monthly. However, six- to twelve-monthly is common practice in patients with normal, stable results. Where a patient has abnormal results, normal practice would require a repeat test shortly after the abnormal result, in order to ascertain whether the

[7] The poisonous effect of some substances, both toxic chemicals and medication, on the kidneys.
[8] Nephrogenic diabetes insipidus is caused by an improper response of the kidney to antidiuretic hormone, leading to a decrease in the ability of the kidney to concentrate the urine by removing free water.
[9] Hyperparathyroidism is overactivity of the parathyroid glands resulting in excess production of parathyroid hormone (PTH). The parathyroid hormone regulates calcium and phosphate levels and helps to maintain these levels.

result was temporary, or ongoing. Some at-risk patients, for instance those with unstable renal function, require more intensive monitoring.

Transfer of care to Dr B and continuing lithium treatment

25. On 11 October 2002, Mr A transferred to Dr B's care. Dr B is a vocationally registered GP working at the medical centre. Dr B continued to prescribe Mr A with lithium.

26. Dr B advised HDC that when he took over Mr A's care in October 2002, he believed that Mr A's lithium treatment was "fully established". Dr B stated:

> "[Mr A] had shown a good clinical response to the lithium treatment and other prescribed medication. As [Mr A] had been on lithium medication for many years before becoming my patient, it was my understanding that the risks of using the prescribed medications, including lithium, would have been explained to [Mr A] by the many specialists involved with [his] care prior to my involvement."

27. Dr B did not inform Mr A of the risks of lithium treatment. Dr B stated that he would not normally go over the risks of medication with a patient who had been on treatment for this length of time.

Initial monitoring of lithium treatment

28. In December 2002 initial blood tests were ordered to monitor Mr A's renal function, and the results were normal. Dr B's monitoring of Mr A's renal function and lithium and creatinine[10] levels between 2002 and 2010 is outlined in the table attached (refer to **Appendix B**).

29. From 2002 until 2003 Mr A was engaged with Community Mental Health (CMH). His diagnosis of bipolar disorder was maintained, and Dr B continued to prescribe lithium treatment. In November 2003 Mr A was discharged from CMH on the basis that his bipolar disorder was stable while he was on medication.

30. During 2003 Mr A experienced episodes of polyuria. On 28 January and 23 December 2003 Dr B ordered tests regarding Mr A's renal function. The results of those tests were normal (refer to **Appendix B)**.

31. In early 2004 Mr A developed symptoms including severe constipation, faecal impaction, vomiting, anorexia and weight loss. Dr B ordered tests to monitor Mr A's renal function on 5 January and 9 March 2004, the results of which were normal. From April 2004 Mr A had periods of chronic dehydration and, according to Dr B's records, "general un-wellness". On 26 October 2004 Mr A's clinical notes record that another GP at the medical centre ordered tests to monitor Mr A's renal function and to measure the level of lithium in his blood. This was the first time that tests had been ordered to check Mr A's lithium levels since he had come into Dr B's care. The results showed no renal function impairment or lithium toxicity, although Mr A's creatinine levels were at the high end of normal.

[10] Creatinine is a waste product made by the muscles. Creatinine passes into the bloodstream and is usually passed out in urine. A high blood level of creatinine can indicate kidney failure.

32. In February 2005 Dr B referred Mr A to the Gastroenterology Outpatient Department. The hospital returned the referral to Dr B owing to "significant resource constraints" as Mr A was considered a "non-urgent" case.

First abnormal results

33. During 2005 Mr A continued to experience intermittent vomiting. The clinical notes show that Dr B ordered tests to monitor Mr A's renal function in January and July 2005. The January test results were normal, but the results dated 19 July showed elevated creatinine levels of 0.15 mmol/L[11] (refer to **Appendix B**). At this time, Mr A had been on lithium treatment for around nine years.

34. Dr B stated that he did not consider that Mr A would have had renal failure in July 2005, because the average duration of lithium treatment prior to development of renal failure is 28 years.[12] Dr B further stated that when Mr A's renal function began to deteriorate he "mistakenly attributed this to the continuing gastrointestinal problems" that Mr A had had since 2004.

35. Dr B did not inform Mr A of the abnormal test results dated 19 July, and he did not order any follow-up tests at this stage. Dr B next ordered tests to monitor Mr A's renal function in December 2005. The tests were undertaken in January 2006, and indicated elevated creatinine levels at 0.15 mmol/L (refer to **Appendix B**).

Referral to Dr C

36. In January 2006, Dr B referred Mr A to a private gastroenterologist, Dr C. Dr C diagnosed Mr A with gastro-oesophageal reflux disease.[13]

37. On 15 February 2006 Dr C reported to Dr B that Mr A continued to lose weight. Dr C noted that he had checked Mr A's lithium levels "to make sure there [were] no toxicity problems contributing to his symptoms". Blood tests dated 17 February 2006, ordered by Dr C, indicated possible lithium toxicity and elevated creatinine (refer to **Appendix B**).

38. On 6 March 2006 Dr C wrote to Dr B advising him that Mr A had a raised creatinine level of 0.15 mmol/L and his lithium level was 1.2 mmol/L[14] and "outside the normal range". Dr C stated: "… [T]here is evidence of mild renal impairment. It would probably be appropriate to reduce [Mr A's] lithium dose …" Dr B's clinical notes for Mr A dated 15 March 2006 state: "Reduce dosages of lithium …"

39. On 12 April 2006 Dr B ordered Mr A's lithium levels and renal function to be re-tested. The results of these tests showed lithium levels within the recommended

[11] Normal creatinine levels are between 0.06–0.12 mmol/L.
[12] This is confirmed by HDC's clinical advisor, Dr David Maplesden, who referred to the 2010 Medsafe prescriber update in his advice.
[13] Gastro-oesophageal reflux disease is a condition where the lower oesophageal sphincter (the muscular ring at the lower end of the oesophagus) is abnormally relaxed and allows the stomach's acidic contents to flow back or "reflux" into the oesophagus. Mr A's diagnosis of gastro-oesophageal disease was not related to his lithium treatment.
[14] Normal lithium levels (0.4–1.0 mmol/L) Possible toxicity (1.0–1.5 mmol/l) Toxic (>1.5 mmol/L). Normal creatinine levels (0.060–0.120 mmol/L) or (50–120 µmol/L).

therapeutic range. On 22 June 2006 Mr A consulted Dr B and, on the same day, another GP at the medical centre ordered renal function tests for Mr A.[15] Again, abnormal results were noted with elevated creatinine (refer to **Appendix B**).

40. However, on 17 August 2006 Dr C wrote to Mr A advising him of his latest laboratory results, stating: "Your lithium level [is] back in the normal range at 0.8mmol/L." There is no record of the tests that Dr C wasreferring to in Mr A's clinical notes.

41. On 21 September 2006 Dr B ordered further blood tests to check Mr A's renal function. The results of these tests were abnormal, showing elevated creatinine (refer **Appendix B)**. These results are not acknowledged in the clinical notes and were not communicated to Mr A, and Dr B did not order a test to ascertain Mr A's lithium levels at this time.

Continuing therapy until 2007

42. Dr B continued Mr A's lithium treatment until June 2007 because he understood that this was "an important medication for [Mr A's] psychiatric condition based on the information from specialists".

Referral to Dr E

43. On 10 January 2007 Dr B referred Mr A to psychiatrist Dr E, for an opinion regarding Mr A's initial diagnosis of bipolar disorder.[16]

44. On 16 May 2007 Dr E ordered blood tests for Mr A. The results were abnormal, showing significantly elevated creatinine levels (refer to **Appendix B)**. On 22 May 2007 further blood tests were ordered by a GP at the hospital, and again the results showed significantly elevated creatinine. On the same day, Dr B referred Mr A for a barium meal examination[17] to assess for gastric reflux. The results showed no abnormalities consistent with his symptoms.

45. On 30 May 2007 Dr E wrote to Dr B advising him to discontinue Mr A's lithium treatment. Dr E stated:

> "The blood test had returned with a raised creatinine level and I suggested to [Mr A] that we discontinue his 400mg of lithium altogether. It was unclear to me whether the raised creatinine level could be due to dehydration from vomiting or whether it was due to his use of Lithium."

46. With regard to Mr A's psychological condition, Dr E stated:

[15] A letter dated 21 June 2006 from the hospital to Dr B states: "GP follow up regarding … [urea and electrolytes], [liver function] … GP to arrange review of psychiatry medications."
[16] Dr B recorded in Mr A's clinical notes "refer [Dr E] for advice".
[17] A barium meal is a diagnostic test used to detect abnormalities of the oesophagus, stomach and small bowel using X-ray imaging. X-rays can highlight only bone and other radio-opaque tissues and would not usually enable visualisation of soft tissue. However, infusion of the contrast medium barium sulfate, a radio-opaque salt, coats the lining of the digestive tract, allowing accurate X-ray imaging of this part of the abdomen.

> "[Mr A] has ongoing concerns about experiencing another hypermanic or manic episode as he had apparently done in the past. Fortunately at this point in time there does not appear to be a major issue of him experiencing a manic episode … Ideally [Mr A] would be stabilised on a mood stabiliser … to prevent him from having a manic episode and to take a non-habit forming antidepressant to counteract anxiety, panic and depression issues."

47. Dr B stopped Mr A's lithium treatment. In June 2007 Dr B referred Mr A to the DHB Inpatient Mental Health Services following persistent vomiting. Tests ordered by a GP at the medical centre were normal (refer to **Appendix B**).

48. Over the next 12 months Dr B did not order any further tests to assess or monitor Mr A's renal function in order to confirm restoration of normal levels following cessation of lithium therapy.

Communication of test results

49. Dr B accepts that he failed to inform Mr A about his renal function test results when he ceased Mr A's lithium treatment in 2007.

50. Dr B did not order blood tests to assess Mr A's renal function until July 2008, the results of which were normal. Dr B ordered repeat renal function tests in November 2009 and February 2010, and the results were abnormal (refer to **Appendix B**).

51. There is no record that Dr B advised Mr A of any of his renal function test results. Mr A stated that he was not aware that he had renal failure until he was admitted to hospital in October 2010 and was diagnosed with chronic kidney disease (CKD). Mr A currently suffers from stage four CKD.[18]

52. Dr B told HDC that while he accepts that his notes do not record that he communicated Mr A's test results to him, it is his usual practice to discuss test results, especially abnormal results, with patients.

Changes made to Dr B's practice

53. Dr B stated that as a result of Mr A's case he has made changes to his practice, including having:

a. implemented routine monitoring of lithium levels and renal function of patients on lithium treatment;

b. extended the amount of time spent doing administration such as reviewing test results and recent consultations, entering data into Medtech and notifying patients;

c. established a system of "red flagging" all abnormal test results that require follow-up;

d. established alerts for patients who require blood tests at their next consultation or when they phone for prescriptions;

[18] There are five stages of kidney function. Kidney function is normal in stage one and minimally reduced in stage two. Kidney function is severely impaired at stage four.

e. arranged with the laboratory to provide a single blood test form for recurrent testing of patients who require regular monitoring;

f. extended his consultation time to allow for review of investigations and plan management while the patient is present;

g. when away on leave, arranged for a colleague to take care of all investigation results and reports, and action those requiring further attention. Dr B advised that he reviews these on his return; and

h. made sure his patients are aware of the potential risks of renal function impairment involved in lithium treatment.

54. Dr B stated that, in retrospect, he recognises that he did not monitor Mr A's lithium treatment adequately. He further stated that he is now aware that abnormal renal function may develop many years after lithium treatment is stopped, so he intends to continue to monitor patients who have previously been on lithium.

55. Dr B also stated that he has informed his colleagues of this complaint and advised them of the changes he has made. He stated that he intends to provide educational sessions for his colleagues on long-term lithium therapy and the risk of renal disease, and that he wishes to implement a practice policy in this regard to prevent any further incidents such as in this case.

56. The medical centre advised HDC that it did not have any written policies with regard to follow-up of test results until it underwent Cornerstone Accreditation in 2006. Prior to 2006 all policies were "unwritten" or "verbal".

57. The medical centre's current policy regarding follow-up and notification of test results places the primary responsibility for ensuring the patient is advised of the test results, on the clinician who orders the test. The policy provides for the recording and tracking of tests. The policy further states:

> "Patients are advised at time of consultation how results will be distributed, usually patients are advised to telephone the practice nurse 1-2 weeks post-test."

58. The medical centre advised that it reviews its policies every three years, but this policy has not changed substantially since 2006. The medical centre also advised that the "Med-Tech system" allows individual practitioners to enter reminders for certain tests and track test results.

Opinion: Dr B

59. Mr A had been prescribed lithium for depression and bipolar affective disorder for approximately six years before transferring to Dr B's care in 2002. Over this period, Mr A appeared to tolerate the therapy well, and the therapy was well established by 2002. Dr B continued to prescribe lithium treatment until 2007 in accordance with specialist advice.

60. Between 2002 and 2010 Dr B failed to monitor effectively for potential side effects of lithium treatment on Mr A, including both lithium toxicity and impaired renal function, and failed to inform Mr A of his blood test results. Mr A was entitled to an appropriate standard of care, which included effective monitoring and follow-up treatment, and being provided with sufficient information in order to make informed decisions with regard to his ongoing care. Consequently, the care provided by Dr B to Mr A was sub-optimal.

Monitoring the effects of lithium — breach

Inconsistent monitoring of renal function and lithium levels

61. Despite Mr A exhibiting ongoing symptoms such as vomiting, weight loss, and dehydration, his clinical records indicate that Dr B did not monitor Mr A's renal function consistently (refer to **Appendix B**) or in accordance with recommended practice. Dr B initially ordered tests for Mr A's renal function approximately four months after taking over his care at the end of 2002. Between December 2002 and July 2005, Dr B monitored Mr A's renal function inconsistently at intervals of between one and eleven months.

62. Dr B also failed to monitor Mr A's blood lithium levels consistently. After Mr A transferred to Dr B's care in 2002, it was over two years before tests were ordered to check Mr A's lithium levels, and these tests were not ordered by Dr B. Further, between 2004 and 2007, Dr B monitored Mr A's lithium levels inconsistently, at intervals of between one and sixteen months.

63. Dr Maplesden advised:

> "While best practice recommendations are for three-monthly testing of both lithium levels and renal function, I would regard 6–12 monthly monitoring in a patient with normally stable results, a stable dose of lithium and normal renal function as probably representing common practice."

Inadequate follow-up of abnormal results

64. Mr A's renal function test results of July 2005 were abnormal. However, Dr B did not order any further tests to monitor Mr A's renal function until December 2005. The results of those tests, which were undertaken in January 2006, as well as tests undertaken in February 2006, were also abnormal.

65. Dr Maplesden further advised:

> "Expected practice would be that the renal function test be repeated shortly after the abnormal result of July 2005 to determine whether the result was a temporary aberration secondary to dehydration or whether it could be a sign of CKD. This was not done."

66. Once Mr A showed consistently abnormal test results with regard to renal function (from July 2005), Dr B should have ordered further blood tests to measure Mr A's lithium levels, in order to ascertain whether Mr A's lithium treatment was affecting his renal function or potentially causing lithium toxicity. However, Mr A's lithium

levels were not tested until eight months later in February 2006, and these tests were ordered by Dr C.

67. In March 2006, on Dr C's advice and due to evidence of impaired renal function, Dr B reduced Mr A's lithium dose. Although follow-up tests for renal function were ordered in April, June and September 2006, both the June and September results were abnormal, and Dr B did not follow up on the abnormal test result of September 2006 by ordering further tests to check for ongoing deterioration in renal function. He did not closely monitor Mr A's lithium levels, which can be elevated in the presence of impaired renal function and lead to toxicity. It was not until approximately eight months later, in May 2007, that Dr E ordered tests for renal function and lithium levels. As a result of those tests, Dr E advised Dr B that Mr A's lithium treatment should be ceased.

68. Following cessation of Mr A's lithium therapy, Dr B should have continued to monitor Mr A's renal function regularly until the results were consistently normal. However, this was not done for over a year.[19] Dr Maplesden advised that, when a follow-up test was eventually done in July 2008, showing that Mr A's renal function had returned to normal, it was reasonable for Dr B to conclude that no further close monitoring of Mr A was necessary.

69. However, Dr B ordered renal function tests again in November 2009 and February 2010, the results of which indicated that Mr A's renal function was impaired. Again, Dr B did not take further action to ascertain the cause of impairment.

70. Dr Maplesden advised that:

> "The concept of irreversible late lithium induced nephrotoxicity…was becoming better recognised at the time of the events in question but I would still not have regarded it as 'common or expected knowledge' outside the realms of psychiatry and renal medicine…However, toxicity…associated with levels outside the recommended range, and the increased risk of elevated lithium levels in the presence of impaired renal function from any cause, has always been well known…"

71. Dr B recorded abnormal test results on several occasions between July 2005 and October 2010. In my view, Dr B failed to follow up Mr A's abnormal test results of July 2005, September 2006, November 2009, February 2010 and October 2010 adequately, by taking steps to ascertain whether Mr A's renal function was being affected by his lithium treatment, or whether his lithium levels were being adversely affected by his impaired renal function.

72. Dr B advised HDC that he was distracted from the cause of the abnormal test results by Mr A's gastric symptoms, which Dr B attempted to address over a prolonged period.

[19] Blood tests in June 2007 related to Mr A's renal function only. Mr A's lithium levels were not measured at this time.

Conclusion

73. I acknowledge that Dr B ordered a number of tests between 2002 and 2010 to monitor Mr A's lithium levels and renal function. In addition, I acknowledge that he took appropriate action in accordance with specialist advice, by reducing Mr A's lithium dosage in 2006, and then discontinuing lithium treatment in 2007.

74. However, as Mr A's GP, Dr B had the responsibility to appropriately manage Mr A's treatment. This included monitoring and responding to any changes in Mr A's condition, whether as a result of any medication or treatment that he was given, or as a result of an illness. In my view, even taking into account Mr A's gastric symptoms, the failure to monitor his treatment adequately between 2002 and 2010, including consistent monitoring of lithium levels and renal function, and the failure to follow up several abnormal test results, means that Dr B failed to provide services with reasonable care and skill and, accordingly, breached Right 4(1) of the Code.

Communication of abnormal test results — breach

75. As Mr A's primary care provider, Dr B was responsible for his day-to-day clinical management. An important aspect of that management was ensuring Mr A was informed of his abnormal test results.

76. As noted above, Dr B recorded abnormal test results for Mr A on 11 occasions between July 2005 and October 2010 (refer to **Appendix B**). While some of those tests were ordered by other doctors, including Dr C and locums at the medical centre, the results were reported to Dr B as Mr A's primary care provider. Mr A stated that Dr B did not inform him of any of those abnormal test results, and he was unaware of any abnormal renal function until he was diagnosed with CKD in October 2010.

77. Dr B stated that it is his usual practice to discuss test results, especially abnormal results, with patients. However, there is no documentation by Dr B of any discussions he had with Mr A regarding the outcome of his test results between 2002 and 2010.

78. Dr B accepts that in 2007 he failed to inform Mr A about his abnormal renal function results. Despite Dr B's comments about his usual practice, I find it more likely than not that Dr B repeatedly failed to inform Mr A of his test results between 2002 and 2010. Accordingly, I find that Dr B breached Right 6(1)(f) of the Code.

79. As a result of Dr B's failure to follow up on Mr A's test results, Mr A was unable to make informed choices about his treatment, particularly regarding his ongoing use of lithium. Accordingly, I find that Dr B breached Right 7(1) of the Code.

Informing Mr A of risks — adverse comment

80. In October 2002 Mr A transferred to Dr B's care. Mr A stated that Dr B did not inform him of the risks of lithium treatment at any time. Dr B stated that when he took over Mr A's care his lithium treatment was "fully established" and that Mr A had shown a good response to the treatment. Dr B advised, reasonably, that it was his understanding that Mr A would have had the risks of the treatment explained to him by the specialists involved in his care, prior to Dr B's involvement. However, there is no evidence that Dr B clarified that with Mr A.

81. Dr B did not initiate Mr A's lithium therapy. Dr Maplesden advised that lithium is generally regarded as a "specialist" drug, usually initiated by a psychiatrist.

82. Dr B took over Mr A's care approximately six years after his lithium treatment began. I accept that, by this time, Mr A's treatment was well established and Mr A had appeared to tolerate the therapy well for six years.

83. The prescribing provider has the responsibility to ensure that the consumer is aware of the risks of the medication the provider is prescribing. However, there are a number of mitigating factors to be considered in this case. As my expert advisor has noted "the responsibility of conveying risks and benefits of [lithium] therapy lies with the prescriber initiating therapy".

84. During the early 2000s the delayed nephrotoxic side effects of lithium were only just gaining clinical recognition. Dr Maplesden advised that he would not have expected a GP to be aware of the requirement to warn the patient of the risk of the development of nephrotoxicity with long-term therapy, unless the risk was "sufficiently great to warrant issuing of a [Medsafe] medication alert". Medsafe did not issue such an alert with regard to lithium until 2010.

85. Furthermore, the lithium-related side effects that Mr A experienced between 2002 and 2010 were uncommon given the relatively short period of time that he had been treated with lithium.

86. I am of the view that prescribing providers, whether or not they are initiating treatment, would be wise to satisfy themselves that the consumer is aware of known material risks associated with the medication the provider is prescribing.[20]

87. The regular monitoring of treatment for a consumer in Mr A's position is intended to manage and respond to, among other things, evolving risks associated with his treatment. It is clear that Dr B was aware of the need to monitor Mr A. Mr A should have been made aware of the ongoing importance of monitoring. By doing so, Dr B would have further empowered Mr A in participating in his care. The provision of full information may well have prompted the consumer to ask directly about the test results himself. In this case, while the risks of nephrotoxicity were not widely known, the implications of impaired renal function were.

88. In light of the circumstances of this case, and the considerations outlined above, I do not consider that Dr B's failure to inform Mr A of the risks of lithium treatment is sufficient to amount to a breach of the Code. However, I do consider that Dr B should be mindful of his obligation to inform patients of the need for regular monitoring when such monitoring is part of recommended clinical practice, and the reasons for such monitoring.

[20] See Opinion 11HDC00440.

Continuing therapy until 2007 — No breach

89. Mr A expressed concerns that Dr B continued to prescribe lithium to him until 2007. I note that Dr B sought reviews from specialists over the time that Mr A was in his care, and continued Mr A's lithium treatment until 2007 pursuant to advice provided.

90. Dr Maplesden stated that it is evident that Mr A's specialists felt that lithium treatment was medically indicated and, in his view, it would have been inappropriate for Dr B to discontinue treatment on his own initiative.

91. I accept Dr Maplesden's advice on this matter and, on that basis, I find that it was appropriate for Dr B to continue treatment with lithium as recommended by the specialists.

Other comment — The medical centre

92. The medical centre did not have any written policies with regard to follow-up of test results until 2006. Prior to 2006 all policies were "unwritten" or "verbal. Dr Maplesden advised that prior to the advent of Cornerstone accreditation in the mid-2000s it would not have been unusual for a practice not to record such policies in detail. He advised that since the advent of Cornerstone accreditation, however, there should be robust written policies.

93. The medical centre's policy since 2006 regarding follow-up of test results states that patients should be advised at the time of consultation how results will be distributed.

94. I acknowledge that the medical centre has implemented a written policy with regard to advising patients of test results, since undergoing Cornerstone accreditation in 2006. The medical centre advised that it undertakes a review of its policies every three years. While the medical centre's current policy regarding follow-up of test results is relatively brief, it clearly leaves the responsibility for ensuring that the patient is informed of test results with the practitioner who orders the test. I consider that this policy is reasonable.

Recommendations

95. I recommend that, within three weeks of the date of the final opinion, Dr B:

- provide a written apology to Mr A for his breaches of the Code, to be sent to HDC for forwarding to Mr A;
- provide HDC with information, including documentation, regarding any educational sessions on long-term lithium therapy and the risk of renal disease that he has provided to his colleagues; and

- provide HDC with a copy of any new medical centre practice policy on providing care to consumers on long-term lithium therapy.

Follow-up actions

96.
- A copy of the final report with details identifying the parties removed, except the expert who advised on this case, will be sent to the Medical Council of New Zealand and the Royal College of General Practitioners, and they will be advised of Dr B's name.
- A copy of the final report with details identifying the parties removed, except the expert who advised on this case, will be sent to the district health board, and it will be advised of Dr B's name.
- A copy of the final report with details identifying the parties removed, except the expert who advised on this case, will be placed on the Health and Disability Commissioner website, www.hdc.org.nz, for educational purposes.

Appendix A — Independent clinical advice to the Commissioner

The following expert advice was obtained from Dr David Maplesden, a general practitioner:

> "1. Thank you for providing this file for advice. I have reviewed the available information: complaint from [Mr A]; response from [Dr B]; extensive GP notes including specialist reports and lab results; ACC documentation including expert reports from [a renal physician] date 31 May and 22 August 2012. [Mr A] complains that [Dr B] failed to inform him of the risks of taking lithium; failed to adequately monitor his lithium therapy including renal function; failed to notify him of abnormal renal function results in 2004 and continued to prescribe lithium until 2007.
>
> 2. [Mr A] has been diagnosed with stage 4 *CKD* [chronic kidney disease] *secondary to lithium nephrotoxicity, idiopathic cardiomyopathy* and *somatic complaints, not yet defined* ([the hospital's]) outpatient letter dated 6 December 2012). [ACC's expert advisor] has provided extensive referenced documentation supporting the notion that [Mr A's] chronic renal disease is secondary to prolonged lithium treatment, a side effect of such treatment identified by some researchers in the late 1980s but well recognized by the medical profession probably only since the turn of the century. I am therefore accepting [Mr A's] renal disease is a direct consequence of his lithium treatment. [Mr A's] providers and [ACC's expert advisor] do not feel his cardiomyopathy is a consequence of the renal disease or lithium treatment as the clinical features are not consistent with lithium-related cardiomyopathy. I am therefore making no further comment regarding this aspect of [Mr A's] complaint (that [Dr B's] prescribing of lithium and failure to monitor led to [Mr A] developing cardiomyopathy and suffering a cerebral ischaemic event). My subsequent advice is confined to the standard of information given to [Mr A] when he was prescribed lithium and when he had abnormal blood test results, the standard of clinical monitoring of his lithium treatment, and the clinical management of his abnormal renal function results.
>
> 3. As a basis for subsequent comments I am using two Best Practice Advocacy Centre (BPAC) publications made available to all GPs at the time — one relating to monitoring of lithium therapy[21] and the other to management of impaired renal function[22]. Relevant points from these publications are reproduced in sections 7 and 8 of this report. These publications represent evidence-based best practice and I recognize there may be somewhat of a gap between best practice and common practice, particularly in regard to lithium monitoring. This has been taken into account when considering any departures from expected practice. Although both publications were first available after [Mr A's] lithium was stopped, I believe the management principles referred to were accepted practice during the period he was under [Dr B's] care. I note there was reference to lithium induced 'nephrogenic diabetes insipidus' in reference 1, but not to nephrotoxicity related to duration of use of the medication (see sections 6 and 7).

[21] BPAC. Lithium in general practice. BPJ. 2007; Issue 3
[22] BPAC. Making a difference in chronic kidney disease. BPJ. 2009; Issue 22

4. Brief summary of relevant 'milestones' from clinical notes

i. 18 June 1996 — [Mr A] seen by psychiatrist [Dr DDr D] on referral from [a] GP. Underlying symptoms of depression and anxiety on a background of chronic dysthymia noted, and a variety of antidepressants trialed at regular review over the next three months, somewhat ineffectively.

ii. 26 September 1996 — [Dr D] reports *I've started him on lithium carbonate 250mg daily, and he's agreed to come and see me again in about three weeks* ie **lithium commenced on this date**. 22 October 1996 — lithium level sub-therapeutic at 0.3 mmol/L but subjective improvement in [Mr A's] mental status so dose unchanged. 16 November 1996 — **lithium increased** to 500mg daily. No documentation of information given to [Mr A] regarding his lithium treatment (risks, side effects, duration, monitoring etc).

iii. [Mr A] saw [Dr D] regularly until July 1998 with further reviews in November 1999 and October 2000. Reference to **increasing dose of lithium** from 500mg daily to 750mg daily in 10 July 1997, and to 500mg bd in October 2000 (see below).

iv. 19 October 2000 — letter of discharge from [Dr D] to [Mr A's] [new GP in another region] noting [Mr A's] past psychiatric history including regular input from [Dr D] since 1996 with a gap from mid-1998 until the end of 1999. Letter includes *we discussed the question of medication, but the most that he would accept for the time being was an* ***increase in the lithium therapy*** *that he's been on for the last four years continuously. I've now lifted the dose of lithium carbonate to 500mg bd, arranging suitable blood tests to make sure that he's still within the therapeutic range ...* GP advised to link [Mr A] with [local] community mental health services, which did occur.

v. Lithium dose recorded in GP notes from 15 September 2000 is 250mg mane, 500mg nocte until 26 July 2002 when the **lithium preparation was changed (and dose marginally increased**) to lithium 400mg tabs one twice daily. Lithium level and renal function monitored relatively closely as per tabulated results (see section 6).

vi. 28 November 2002 — First review by [the DHB's] community mental health services. Baseline bloods ordered with lithium level 0.8 mmol/L and *his liver/kidney functions are normal.* Regular review under the service over next twelve months. No change to lithium dose which is currently recorded as 400mg bd (apparent the increase advised by [Dr D] (see above) was never implemented).

vii. 31 October 2002 — GP care transferred to [Dr B] with first consultation on this date. [Previous GP's] notes indicate transfer of old notes to [Dr B's] surgery on 20 March 2003.

viii. 10–13 June 2003 — admission to [the DHB's] inpatient mental health services following exacerbation of depressive symptoms with agitation. No mention of abnormal blood tests. Lithium dose unchanged.

ix. 5 November 2003 — formal discharge of [Mr A] from [the DHB's] community mental health services back to [Dr B's] care, with final DHB psychiatrist review scheduled for three months hence.

x. 26 October 2004 — GP consultation [Dr F]. History of dizziness, parasthesiae and vomiting. Blood tests, including serum lithium, ordered. Mild elevation in serum creatinine for the first time on record. Vomiting symptoms persisted thereafter (see below).

xi. 22 February 2005 — letter from [the DHB] declining [Dr B's] referral of [Mr A] for gastroenterology review (referral sent 6 January 2005) for symptoms of persistent vomiting and rectal bleeding.

xii. 29 July 2005 — GP consultation [Dr B] — *vomiting ++, pre-syncopal, dehydrating with ↑ creat of 0.15. Needs admission for Ix and Rx if persists.*

xiii. 15 February 2006 — review by gastroenterologist [Dr C] following referral from [Dr B] (18 January 2006) with a one year history of persistent vomiting and weight loss. *There is polydipsia and he drinks in excess of 3 litres of water a day … the polydipsia in conjunction with being on lithium raises the possibility of possible nephrogenic diabetes insipidus. I have therefore checked his serum osmolality and sport urine for electrolyte values.* Empirical trial of Somac commenced.

xiv. 6 March 2006 — letter from [Dr C] to [Dr B] regarding [Mr A's] blood test results: *He had a raised serum creatinine of 0.15 when this was done on 17th February. Normal serum sodium and potassium. His serum lithium level was 1.2 mmol/L and outside the normal range … in summary then there is evidence of mild renal impairment. It would probably be appropriate to reduce his lithium dose given the renal impairment and raised serum level … I suggest that you repeat the above investigations after you have reduced his lithium … after a certain period of time. I would appreciate copies to myself.* **Lithium reduced about this time to 400mg mane.**

xv. 21 June 2006 — assessment in the DHB medical outpatient clinic with history of weight loss, anorexia and vomiting. Normal examination and response noted to recent commencement of Somac, but perhaps worsened by quetiapine. *GP to follow up regarding CBC, U&Es, LFTs, ESR and CRP*. Psychiatrist review of medication recommended. No other follow-up arranged.

xvi. 10 August 2006 — letter from [Dr C] to [Dr B] following review of [Mr A]. GI symptoms including vomiting are persisting but improved alertness following reduction in lithium and quetiapine doses. Examination normal. Further blood tests organized and reduction in Epilim advised. Letter to [Mr A] (cc [Dr B]) dated 17 August 2006 noted reduction in lithium levels to 0.8 mmol/L and *the liver function tests* [should be kidney function] *still look mildly impaired with a serum creatinine of 150 though this is not really high.*

xvii. 10 January 2007 — [Mr A] referred by [Dr B] to psychiatrist [Dr E] for medication review given ongoing GI symptoms perhaps related to current medications. [Mr A] was subsequently seen by [Dr E] on several occasions until at least May 2008 (last letter on file from him).

xviii. 30 May 2007 — **Lithium stopped** by [Dr E]**.** Letter from [Dr E] includes *the blood test had returned with a raised creatinine level and I suggested to him that we discontinue his 400mg of lithium all together. It was unclear whether the raised creatinine level could be due to dehydration from vomiting or whether it was due to his use of lithium … I also gave him a further lab test form to check his creatinine in a week's time* (no result of this test on file if it was done).

xix. 7–20 June 2007 — admission to the DHB inpatient mental health services following exacerbation of anxiety with marked weight loss and vomiting. No mention of abnormal blood tests in discharge summary or subsequent outpatient letter dated 29 June 2007.

xx. 29 January 2008 — formal discharge from [the DHB's] Mental health & Addiction Services. Ongoing regular review by [Dr E]. 5 May 2008 — letter from [Dr E] to [Dr B]: diagnosis *Generalised anxiety disorder ?bipolar disorder in remission.*

xxi. 26 July 2010 — Sigmoidoscopy performed (normal) following referral for rectal bleeding and chronic constipation.

xxii. 10–27 October 2010 — inpatient admission [the hospital] following referral from GP with history of increasing shortness of breath, hypertension and visual symptoms. Diagnosed with idiopathic cardiomyopathy, acute ischaemic cerebral event (vision loss) likely related to the cardiomyopathy, chronic renal impairment and hypertension. Fully investigated and discharged on multiple medications with renal and cardiology follow-up.

xxiii. 3 December 2010 — letter from the DHB [nephrologist] to [Dr B] includes: *I have gone back extensively and looked at laboratory data dating back to 2003 on this gentleman. He has had chronic kidney disease dating back to at least 2007 when serum creatinine was in the 165 µmol/L range.* Ultrasound has shown bilateral small scarred kidneys

and urinalysis showed mild proteinuria only. *This is very consistent with his previous longstanding lithium use and I do not feel it is related to his newly diagnosed cardiomyopathy.*

xxiv. 5 May 2011 — nephrologist letter includes results of renal biopsy performed 25 March 2011: *This came back showing significant fibrosis and previous interstitial scarring and could well be most consistent with his previous lithium use* …. Ongoing management is through [the DHB's] renal and cardiology services.

5. Sequential blood results (only results relevant to the complaint are recorded) and associated comments. Results available from 1 August 2000 so level of monitoring prior to this cannot be confirmed:

Date	**S Lithium[23]**	**S Creatinine/eGFR[24]**	**Comment**
1 Aug 00	0.8	0.087mmol/L	[GP]
19 Oct 00	0.8		[GP]
6 Nov 00	1.0		[GP]
1 Jun 01	1.0	0.09	[GP]
9 Oct 01	1.0		[GP]
12 Aug 02	0.9	0.075	[GP]
5 Dec 02			Bloods ordered by [Dr B]. No lithium/renal function
28 Jan 03			Bloods ordered by [Dr B]. No lithium/renal function
23 Dec 03		0.10	[Dr B]
5 Jan 04			Bloods ordered by [Dr B]. No lithium/renal function
9 March 04			Bloods ordered by [Dr B]. No lithium/renal function
26 Oct 04	0.9	0.12	Ordered by [Dr F] — [Mr A] complaining of dizziness and vomiting
6 Jan 05			Bloods ordered by [Dr B]. No lithium/renal function [Mr A] still complaining of symptoms above

[23] Therapeutic range 0.5–1.0 mmol/L

[24] Normal range creatinine 0.05–0.12 mmol/L (units changed to µmol/L mid-2006: normal range 60–105 µmol/L). Normal range estimated glomerular filtration rate (eGFR) >60 ml/min./1.73m^2 with pathologist comment on results that results between 30 and 60 *may require further investigation.*

19 July 05		0.14/57	[Dr B]
4 Jan 06		0.15/53	[Dr B]
17 Feb 06	1.2	0.15	Referred to in letter from [Dr C]. Lithium reduced and [Dr B] advised to arrange follow-up tests
12 April 06	0.7		[Dr B] — no renal function on record
22 June 06		154 µmol/L/51	Ordered by [Dr G] at medical OPC appointment, [Dr B] advised to follow-up.
21 Sept 06		163/47	[Dr B]. Note deterioration in renal function.
19 May 07	0.7	165/46	Ordered by psychiatrist [Dr E]
22 May 07		182/41	[Dr E]. **Lithium stopped.**
29 July 08	No further levels reqd.	101/>60	[Dr B]. Renal function now normal although [Mr A's] GI symptoms persisting
5 Nov 09		145/53	[Dr B]
16 Feb 10		143/54	[Dr B] (no indication abnormal renal function was discussed with [Mr A] on either occasion)
8 Oct 10		206/33	[Dr I]. [Mr A] referred to hospital shortly after.
26 Jan 11		196/35	Selection of results follow to represent levels post-cardiomyopathy therapy
22 March 11		179/39	
2 Feb 12		170/41	
7 Jan 13		159/44	

6. Comments

(i) [Dr B] acknowledges in his response that his monitoring of [Mr A's] lithium levels and renal function was suboptimal, and he has since changed his practice in this regard to better represent best practice guidelines. He notes he was distracted from the likely cause of the deterioration in [Mr A's] renal function (lithium related nephrotoxicity) because of [Mr A's] GI problems, particularly persistent vomiting, that might have caused chronic dehydration and secondary renal impairment. [Dr B] involved specialists in the investigation and management of

[Mr A's] GI symptoms, and sought specialist advice in psychiatric medication management throughout his period of care of [Mr A]. [Dr B] notes also that it was somewhat unusual for [Mr A] to have developed lithium related nephrotoxicity within ten years of commencing treatment although, as noted by [ACC's expert] in his ACC report, this situation may occur. [Dr B] was somewhat reassured when [Mr A's] renal function returned to normal following cessation of his lithium treatment, but was unaware that effects of lithium related nephrotoxicity can become apparent some time after stopping the treatment. He acknowledges that, in hindsight, he should have referred [Mr A] for specialist review when his renal function deteriorated again from November 2009, and has since changed his practice to ensure such referral does occur.

(ii) [Mr A] complains that he was not adequately informed of the risks of lithium therapy, and that it may have been prescribed inappropriately. I feel the responsibility of conveying risks and benefits of therapy lies with the prescriber initiating therapy (in this case [Dr D]) and I cannot determine from the available notes the level of information given to [Mr A]. I note [the ACC expert's] comments that it might have been reasonable not to have discussed the risk of lithium related nephrotoxicity in 1996 as the evidence of an association between the medication and the condition had not been established unequivocally at this time. Lithium therapy is normally initiated by, or in conjunction with, a specialist psychiatrist and I would not therefore expect a GP to be aware of a change in risk profile (requirement of warning regarding risk of long-term therapy and development of nephrotoxicity) unless the risk was sufficiently great to warrant issuing of a medication alert. In 2010 Medsafe did issue a prescriber update[25] (available to GPs but such publications not necessarily highlighted or prioritized) that included the following information:

Prescribers are reminded that long-term lithium therapy can cause renal failure along with other metabolic adverse effects including hypothyroidism, weight gain, and hyperparathyroidism. Renal function, including glomerular filtration rate (GFR) should be measured regularly even after 10–15 years of therapy. If renal impairment develops, advice from a nephrologist and/or psychiatrist should be sought as discontinuing lithium therapy may not [be] possible for all patients. Lithium is associated with a number of renal adverse effects including nephrogenic diabetes insipidus (which affects up to 40% of patients), chronic kidney disease (CKD) and renal failure … The Centre for Adverse Reactions Monitoring (CARM) has received a total of nine reports of renal failure in association with lithium use. Importantly, six of the reports were received in the last two years. The mean age of patients was 53 years (range 36–77 years) and the average duration of lithium therapy prior to development of CRF was 28 years (range 14–38 years). In at least one case, renal function continued to deteriorate despite lithium being discontinued.

In some ways this publication may have been reassuring regarding the average duration of therapy prior to development of nephrotoxicity. Nevertheless, the publication did emphasize the importance of regular monitoring and seeking

[25] Medsafe. Renal dangers associated with long-term lithium use. *Prescriber Update 2010;31(3):20*

specialist advice should renal impairment be noted. In summary, I feel [Dr B's] management of [Mr A] with respect to information regarding risks of lithium therapy was consistent with expected standards given he did not initiate the therapy and took over [Mr A's] care some six years after therapy had been commenced.

(iii) The issue of whether lithium was an appropriate treatment for [Mr A] is outside the scope of this report. The medication was initiated by a specialist psychiatrist in 1996 and not stopped until May 2007 (and then because of [Mr A's] impaired renal function) in spite of numerous reviews by a variety of specialist psychiatric providers over that period. It is evident these providers felt the medication was clinically indicated and it would have been inappropriate for [Dr B] to have stopped the medication without specialist advice or input.

(iv) Monitoring of patients on lithium therapy (see section 7): Records from 1 August 2000 to 12 August 2002 indicate [Mr A's] lithium level was monitored regularly with time between tests ranging from one month to a maximum of ten months (six levels performed in the two year period). Renal function was checked on three occasions over the two year period (all results normal) with the maximum time between tests being 14 months. While best practice recommendations are for three-monthly testing of both lithium levels and renal function, I would regard 6–12 monthly monitoring in a patient with normal stable results, a stable dose of lithium and normal renal function as probably representing common practice. [Monitoring from 2000–2002] was essentially consistent with accepted practice at the time. However, following transfer of care to [Dr B] it was over two years before [Mr A] had a lithium level performed (ordered by [Dr F] — result normal) then another 16 months before the next test (ordered by [Dr C] — mildly elevated) in spite of [Mr A] suffering ongoing vomiting and other symptoms — some of which could be related to lithium toxicity, or consequences of which (eg dehydration) could lead to lithium toxicity over this period. When renal function tests were noted to be abnormal in July 2005, lithium levels should have been determined and more closely monitored, yet this was not done for a further seven months (in spite of persisting renal function abnormalities) and then it was initiated by [Dr C]. Following a reduction in lithium dose the test was repeated by [Dr B] at an appropriate time interval (result normal) and then rechecked 13 months later by [Dr E]. In my opinion, the failure by [Dr B] to ensure [Mr A's] lithium level was monitored at least annually and more frequently when he developed ongoing vomiting symptoms and impaired renal function, was a moderate departure from expected standards.

(v) Monitoring and management of renal function:

One of the reasons for regular monitoring of renal function in patients on lithium treatment is because of the potential for nephrotoxicity from the medication, and the need to adjust the dose (or stop therapy) in patients with renal impairment from unrelated causes. On taking over [Mr A's] care, [Dr B] first rechecked renal function in December 2003 — about 10 months after the previous test. Both were normal. The next renal function result was dated October 2004 (ordered by [Dr F]

when [Mr A] was unwell) and was at the upper limit of normal at 0.12 mmol/L. Despite [Mr A] remaining unwell and on lithium, the next renal function test was not undertaken until July 2005. This was abnormal at 0.14mmol/L with reduction in eGFR at 57. [Dr B] noted the abnormal results in the clinical record, and [Mr A's] ongoing GI symptoms, with an intention to consider hospital admission if the symptoms and biochemical abnormalities persisted (see 4 (xii)). [Mr A] states he was not informed of the abnormal test at this stage or at any other stage before his CKD was diagnosed in late 2010. [Dr B] did not repeat a renal function test for a further six months (January 2006) and this result was similar to the previous one. Expected practice would be that the renal function test be repeated shortly after the abnormal result of July 2005 to determine whether the result was a temporary aberration secondary to dehydration or whether it could be a sign of CKD. This was not done. Once persistent decrease in eGFR and elevation in serum creatinine was confirmed, management as discussed in section 8 should have been undertaken, with particular consideration of lithium as a potential nephrotoxic agent. Certainly, close monitoring of renal function (at least three-monthly) should have followed. In fact [Mr A] did have three-monthly (approximately) testing of renal function between January and September 2006 although two of the three tests were ordered by specialists ([Dr C] and [Dr G]), and all tests from July 2005 to June 2006 were consistent with stage 3 CKD but remained relatively stable. [Dr C] informed [Mr A] of his abnormal results but referred in error to liver impairment (see section 4(xvi)), and did not indicate to [Dr B] that any particular action, other than monitoring of renal function, was required. However, [Dr C] had initiated a reduction in lithium dose, presumably recognizing the potential for nephrotoxicity as well as the serum level being outside the therapeutic range. [Dr B] did organize follow-up bloods following receipt of the results from medical outpatients ([Dr G] — June 2006), and these results (September 2006) showed a further deterioration in renal function. This was not acknowledged in the clinical notes and there was no further follow-up for eight months until psychiatrist [Dr E] organized bloods (May 2007), recognized possible lithium related nephrotoxicity and stopped [Mr A's] lithium. A follow-up test was supposed to have been undertaken shortly after cessation of lithium (see 4 (xviii)) as would be expected but for some reason was never done. It is not clear whether [Mr A] had blood tests done during his inpatient psychiatric admission of 7–20 June 2007 (no results on GP file) and [Dr B] did not review [Mr A's] renal function until July 2008, some 14 months after the previous significantly abnormal result. It may be there was some confusion over where the responsibility for ongoing testing lay, or an assumption testing was done during the inpatient admission and was normal. Nevertheless, it is somewhat disturbing to see an abnormal result not followed up for over a year. Thankfully the result of July 2008 indicated [Mr A's] renal function had returned to normal, and I think it was reasonable for [Dr B] to assume at this stage that no further close monitoring was required given [Mr A] was no longer taking a potentially nephrotoxic medication and had normal renal function (although the potential for lithium to cause renal impairment even years after cessation was not recognized). However, [Dr B] did repeat renal function tests in November 2009 and February 2010 with impairment noted to a level similar to results of 2005-mid 2006. It is not apparent he took any further action on these results nor discuss them with [Mr A]. Further monitoring was not

organized and in October 2010 marked deterioration in renal function was noted on a test ordered by [Dr I] as part of assessment of a recent deterioration in [Mr A's] general physical condition. This resulted in hospital admission and diagnoses as outlined in section 4(xxii–xxiv).

(vi) There may be some mitigating factors in this case including the distraction of [Mr A's] persisting upper GI symptoms which [Dr B] was attempting to address over a prolonged period, a possible lack of specific advice given by [Dr C] and [Dr E] to [Dr B] regarding appropriate further management of [Mr A's] identified renal abnormalities and their association with lithium in 2006 and 2007 and the apparent return of normal renal function in 2008. However, even taking these factors into account I think it is apparent [Dr B's] monitoring, investigation and management of [Mr A's] impaired renal function fell well short of expected practice particularly given his concurrent lithium therapy until May 2007, and his young age (born in 1980). Taking into account the factors discussed above, including the apparent failure by [Dr B] to inform [Mr A] of his impaired renal function at any stage (per the complaint), I feel [Dr B's] management of [Mr A's] impaired renal function departed from expected standards to at least a moderate degree. However, I cannot state that earlier intervention, including earlier cessation of lithium, would necessarily have prevented the nephrotoxicity experienced by [Mr A] given the nature of his condition.

7. From the BPAC publication *Lithium in general practice. BPJ. 2007; Issue 3:*

(i) *Recommended baseline tests and ongoing monitoring are described in Table 2* (reproduced below). *There may be slight local variations in these guidelines. As well as biochemical monitoring it is important to look for and educate patients about physical signs and symptoms associated with adverse effects and toxicity. These include tremor, tiredness, lethargy, nausea, vomiting and diarrhoea, dehydration, polydipsia, polyuria and nocturia. Although baseline tests will be carried out when lithium is initiated by a specialist, check that the results are complete and readily available for reference.*

Lithium baseline tests and monitoring.[6,9] (adapted from Livingstone, 2006; Waitemata DHB 2006)

	Baseline	Routine maintenance	Comments
Serum lithium concentrations	Important to establish reliable steady state concentration associated with therapeutic response	3-monthly	Monitor more frequently in high risk patients, e.g. those on potentially interacting drugs, poor compliance, elderly, unstable renal function, physical illness
Thyroid Function	Baseline thyroid function (T4, TSH)	TSH 3 months after initiation and then 6-monthly	T4 not routinely required. Monitor for symptoms of hypothyroidism
Electrolytes	Baseline	Check with lithium serum levels every 3 months	Particularly important to monitor sodium as it competes for reabsorption in proximal renal tubule
Serum creatinine and renal function	Exclude renal disease. Baseline creatinine and estimation of renal function	Check at same time as lithium levels, at least every 3 months	Estimate renal function using the Cockcroft and Gault Equation* based on ideal body weight
Serum calcium and magnesium	Baseline	Check every 2 years	Lithium may rarely cause hypercalcaemia and hypermagnesium
Parathyroid Hormone (PTH)			Measure only if serum calcium is elevated. PTH must be interpreted relative to serum calcium measurement on the same specimen
Weight	Baseline weight	Monthly, reduce frequency after 6–12 months if weight is stable	Encourage self-monitoring and weight control measures
Pregnancy Test	Baseline in women of childbearing age		
ECG	Baseline in patients with cardiac problems or aged over 45 years	12-monthly in patients with cardiac problems or aged over 45 years	Conduct more frequently if clinically indicated

*The bpac creatine clearance calculator is based on the Cockroft-Gault equation

(ii) *Adverse effects are usually related to serum lithium concentrations and are infrequent at levels below 1.0 mmol/L. Mild gastrointestinal effects (mild nausea, vomiting and diarrhoea), vertigo, muscle weakness and a dazed feeling may occur initially, but frequently disappear after stabilisation. Fine hand tremors, polyuria and polydipsia (mild thirst) may persist. Mild polyuria may not be of concern but may be troublesome and the possibility of diabetes insipidus should be considered. Skin conditions including acne, psoriasis, generalised pustular psoriasis, rashes and leg ulcers can be aggravated by lithium treatment. Lithium has several less common but important metabolic adverse effects. Prevention and avoidance of risk factors are important keys to management (Table 1). Patients and their families/carers should be educated about early warning signs of all adverse effects, and the need for immediate advice if clinical signs of lithium toxicity such as severe or persistent diarrhoea, vomiting, tremor, mild ataxia, drowsiness or muscular weakness occur.*

(iii) *Nephrogenic diabetes insipidus: Lithium is the most common drug cause, affecting 10% of patients treated for 15 years or more. Risk correlates with duration of lithium treatment.*

Presents as polydipsia and polyuria (24 hour urine volume > 3 L). Dehydration, lithium intoxication and deteriorating renal function may occur and renal impairment may be permanent. Risk factors include long term treatment, concurrent use of long term NSAIDs, chronic physical illness and increasing age. Avoidance includes careful monitoring and awareness of risk factors. Management may include shared care with renal specialist and switch to alternative treatment.

(iv) *There is debate in the literature about how long mood stabiliser treatment should be continued and various criteria have been proposed. Local consensus is to continue treatment for at least six months after a first manic episode. The criteria for long-term maintenance treatment varies but commonly includes at least two episodes of mania or depression … Discussion about discontinuing lithium treatment will usually be done in consultation with a specialist. Indications for discontinuing treatment include:*

- *Lack of response, given an adequate dose for an adequate time period.*
- *Renal failure or worsening renal insufficiency.*
- *Cardiac insufficiency.*
- *Ongoing poor compliance with medication (where interventions to improve compliance have been ineffective).*
- *Intolerable adverse effects to lithium.*
- *Remission of bipolar disorder for an adequate period of time in liaison with specialist*

8. From the BPAC publication *Making a difference in chronic kidney disease. BPJ. 2009; Issue 22:*

(i) The following recommendations were made with respect to actions based on eGFR results (table below). As noted in the tabulated results (section 6) eGFR was reported specifically if <60, or as >60, until about 2008/9. The recommendations noted below are based on later processes (see footnote at base of table and also the pathologist recommendations noted in footnote 4, section5).

eGFR	Action
>90	'No further action' unless known, or suspected, structural or urinalysis abnormalities.
60–89	Urinalysis required to check for evidence of kidney disease. If negative and no other markers of renal disease 'no further action' required. If positive determine CKD stage.
<60	Exclude acute renal failure. Determine CKD stage. Urinalysis required to check for haematuria/proteinuria.

N.B. Most laboratories report the eGFR either as >90 mL/min/1.73m^2 or, if less

than this, as an exact figure. Some laboratories only give exact figures if the result is less than 60.

(ii) Local CKD management guidelines[26] define Stage 3 chronic kidney disease CKD as eGFR 30–59 with management advice including:

• *monitor eGFR 3 monthly*

• *avoid nephrotoxic drugs*

• *prescribe antiproteinuric drugs (ACE inhibitors and/or ARBs) if appropriate*

• *address common complications*

• *ensure drug dosages appropriate for level of kidney function*

• *consider indications for referral to a nephrologist*

9. It might be reasonable to consider an educational approach as part of resolution of this complaint. If this is agreed I recommend the following:

(i) [Dr B] review the references quoted in this report and provide a CME session to his peers on the topics of expected practice in management of patients on long-term lithium therapy and identification and management of chronic kidney disease in general practice

(ii) [Dr B] undertake a two pass audit of patients in his practice on long-term lithium therapy to determine as baseline the number of patients being monitored in accordance with relevant guidelines, what process improvements need to be undertaken in light of the first pass results, and demonstration of process improvements and adherence to guidelines in the second pass results."

Subsequent independent clinical advice to the Commissioner

The following further expert advice was obtained from Dr David Maplesden, dated 14 October 2013:

"I have reviewed [Dr B's] response dated 17 June 2013 in which he comments on my original advice.

1. [Dr B] reiterates the complex nature of [Mr A's] medical issues which tended to somewhat obscure any direct link between his lithium use and his deterioration in renal function. His co-morbidities, and the need for involvement of other specialist providers in his care, did mean the pathways of responsibility for ordering and follow-up of blood tests was not always clearly defined. This has been regard[ed] as a mitigating factor in my original advice but as [Dr B] states, the very nature of [Mr A's] condition meant it was important there was one person coordinating his

[26] Kidney Health NZ. Chronic Kidney Disease (CKD) Management in General Practice. 2009. Available at: http://www.kidneys.co.nz/Resources/Health-Professionals/ Accessed 7 May 2013

care including ensuring there was adequate monitoring of his lithium levels and renal function, and that role was [Dr B's] responsibility.

2. [Dr B] queries whether the lithium monitoring guidelines quoted in my original advice represented expected practice for the time of much of [Mr A's] care. I accept the guidelines quoted represent evidence-based best practice but a study of earlier documentation (see below) suggests the current recommendations have not changed substantially this century. From personal experience as a locum in many practices around the country, I am aware there is likely to be a significant gap between common practice and expected practice in regard to lithium monitoring although I am not aware of any local studies confirming this. Nevertheless, the situation with [Mr A] was somewhat different to a patient who was physically well with no intercurrent illnesses and whose lithium levels had always been stable, in whom drug level monitoring less frequently than recommended might have been acceptable. [Mr A] had physical problems which could disturb renal function (which in turn could affect lithium levels). He then showed abnormal renal function and had exhibited lithium levels outside the therapeutic range. These factors made close monitoring of his lithium levels, as per guideline recommendations, an important issue.

3. A 2002 journal article[27] emphasizing the importance of adherence to lithium monitoring guidelines included the following pertinent comments: *Despite a steady decline in the use of lithium over the past decade, lithium treatment remains a major cause of negligence claims in the care of patients with psychiatric disorders. Lack of regular lithium monitoring, the use of interacting medications and failures in communication are the major problems … Monitoring patients on lithium is usually not a complex technical task and the broad parameters of how it should be conducted have been established for many years. What, however, has sometimes been lacking is a systematic means of ensuring that patients are safely, consistently and efficiently monitored in the setting most appropriate to their needs. Many clinicians and geographical areas have local systems that promote good practice, but unfortunately few have effectively bridged the communication gap that can occur between a patient's psychiatrist and the general practice that provides most patient prescriptions and medical care. Differences in individual psychiatrists' approaches may also generate confusion for GPs. Such factors perpetuate the liability for ongoing problems and litigation.*

4. [Dr B] describes his practice policy for handling of test results. This policy and process is consistent with expected practice.

5. [Dr B's] response shows he has quite appropriately treated this incident as a serious event and has made several relevant improvements to his practice. He plans to provide further education to his peers on current recommendations regarding lithium monitoring and side effects. He has accepted responsibility for inadequate monitoring of [Mr A's] condition and therapy, and has done so in a constructive and professional manner. While there is no additional information

[27] Nicholson J et Fitzmaurice B. Monitoring patients on lithium — a good practice guideline. The Psychiatrist (2002)26: 348–351

> contained in the response that alters the overall content of my original advice, I am confident that the approach [Dr B] has taken will result in an improvement in monitoring and care of patients taking lithium (and other medications requiring regular monitoring) within his practice."

The following Questions were also put to Dr Maplesden:

1. Dr B became a fellow of the Accident and Medical Practitioners Association in 2001. He did not become a fellow of RNZCGP until 2012. Do you have any concerns regarding his scope of practice when providing care to Mr A?

Dr Maplesden advised that this is acceptable, provided Dr B was in a collegial relationship. He did not have any concerns.

2. Your advice has focussed on the care provided by Dr B. Do you have any comments on the care provided by the medical centre?

Dr Maplesden said that he considered the care here to be individual clinical decisions by the doctor, not a systems issue.

3. Is there any other information you would consider useful if you were to give further advice on this case?

Dr Maplesden said that while the records from before 2000 are missing, these aren't highly relevant as blood tests for lithium were normal after 2000. He felt that the complainant's concern that he should not have been prescribed lithium in the first place would be difficult to establish, as different psychiatrists have different opinions on it.

4. In your advice you stated at paragraph ii) "I feel the responsibility of conveying risks and benefits of therapy lies with the prescriber initiating therapy … and I cannot determine from the available notes the level of information given to [Mr A] ..."

[You have previously advised that it is a provider's responsibility to satisfy themselves that a consumer is aware of the risks of a medication and that it is a suitable medication for that consumer, rather than relying on the fact that other providers had previously prescribed the medication for the consumer.]

Could you please advise what factors set these cases apart?

Dr Maplesden stated:

> "[Dr B] took over [Mr A's] care many years after the lithium had been initiated, and [Mr A] had seen a variety of health providers in the interim. Lithium therapy is rarely initiated by a GP and is generally regarded as a 'specialist' drug, usually initiated by a psychiatrist. However, given GPs often provide ongoing prescriptions, they should have a working knowledge of the common adverse effects of the drug and requirement for monitoring serum levels. The main differences between this case and the case you quoted are: the nature of the medication (specialist drug initiated by psychiatrist versus drug most commonly

initiated in primary care); the length of time since initiation and apparent tolerance of the drug over this period; the relatively uncommon nature of the side effect experienced by [Mr A] — that side effect gaining increasing clinical recognition (within the medical fraternity) in the period after he had commenced the drug; in the other case, the GP was re-initiating a therapy the patient had not taken for a couple of years, and was doing so in the face of recognised risk factors which may alter with time (patient age, smoking status, weight) associated with that medication. In this case [Dr B] was merely maintaining therapy [Mr A] had apparently taken for many years without problems, and that had been effective for him."

5. You also stated at paragraph ii) of your advice on this case that "Lithium therapy is normally initiated by, or in conjunction with, a specialist psychiatrist and I would not therefore expect a GP to be aware of a change in risk profile …". In light of this, who would you expect to hold responsibility for determining dosage/whether to continue lithium therapy etc?

Dr Maplesden stated:

"Given [Mr A] had not seen a psychiatrist for some time, I would expect the GP to maintain the therapy previously prescribed provided the patient remained well (mentally and physically), tolerated the drug and had drug levels monitored as per standard recommendations. If drug levels became too high, I would expect a GP to be capable of prescribing a reduced dose of the medication and again to monitor the outcome. If control of the patient's psychiatric illness became suboptimal for any reason, I would generally expect a psychiatrist to be involved with treatment adjustments (with respect to bipolar disorder) — verbal advice might be sufficient. Similarly, if the patient had to stop their mood stabiliser because of side effects, I would expect psychiatric consultation to be part of the subsequent management plan."

6. Would your advice differ if [Mr A] did not have a psychiatrist involved in his care during a particular period?

Dr Maplesden stated:

"If [Mr A] had been seeing a psychiatrist regularly at the time of the events in question, the only differences in my advice would be that recognition of lithium nephrotoxicity might have been expected somewhat earlier given the increased familiarity psychiatrists have with the drug, and more regular monitoring of drug and renal function levels might have been expected — either initiated by the specialist or undertaken by the GP on specialist advice and with results going to both parties."

7. Dr Maplesden further noted:

"The concept of irreversible late lithium induced nephrotoxicity (independent of serum level of the drug) was becoming better recognised at the time of the events in question but I would still not have regarded it as 'common or expected knowledge' outside the realms of psychiatry and renal medicine … However, toxicity (including nephrotoxicity) associated with levels outside the

> recommended range, and the increased risk of elevated lithium levels in the presence of impaired renal function, from any cause, has always been well known, hence [Dr C's] recommendation to reduce [Mr A's] dose and review serum levels when he notes serum level above the recommended range."

8. Can you please advise whether you would expect [the medical centre], to have had any policies in place (between 2002 and 2010) with regard to:

a) Monitoring of patients and follow-up of test results; and/or
b) Informing patients of test results.

The medical centre has provided a copy of their policies in this regard dated 2012. The medical centre advises that these policies have been implemented since 2006, but that they have not changed materially since then.

I would also appreciate your comment on the adequacy or otherwise of these policies. Dr Maplesden stated:

> "I would expect the practice to have had policies in place for follow up of test results and informing patients of test results. Prior to the advent of Cornerstone accreditation (mid-2000s) these policies might not have been recorded in detail but certainly since that time there should be robust written policies. The policy you attached is less detailed than some I have reviewed but is adequate and gives the individual practitioner some leeway over how results are dealt with (most likely depending on clinical urgency). I would not expect written policies on monitoring of patients other than for formal national screening programmes such as cervical smears. However, some practices may develop such policies if they have nurses managing specific recall areas (eg INR blood tests) or in response to an identified need (eg practice audit shows significant variation in practice (and from best practice) between partners for monitoring of patients on lithium)."

Appendix B — Summary of care for Mr A between 1996–2010, regarding monitoring of lithium and creatinine levels[28]

Date	Dr B	Specialists /other providers	Lithium/ Creatinine/ Results
1996–2000		Psychiatrist Dr D	
2000–2002		GP (another region)	
12/08/2002		GP (another region)	lithium 0.9, creatine 0.075
11/10/2002	Transferred into Dr B's care		
31/10/2002	Consult with Dr B "Bipolar disorder, on Risperidone, Epilim…"		
19/11/2002	Consult with Dr B "Bipolar disorder well controlled — advice."		
28/11/2002 –2003		Mr A engaged with Community Mental Health.	
5/12/2002	Consult with Dr B re swollen, non tender left ankle. Lab referral for renal function.		
15/01/2003	Consult with Dr B		
28/01/2003	Consult with Dr B. Lab referral, hormones, testosterone, thyroid.		
17/03/2003	Consult with Dr B re application for invalids		

[28] Normal lithium levels (0.4–1.0 mmol/L) Possible toxicity (1.0–1.5 mmol/L) Toxic (>1.5 mmol/L). Normal creatinine levels (0.060–0.120 mmol/L) or (50–120 µmol/L) Note, units changed from mmol/L to µmol/L in mid 2006.

	benefit.		
18/03/2003	Consult with Dr B re ability to work.		
10–13 June 2003		Admission to the DHB inpatient mental health services	
17/06/2003		Consult with unnamed Dr, medical centre, "constipated and now bleeding pile…no change appetite/weight…"	
23/12/2003	Consult with Dr B "++ swollen feet; eats 1 x per day; light headed; N with food..." Lab referral — Liver tests, Renal — Urea, Renal — Creatinine.		creatinine 0.10 mmol/L
24/12/2003	Consult with Dr B. Lab referral "Thyroid — T4 Thyroid…"		
5/01/2004	Blood tests ordered by Dr B		
9/01/2004	Consult with Dr B. Lab referral "Thyroid — T4 Thyroid…"		
27/01/2004	Consult with Dr B re off work certificate.		
9/03/2004	Blood tests ordered by Dr B		
16/03/2004		Consult with unnamed Dr at the medical centre re insect bite on left	

		shin.	
17 and 18/03/2004	Consults with Dr B re cellulitis of left calf.		
27/08/2004	Consult with Dr B, poor sleep patterns, fatigue…constipation. 75kg."		
26/10/2004		GP consultation with Dr F, medical centre. "Feels dizzy, pins and needles and vomiting, gets shaky, headaches…suggest do bloods…Suggest decrease quetiapine to 50 BD priadel the same, epilim to 1 BD" Lab referral. Mild elevation in serum creatinine for the first time recorded.	lithium 0.9 mmol/L, creatinine 0.12 mmol/L Creatinine on the high end of normal
10/11/2004	Consult with Dr B "Review of Sx — note made of 6 Epilim."		
6/01/2005	Blood tests ordered by Dr B. Mr A complaining of Dizziness, "faint feeling, Vomiting ++".		
3/02/2005	Consult with Dr B. "Constipation +++ and now also V ++, up to 7 x day. despite laxatives…poor appetite…"		
04/02/2005		Consult with unnamed Dr at the medical centre. "X-	

		ray results given normal findings."	
22/02/2005	Dr B referred Mr A to the Gastroenterology Outpatient Department.	Referral returned by hospital as "non-urgent" case.	
29/03/2005	Consult with Dr B "V due to poor bowel motility."		
12/05/2005	Consult with Dr B "Ongoing constipation, with blood always in stool, constipation followed by diarrohoea…" Weight 70.		
19/07/2005	Blood tests ordered by Dr B		creatinine 0.15 mmol/L elevated creatinine
25/07/2005	GP consultation with Dr B. Vomiting and dehydration. Bloods ordered		
29/07/2005	Consult with Dr B "Vomiting… dehydrating, with ^creat of 0.15. Needs admission for Lx and Rx if persists."		
30/07/2005	Consult with Dr B "…eats dinner only, small amounts only, with constipation, relieved with Codalax but as diarrhoea."		
1/08/2005	Consult with Dr B		
2/08/2005	Consult with Dr B		
22/08/2005	Consult with Dr B "return of sx of diarr…"		

	Weight 69		
29/09/2005	Consult with Dr B		
12/10/2005	Consult with Dr B		
12/12/2005	Consult with Dr B "Still anorexia… managing very little food and ongoing abd pain…"	Lab referral — Renal Creatinine.	
28/12/2005	Consult with Dr B.		
4/01/2006	Blood tests ordered by Dr B		creatinine 0.15 mmol/L Abnormal results indicating elevated creatinine
18/01/2006	Consult with Dr B. "refer [Dr C] as ongoing SX."		
13/02/2006	Consult with Dr B		
15/02/2006	Dr B referred Mr A to Gastroenterologist Dr C with one year history of vomiting and weight loss.	Gastroenterologist Dr C diagnosed with gastro-oesophageal reflux disease. Letter from Dr C to Dr B "I have checked his lithium…levels..."	
17/02/2006		Results of blood tests referred to in Dr C's letter of 15/02/2006.	lithium 1.2, creatinine 0.15mmol.L Abnormal results indicating possible lithium toxicity and elevated creatinine

6/03/2006		Gastroenterologist Dr C wrote to Dr B advising that Mr A had raised serum creatinine. — reduce lithium dose.	lithium levels 1.2, creatinine 0.15 mmol/L Abnormal results indicating possible lithium toxicity and elevated creatinine.
15/03/2006	Consult with Dr B. "reduce dosages of Lithium and Epilim."		
12/04/2006	Blood tests ordered by Dr B		lithium 0.7 No lithium toxicity
18/05/2006	Consult with Dr B. Irritable bowel.		
21/06/2006		Assessment in the DHB medical outpatient clinic with history of weight loss, anorexia and vomiting.	
22/06/2006	Consult with Dr B. "To reduce Quetiapine to one bd as s/e may be affecting him; consider stopping completely."	Blood tests ordered by Dr G, Out Patients, Dr B advised to follow up	creatinine 154 µmol/L Abnormal results, elevated creatinine
4/07/2006	Consult with Dr B. "To reduce Seroquel. Has had several episodes of tachycardia with some irreg beats…"		
10/08/2006		Letter from Gastroenterologist Dr C to Dr B following review of Mr A. GI symptoms including vomiting persisting but	

		improved alertness following reduction in lithium quetiapine doses.	
17/08/2006		Gastroenterologist Dr C wrote to Mr A (cc Dr B) advising him that his lithium level was back in the normal range.	Lithium 0.8 mmol/L
5/09/2006	X-ray and Barium Enema ordered by Dr B		"No cause for symptoms demonstrated"
21/09/2006	Blood tests ordered by Dr B, noted deterioration in renal function		creatinine 163 µmol/L Abnormal results, elevated creatinine
2/10/2006	Consult with Dr B. Noted Mr A was "coping well" on current medication.		
10/01/2007	Dr B referred Mr A to psychiatrist Dr E for an opinion regarding Mr A's initial diagnosis of bipolar disorder.		
16/05/2007		Blood tests ordered by Dr E	lithium 0.7, creatinine 165 µmol/L Abnormal results, significantly elevated creatinine
22/05/2007		Bloods ordered by a GP at the DHB	creatinine 182 µmol/L Abnormal results, significantly elevated creatinine

22/05/2007	Stomach examination by Barium meal ordered by Dr B		No abnormality found to explain symptoms
25/05/2007		Consult with unnamed Dr at the medical centre.	
26/05/2007		Consult with Dr I (medical centre.) "Eaten nil since Tuesday."	
29/05/2007		Consult with Dr I (medical centre.) "Seeing [Dr E] tomorrow…"	
30/05/2007		Psychiatrist Dr E responded to Dr B advising him to discontinue Mr A's lithium treatment.	
06/06/2007	Dr B referred Mr A to the DHB inpatient mental health services regarding persistent and notable vomiting and anorexia.		urine creatinine/24 10.0 mmol/L (normal range 9.0–18.0)
7–20 /06/2007		Admission to the DHB inpatient mental health services following anxiety, weight loss and vomiting. No mention of abnormal blood tests in discharge summary.	
26/06/2007	Consult with Dr B. "Ongoing LOW and panic associated N and V and anorexia…dry mouth s/e of Rx and thus wants to stop Rx		

	— encourage ongoing use but consider dose reduction."		
19/11/2007		Consult with Dr H (medical centre). "Constipation…"	
20/11/2007		Consult with Dr H (medical centre) "seeing [Dr E]. Private."	
3/12/2007		Consult with Dr H (medical centre)	
31/12/2007		Consult with unnamed Dr at the medical centre.	
17/01/2008	Consult with Dr B. Notes "Faecal Impaction ++ Fleet enema stat."		
29/01/2008		Formal discharge from the DHB Mental Health and Addiction Service.	
21/02/2008	Dr B notes "Consider Busiprone." (a drug used for the treatment of anxiety disorders.)		
5/05/2008		Letter from psychiatrist Dr E to Dr B, diagnosis "generalised anxiety disorder ?bipolar disorder in remission."	
01/07/2008	Consult with Dr B. Noted anxiety and depression. Weight 54.		
29/07/2008	Consult with Dr B "Ongoing low weight,		creatinine 101 µmol/L (normal

	with good soft diet…" Weight 54. Bloods tests ordered by Dr B — Creatinine, Renal.		50–120)
15/09/2008	Consult with Dr B. Weight 52.		
2008		Mr A was seen by psychiatrist Dr E on several occasions until at least May 2008. (no further info regarding dates)	
09/01/2009		Consult with a doctor (medical centre) for a bee sting. "Low BMI longstanding, anxiety evident: Mental health hx noted."	
15/07/2009	Consult with Dr B. "Struggling with …Depression, exacerbated by loss of job and inability to find a new one…" Weight 65.		
07/08/2009	Consult with Dr B, Mr A "keen to find appropriate work."		
24/08/2009		Consult with Dr F (medical centre). "Diarrhoea, vomiting, headache and fever."	
23/10/2009	Consult with Dr B.		
5/11/2009	Blood tests ordered by		creatinine 145

	Dr B		µmol/L Abnormal results, elevated creatinine
31/12/2009	Consult with Dr B "migraines and epistaxes."		
13/01/2010	Consult with Dr B "unable to manage work owing to anxiety and stress…"		
12/02/2010	Blood tests ordered by Dr B.		(results on 16/02/10)
16/02/2010	Blood tests ordered by Dr B		creatinine 143 µmol/L Abnormal results, elevated creatinine
17/03/2010	Consult with Dr B "Malaise, lethargy, headaches."		
31/05/2010	Consult with Dr B "Weight loss of 7kg over 6/52."		
4/06/2010	Consult with Dr B, Faecal impaction. Prescribed Microlax and Movicol.		
29/08/2010		Consult with doctor (medical centre), fevers aches and cough. "flu like illness."	
30/09/2010		Consult with Dr F (medical centre). Pneumonia. Augmentin prescribed.	

4/10/2010		Consult with Dr F (medical centre) Chest improving, pneumonia improving.	
7/10/2010		Consult with Dr H and Dr F (medical centre). Mr A coughing, lungs clear.	
8/10/2010		Consult with Dr I (medical centre). Mr A losing vision, persistent cough and vomiting for previous 4–5 weeks. Mr A was admitted to hospital and diagnosed with CKD.	Creatinine 206 µmol/L Abnormal results, significantly elevated creatinine