

**IN THE DISTRICT COURT
AT WELLINGTON**

**I TE KŌTI-Ā-ROHE
KI TE WHANGANUI-A-TARA**

[2021] NZACC 35

ACR 375/17

UNDER	THE ACCIDENT COMPENSATION ACT 2001
IN THE MATTER OF	AN APPEAL UNDER SECTION 149 OF THE ACT
BETWEEN	PAUL KINNEY Appellant
AND	ACCIDENT COMPENSATION CORPORATION Respondent

Hearing: 27 November 2020

Appearances: Mr P Sara for the appellant
Mr H Evans for the respondent

Judgment: 15 February 2021

**RESERVED JUDGMENT OF JUDGE C J McGUIRE
[Section 25 – Accident; Section 26 – Personal Injury; Causation]**

[1] At issue on this appeal is the correctness of the respondent’s decision of 22 February 2017 to decline cover for lumbosacral discitis on grounds that there was no causal link between the discitis and a covered accident.

Background

[2] The appellant was granted cover for the sprain of his hamstring tendon, left side, following an accident at his home on 11 December 2016. The description of the accident in the claim form was “pulling fire out of hearth and hurt leg”. It seems common ground that this accident occurred while the appellant was in the process of removing a log burner off the hearth at his home.

[3] He lodged a further claim on 28 January 2017 for an accident that occurred that same date. The description in the claim form was “lifting heavy furniture”. ACC’s later cover assessment recorded that the appellant was removing and putting down new carpet at home and lifting heavy furniture on 25 January 2017.

[4] Under the diagnosis section of the claim form of 28 January 2017 is “staphylococcus aureus discitis”.

[5] On 2 February 2017, the appellant attended at the Dunedin Hospital Emergency Department with acute back pain. The notes record:

Clearing house and lifting carpets all day Monday.

Woke up Tues am severe back pain.

After getting up pain eased through day.

Wednesday went to work, when sat in truck noticed severe pain again.

Came on gradually, seen GP yesterday – given tramadol.

Pain gradually increased, unable to get into car so ambulance called.

Pain now 10/10 felt central lumbar region, radiates to right side and down right buttock.

[6] An x-ray was taken on 2 February 2017 with the following findings:

Alignment is mildly straightened but otherwise satisfactory.

There is similar intervertebral disc height narrowing at the L5/S1 level.

Otherwise vertebral body and intervertebral disc height is maintained throughout with no fracture seen.

The sacroiliac joints are normal.

[7] The appellant was admitted to hospital on 3 February after presenting with lumbosacral regional back pain and a fever.

[8] An MRI scan of his spine on 6 February 2017 included the following findings:

Alignment is satisfactory.

There is marked loss of disc height at L5/S1 level with radial tearing/fluid seen within the disc. There is anterolateral and posterior marginal osteophytes and adjacent fatty end plates changes.

There is mild generalised disc bulge at L5/S1 with a posterior annular tear. mild bilateral foraminal narrowing. There is significant thickening of the epidural fat posterior to the L5/S1 vertebral bodies and disc. Mild oedema is seen in the anterolateral soft tissues around the L5/S1 disc space.

Post contrast, there is peridiscal enhancement at this level and also patchy enhancement involving the thickened epidural fat. Non enhancing areas are seen within the fat but no discrete epidural collection is seen. Enhancement is seen around the thecal sac extending down to the mid S2 level. Mild enhancement is seen involving the anterior and left lateral aspect of the L5/S1 disc. There is mild to moderate canal narrowing at the L5/S1 level.

Lumbosacral spine otherwise unremarkable.

Comment

The appearances are consistent with L5/S1 discitis with associated thickening and inflammation involving the anterior epidural fat at this level. No discrete epidural collection seen. Inflammation also seen around the thecal sac extending down to the mid S2 level.

[9] The discharge summary recorded that the appellant had long standing lower back pain.

[10] His discharge diagnosis on 3 February 2017 was staphylococcus aureus discitis. He was discharged on 3 February but admitted the same evening with severe back pain.

[11] On 22 February 2017, ACC's medical advisor, Dr Walker, commented:

The client has developed an unusual infection involving the L5/S1 disc region called discitis. This is unrelated to lifting. Bacteria in the blood stream lodge in this area and result in infection. Staphylococcus aureus is the organism most commonly found. The discitis is the cause of the client severe pain and his becoming increasingly unwell.

[12] By letter dated 22 February 2017, ACC declined the appellant's claim because the discitis was caused by a staphylococcus aureus infection, not the covered accident.

[13] The appellant saw Mr Hodgson, orthopaedic surgeon, on 17 March 2017. In his report Mr Hodgson said he had treated the appellant conservatively in 1997 and

1998 for back pain. At that time the appellant had moderate narrowing of the L4/5 canal and a mild bulge on the left-hand side of the L4/5 disc. His opinion was that flu had caused the appellant's staphylococcus aureus discitis.

[14] Mr Hodson also said:

Paul has had an attack of staphylococcus aureus lumbosacral discitis which has probably come about as a result of the flu. He has injured his back lifting furniture.

This is the most likely scenario. It is quite significant bad luck but it is difficult to say that one led to the other in regards to his ACC decision.

I am not sure that I can state that one has led to the other.

[15] The appellant saw Dr Wolfgang at the medicine department of Dunedin Hospital on 23 March 2017. In her report she said:

He had been ripping up carpet and about 7-10 days later – developed acute back pain. After trauma to an area, increase protein up regulation can happen to repair damaged muscles. These proteins (vimentin) increase binding of bacteria like streptococcus pyogenes and S. aureus. The trauma may be mild as it was in this case.

[16] On 29 May 2017, the medical advisor, Dr Walker, gave further advice. He said: “In summary the client has had discitis caused by staphylococcus aureus. The staphylococcus aureus has entered the disc via the blood stream.”

[17] On 16 June 2017, Dr Wolfgang reported further after being asked to clarify why the back injury may have led to the secondary infection. She said:

... if the bony surface is intact, then the bacteremia will “seed” a distant foci. Trauma or disruption of the bone surface must occur first. S. aureus bacteremia has been associated with mild trauma and even with insect bites. Given the antecedent trauma one week prior to admission, this did likely play a role in the subsequent infection.

[18] Mr Hodgson provided a further brief report to ACC on 4 July, stating:

Mr Kinney has suffered an acute injury to his lumbosacral disc.

I suspect this led to inflammation, bleeding and subsequent infection secondary to haematogenous spread of the bacteria staphylococcus aureus that led to his acute discitis and the need for admission to hospital under the care of Mr Chris Birks.

[19] Mr Hodgson reported again on 10 August 2017, included is this:

The Corporation wrote to me in June 2017 and I replied on 4 July 2017 as outlined in my letter.

You will see that I had stated Mr Kinney had suffered an acute injury to his lumbosacral disc (lumbosacral disc protrusion). He developed the onset of his back pain and discomfort while moving furniture and lifting carpet in his house.

Unfortunately, at the time of this accident he was also suffering from a “flu like illness” and unbeknown to him his altered and weakened immune system allowed the staphylococcus aureus infection to become blood borne into his blood stream and settle out in the lumbosacral disc. This disc developed a full blown infection and lumbosacral discitis.

...

The onset of his symptoms has been acute and severe and this has followed closely after the incident that occurred while lifting carpet and shifting furniture.

Unfortunately the bacterial infection has gained hold into the injured disc and this has led to the acute severe infective lumbosacral discitis as confirmed on clinical radiologic ground along with the blood tests as outlined.

[20] Dr Walker provided a further report on 24 August 2017. In his summary he said:

The client has developed a staphylococcal discitis. This is a relatively rare infection where there is infection of the disc space and usually a degree of infection in the adjacent vertebral body.

The discitis has developed in the context of a febrile illness. The client’s principle risk factor for this condition appears to be degenerative spondylosis at the L5/S1 disc space.

The client hasn’t had an acute disc protrusion. Acute disc protrusions are not recognised in the medical literature as a cause of discitis.

[21] Mr Hodgson responded to Dr Walker’s report on 29 September. In his summary he said:

I do not accept Dr Walker’s timeline or information of what has happened.

Mr Kinney has not suffered an acute disc protrusion that has caused discitis.

He has suffered a disc protrusion at his lumbosacral level, that has occurred as a result of a trauma.

Unfortunately this disc injury has made the disc vulnerable to circulating bacteria in his blood, following the flu like illness and due to the vulnerable

nature of the disc after injury he has developed a secondary discitis in the disc as a result of this.

Dr Walker is correct in saying “the primary infection was not identified”. In over 50% of patients with staph aureus discitis the primary infective source is not identified.

[22] The matter proceeded to a review hearing, and in a decision dated 2 November 2017, the Reviewer dismissed the application.

[23] Following the review, a further report was obtained from Dr Wolfgang on 10 July 2018. She acknowledged that what is in contention in this case is how the metastatic deposits of *S. aureus* lodged in the lumbosacral disc. She says:

Thus some damage to the bone had to be established before mere *S. aureus* bacteremia would lead to bone infection. This is in agreement with Mr Hodgson hypothesis that damage had been done to the disc leading ... to the secondary discitis.

[24] Dr Wolfgang was further asked if she could say when the infection started.

[25] In her report of 11 July 2018, she said it was rather difficult to say when the bacteremia started. However, she says it is likely that the appellant had the initial inoculation of bacteria about one to three weeks prior to presentation.

[26] In a further follow up email of 11 July 2018, she said it was most likely that he had the injured disc and then had the bacteremia infection. She said: “you need trauma to the disc for the infection to set up”.

[27] The Corporation sought and obtained a further medical report from Richard Everts, specialist physician. Although he did not speak to the appellant, he reviewed the clinical information.

[28] In terms of a traumatic cause Mr Everts said:

In Paul’s case, the reports of the timing and nature of the possible traumatic events are mixed. The physiotherapy and medical notes from 1, 2 and 3 February 2017, when Paul presented with the acute infection, do not indicate a significant traumatic event. On 11 December Paul moved a heavy hearth, then felt what sounds like radicular nerve pain in his leg. On 23 January Paul was lifting carpet into a utility when he felt back pain. On Monday 30 January Paul was lifting carpet during the day but did not report any acute pain on that day.

These are “activities” rather than specific traumatic events. As a labourer it is likely that lifting objects was part of his normal work, although these objects may not be as heavy as a hearth or rolls of carpet. I accept that strenuous or awkward activities may sometimes cause muscle strain or disc damage, but from what I have read in Paul’s reports I think it is unlikely that any of these was severe enough to cause significant inflammatory or haemorrhagic damage to his deep tissues.

[29] He says further:

If the hearth lifting on 11 December caused trauma, I think this would have largely resolved (his leg pain and SLR test had resolved) by the time Paul’s spine was seeded with staphylococcus aureus, which I estimate occurred sometime between 17 and 29 January 2017. If carpet lifting into a utility truck occurred on 23 January 2017 and this caused trauma this could have been seeded by infection and presented in the timeframe. If the carpet lifting on 30 January caused trauma, the time between this event and the onset of pain the next morning was probably too short to represent consequent infection of traumatised tissue.

[30] Mr Everts concludes:

The staphylococcus aureus most likely entered Paul’s blood in late January 2017 from his nose or throat or from a minor break in his skin, then floated around and landed in the L5/S1 disc. The disc level is a common site for haematogenous staphylococcus aureus infection and in Paul’s case may have been vulnerable because of longstanding degenerative disease.

[31] The Clinical Advisory Panel (the Panel) considered this case and reported on 16 May 2019. The report included:

The CAP concluded that, based on the available information, the staphylococcus may have been colonised in his nostrils or other body parts, breathed during his flu, or from another source. The primary source of Mr Kinney’s staphylococcus bacteria was never identified. We also don’t know why the staphylococci “chose” to settle on L5/S1. Where it came from and why it invaded are not questions relevant to Mr Kinney’s management and recovery.

In summary, the CAP concluded that the most likely explanation is that the staphylococci invaded a pre-existing, long standing area of wear and tear at L5/S1 and unfortunately caused a serious life threatening infection that was not trauma related. There is no evidence of any causal relationship or contribution from the 1997, 2007 events, or the 11/12/2016 or 23/01/2017 ACC covered back sprains, or any combination of these.

[32] Dr Wolfgang was asked whether she agreed with Dr Everts that significant trauma is required for the antecedent injury.

[33] On 31 May 2019, Dr Wolfgang replied by email:

Yes I think the statement of significant trauma is key. It will be difficult to prove a proper “accident” in this case or was the carpet incident just normal work. I think the ACC legislature is great when a clear traumatic event can be shown (i.e. falling off a horse) but for normal work related repeated stress, it is very difficult to substantiate.

[34] Dr Wolfgang followed this up in a further email of 13 June 2019:

... but I can infer you are asking “is lifting carpet enough trauma to lead to this? So I think yes, this sort of trauma could lead to minor haemorrhage which then could “feed bacteria” during episode of transient bacteraemia. The pathophysiology is that trauma is necessary to get the bacteria to adhere as healthy bone is relatively resistant to infection in adults.

[35] Mr Hodgson finally reported on 5 November 2019, saying amongst other things:

It is always difficult to know why it would seed into one disc and not another disc, but as I have stated in my opinion, I consider that Paul had suffered a lumbosacral disc disruption or prolapse when he injured himself, initially in December and then exacerbated in the week end while conjointly suffering from the flu.

Unfortunately for Paul while he had the flu, he was rendered immunologically compromised as a result. The staphylococcus aureus bacteria (a normal commensal bacteria found in the nose or on the skin) seeded itself through his bloodstream and into the inflamed area at the lumbosacral disc.

...

At this stage, on the balance of probabilities, I believe Mr Kinney had suffered an injury to his lumbosacral disc in December and then exacerbated this in January. In January he developed the onset of flu that depressed his immune status and allowed the staphylococcus aureus bacteria to seed into his bloodstream and cause the septic lumbar discitis.

The Appellant’s Submissions

[36] Mr Sara referred to the appellant’s statement of evidence given at the review hearing. He describes the incident on 11 December 2016 with the log burner where after pulling it backwards he stood up and felt as though he had pulled something in the back of his left leg.

[37] After seeing a locum on 15 December, he was referred to physiotherapy and had approximately four treatments. He said that by 19 January he no longer had pain

down his leg but that two days later he became involved in the process of recarpeting his house and that on 23 January, in the process of trying to get a carpet roll onto the back of his Ute, he felt something give in his back. He said the pain in his lower back was about 5 or 6 on the pain scale. He worked that week with his sore back, but on 28 January 2017 he felt increasingly unwell and the following day had flu like symptoms. Two days later on 31 January 2017, he lifted a tree in a pot onto the back of his vehicle and afterwards noticed his back getting sore and had a restless night.

[38] He saw his physiotherapist on 1 February 2017, but there was no improvement. The following day he saw his doctor who prescribed pain relief, but the pain was such that he decided he needed to go to hospital, and he was taken there by ambulance. He was in hospital for two weeks while the infection was treated.

[39] Mr Sara carefully recites the competing medical opinions. He submits that the issue of the source of the infection is irrelevant as all that is important is that the bacterium got in.

[40] He notes that the Clinical Advisory Panel agrees that the staphylococci settled on the L5/S1 disc and the state of the disc was “damaged” and “weakened”.

[41] Significantly, however, he says the Clinical Advisory Panel went on to say: “We also don’t know why the staphylococci “chose” to settle on L5/S1.”

[42] Mr Sara submits that the doctors involved in this case who are infectious disease experts are best qualified to answer this question, they being Dr Wolfgang and Dr Everts.

[43] Mr Sara notes that Dr Everts is uncertain regarding the degenerative disc disease “theory”.

[44] He notes Dr Everts said:

On the one hand, I have not seen any research that specifically links the risk of pyogenic discitis with degenerative disc disease. So Paul’s L5/S1 disc may have been particularly vulnerable to infection as a result of a long history of degenerative disc disease, although this is not certain.

[45] He notes that Dr Wolfgang did not agree that there needed to be the degree of pathophysiological change as suggested by Dr Everts and that the traumatic event of lifting a carpet, allowing the lumbar spine region to become inflamed, led to the seeding of bacteria to that disc space.

[46] And Mr Sara poses this question, “Why did the infection target one particular area of Mr Kinney’s spine, rather than other areas which are demonstrably affected by degenerative disc disease?”.

[47] He submits that Dr Wolfgang’s answer is that an injury is the key to the establishment of infection and that she goes on to say that the degree of trauma does not need to be substantial.

[48] Mr Sara submits that the key to resolving this case is determining whether the appellant’s L5/S1 disc was merely in a degenerative state consistent with a degenerative disc disease, or whether it was injured.

[49] In this regard he submits that Mr Hodgson is most likely to be authoritative. Like Dr Wolfgang he observes that Mr Kinney had degenerative changes in other areas of his spine, particularly at L2/3 and L3/4, but these were not affected by the bacteria.

[50] Mr Sara notes Mr Hodgson’s opinion that something acute happened at the lumbosacral disc, firstly in December, then exacerbated by the incident in January by pulling up carpet.

[51] According to Mr Hodgson:

This in my opinion was the lumbosacral protrusion that led to the onset of the irritation of the right S1 nerve root and the “so called” symptoms of hamstring pain he suffered from.

...

The “classic distribution” of nerve root pain to the leg is usually to the hamstring area. It is associated with irritation and inflammation of the S1 nerve root compression caused by a disc protrusion.

The likelihood of this prolapse leading to significant secondary inflammation of the disc and around the nerve root is high. In my opinion, the staphylococcus aureus bacteria has seeded itself into this area and caused the infective lumbar discitis which he then suffered from.

[52] Mr Sara submits that, to a substantial degree, the Panel concurs with this view. He says that although the Panel cannot bring itself to use the word “injury”, it nevertheless concedes that the L5/S1 disc was damaged and weakened.

[53] Mr Sara submits the appellant is entitled to cover for the condition correctly diagnosed by Mr Hodgson, namely lumbosacral disc disruption or prolapse. He is entitled to cover for the infection which then followed to the spinal area, by virtue of s 20(2)(g) of the Act.

The Respondent’s Submissions

[54] In his submissions on behalf of the Corporation, Mr Evans acknowledges that while a person has cover for personal injury caused by gradual process, disease or infection consequential on personal injury suffered by the person for which the person has cover, *McDonald v Accident Rehabilitation and Compensation Insurance Corporation* is the primary authority relating to pre-existing degenerative changes.¹

[55] In that case the High Court cited with approval the comments of the District Court in *Hill v Accident Rehabilitation and Compensation Insurance Corporation*, namely:²

If medical evidence establishes there are pre-existing degenerative changes which are brought to light or which become symptomatic as a consequence of an event which constitutes an accident, it can only be the injury caused by the accident and not the injury that is the continuing effects of the pre-existing degenerative condition that can be covered. The fact that it is the event of an accident which renders symptomatic that which previously was asymptomatic does not alter that basic principle. The accident did not cause the degenerative changes, it just caused the effects of those changes to become apparent and of course in many cases for them to become the disabling feature.

¹ *McDonald v Accident Rehabilitation and Compensation Insurance Corporation* [2002] NZAR 970 (HC).

² At [26] citing *Hill v Accident Rehabilitation and Compensation Insurance Corporation* DC Huntly 189/98, 1 September 1998 at 13.

[56] Mr Evans submits that based on the medical tests and studies, the bacteria probably landed on the appellant's L5/S1 disc two days to two weeks before the onset of his symptoms on 31 January 2017.

[57] He submits that the traumatic activities in December 2016 and January 2017 were not significant enough to cause substantial haemorrhage or inflammatory damage and were not associated with objective signs of trauma.

[58] He submits that the *S. aureus* invaded a pre-existing, long standing area of wear and tear at L5/S1 and caused a serious life-threatening infection, which was not trauma related.

[59] He submits that Mr Hodgson's opinion that the appellant suffered an acute disc protrusion is not supported by any contemporaneous medical records. He says, in contrast, the records show that the appellant was able to perform heavy work on the accident date and more heavy work two days after the accident and that this capacity would be inconsistent with suffering an acute disc protrusion.

[60] He refers to Dr Everts' opinion that the carpet lifting incident on 23 February did not cause the appellant's infection as there was no discrete traumatic event nor was the possibility of a traumatic event documented nor did the appellant's history of doing more physical work during the subsequent week indicate a substantial haemorrhage or inflammatory traumatic event.

[61] Mr Evans submits the appeal should be dismissed.

Decision

[62] Over the relevant period from December 2016 to February 2017, the appellant had two claims accepted by ACC. Although various documents on the file suggest otherwise, it is reasonably clear that the appellant injured himself on 11 December 2016 while in the process of removing a yunca log burner off the hearth. He was granted cover for that injury which was described in the claim form as "sprain, hamstring tendon".

[63] It is noted that on referral to physiotherapy, the physiotherapist concluded on 15 December 2016 that the injury was to the appellant's back rather than his leg.

[64] In the evidence submitted by the appellant to the Reviewer, he says that by 19 January 2017 he no longer had pain down his leg, but he could not recall whether he had pain in his lower back as well.

[65] Then followed the task undertaken by the appellant of removing carpet from his house from approximately 21 to 23 January 2017.

[66] During that time, while loading a roll of carpet onto the back of his Ute, he felt something give in his back. From a pain score of 2 or 3, this quickly increased to about 5 or 6 according to the appellant. He said he probably took some kind of pain relief.

[67] On 25 January, he went to work with his back still sore, but the pain was at about 3 on the pain scale.

[68] It was on the following Saturday night, 28 January, that he became unwell with flu like symptoms. He took a day off work on 30 January 2017 as well as the next day when he felt no better. On Tuesday 31 January, after lifting a potted plant into the back of his vehicle he noticed his back getting sore with a pain level of about 6. The following day, 1 February 2017, he went to work but by lunch time the pain was intolerable, and he went to his physiotherapist for treatment. He then had a bad night, and the next morning his wife took him to the doctor. He says he struggled to get in and out of the car. The doctor prescribed pain relief, but his pain worsened; according to him, up to 10 out of 10, and he was taken to hospital in an ambulance. He was discharged the following morning with stronger pain relief but was readmitted the same day and was in hospital for two weeks while the infection was treated.

[69] The second ACC injury claim form with an injury described as staphylococcus aureus discitis was filed by the Southern DHB and dated 28 January 2017. The

description of the injury in that claim was: “Heavy lifting furniture ... household or garden chore ... lifting/carrying/load.”

[70] It needs to be said that there is some disconnect relating to dates with the claim form dated 28 January 2017. Logically, it should have been the date of his admission to hospital on 2 February 2017.

[71] Nothing turns on his inaccuracy.

[72] The challenge for the Court in this case is to decide whether or not on the balance of probabilities the appellant has proven that he has suffered a personal injury caused by infection consequential on a personal injury for which he has cover, as s 20(2)(g) requires.

[73] What is clear is that the appellant suffered a staphylococcus aureus infection in his L5/S1 disc. The real issue is whether while moving carpet and furniture the appellant did suffer an injury to his L5/S1 disc and whether the infection was consequential on that injury thus resulting in a personal injury for the purposes of s 20(2)(g), namely lumbosacral discitis.

[74] It is fair to say that the issue for determination in this case has been the subject of detailed consideration by all those clinicians asked to give opinions.

[75] I acknowledge that all the medical views in this case are carefully considered. Dr Wolfgang, from the Infectious Disease Clinic at Dunedin Hospital, plainly has significant relevant expertise. She noted that for the staphylococcus aureus infection to take hold, trauma or disruption of the bone surface must first occur. She notes in her report of 16 June 2017 that *S. aureus* bacteremia has been associated with mild trauma and even with insect bites.

[76] Later, in an email dated 13 June 2019, addressing the question again of whether there needs to be significant trauma, she says:

... I can infer you are asking “is lifting carpet enough trauma to lead to this?”
So I think yes, this sort of trauma could lead to minor haemorrhage which then could “feed bacteria” during episode of transient bacteraemia. The

pathophysiology is that trauma is necessary to get the bacteria to adhere as healthy bone is relatively resistant to infections in adults.

[77] She then says:

I don't know exactly how much trauma is needed to cause this – Mr Kinney clearly gives a history of significant traumatic work which likely did inflame the area. We are estimated to have 200 episodes of transient bacteraemia per year. Bacteria often cannot attach to healthy bone/heart valves but when inflamed they can attach forming a clump of bacteria leading to the issue of Mr Kinney's discitis.

[78] In support is the appellant's orthopaedic surgeon, Mr Hodgson. In his final report of 5 November 2019, he says:

It is always difficult to know why it would seed into one disc and not another disc, but as I have stated in my opinion, I consider that Paul had suffered a lumbosacral disc disruption or prolapse when he injured himself, initially in December and then exacerbated in the weekend while conjointly suffering from the flu.

Unfortunately for Paul, while he had the flu, he was rendered immunologically compromised as a result. The staphylococcus aureus bacteria (a normal commensal bacteria found in the nose or on the skin) seeded itself through his bloodstream and into the inflamed area at the lumbosacral disc.

[79] The Corporation obtained a detailed report from Mr Everts, specialist physician.

[80] Mr Everts notes that he has provided ACC with some 325 expert reports on infection claims and many have involved cases in which infection develops after possible trauma. Mr Everts notes six questions:

- Was there a clearly defined acute event, like a sudden fall, direct blow or forceful twist with immediate pain which was likely to cause a substantial haemorrhagic or inflammatory injury to deep tissues?
- Was the traumatic event prospectively documented?
- Was there documentation of objective physical evidence of trauma?
- Were intraoperative findings consistent with infected injury?

- Was there a temporal link between the traumatic event and the deep infection that is reasonable considering the incubation period of the proven or likely causative micro organism?
- There should not be other circumstances that overwhelmingly or predominantly contributed to the infection and its sequelae compared with the traumatic event.

[81] In the appellant's case Mr Everts notes that the reporting of the timing and nature of possible traumatic events are mixed and the medical notes from 1, 2 and 3 February 2017 did not indicate a significant traumatic event.

[82] He goes on:

I accept that strenuous or awkward activities may sometimes cause muscle strain or disc damage, but from what I've read in Paul's reports I think it unlikely that any of these was severe enough to cause significant inflammatory or haemorrhagic damage to his deep tissues.

[83] Mr Everts notes that there was no obvious physical examination or MRI evidence of acute trauma.

[84] He also says:

In Paul's case there was no discrete acute traumatic event. Instead, his pain gradually increased, which is suggestive of a primary infective process rather than a traumatic event that was subsequently complicated by infection.

[85] However, he also notes that if the lifting of the carpet onto the utility vehicle occurred on 23 January 2017 and caused trauma, this could have been seeded by infection and presented in the timeframe.

[86] Mr Everts concludes from his evaluation that trauma was not the primary cause of the appellant's infection.

[87] The Panel's report of 16 May 2019 is detailed. Amongst other things it says:

The normal 1997 bony x-ray mentioned in Mr Hodgson's 10/08/2017 report looks at bones and does not exclude degenerative disc changes. In fact, Mr Kinney's L5/S1 disc already had signs of disc degenerative disease documented in 2007, and his lower lumbar L3/4 and L4/5 vertebrae had disc changes noted as far back as 1997 according to Mr Hodgson's 10/08/2017

report. The CAP noted that those findings are most consistent with progressive degenerative changes as opposed to being the result of one off trauma. The CAP agreed that the underlying cause of Mr Kinney's disc degeneration was tissue "weakening" as noted by Mr Hodgson. But this was not related to Mr Kinney's lifting carpets and furniture or ripping out carpet. The CAP noted the medical evidence indicates that Mr Kinney's "weakening" at the L5/S1 area is due to his genetic inheritance, aging, nutritional compromise, and loading history. This can weaken discs to such an extent that structural failure occurs during activities of daily living. ...

The predominant cause of his L5/S1 disc failure, however, was the tissue weakening that came before, not at, his 2016 and 2017 renovation activities. The CAP notes that the L5/S1 structural failure was the end stage of what is primarily a progressive disease process. The staphylococcal bacteria then conquered this area of "damage" and "weakening" causing Mr Kinney's vertebral osteomyelitis and secondary discitis. This infection was therefore, not causally related to any single accident in 1997, 2007, 2016 or 2017, or any combination of these.

...

In summary the CAP concluded that the most likely explanation is that the staphylococci invaded a pre-existing, long standing area of wear and tear at L5/S1 and unfortunately caused a serious, life threatening infection that was not trauma related.

[88] The Panel acknowledges that the appellant's activities in December 2016 and January 2017:

... were related to the onset of his low back and left leg pain. The predominant cause of his L5/S1 disc failure, however, was the tissue weakening that came before, not at, his 2016 and 2017 renovation activities. The CAP notes that the L5/S1 structural failure was at the end stage of what is primarily a progressive disease process.

[89] However, the Panel stops short of saying that the activities in question caused no lumbosacral disc protrusion, albeit a minor one, as a discrete injury over and above the acknowledged degenerative changes.

[90] It needs to be acknowledged that all the information on the file provides a picture of the appellant as a conscientious, hard working man who gets on with the job and who is not held back by back injuries, discomfort or pain that many others would regard as significant and debilitating.

[91] In his evidence before the Reviewer, the appellant describes the events from 21 January 2017 onwards as follows:

We were in the process of re-carpeting the house. In order to do that all the furniture had to be shifted out to the...out of the house to the garage...The whole process took three days ...

All the carpet which had been lifted had been rolled up and I intended to take one trip to the tip and dump it...

The process of trying to get the carpet rolled up...a role back into the back of the ute involved twisting, lifting and pushing. In the process of doing so I felt something in my back. It was—it was painful and around two or three on the pain scale.

The roll of carpet was too heavy. I estimated it to be 50-60 kilos but bulky and awkward to lift. I managed to force the carpet into place and then I stopped work...

I went inside ... and I noticed that my back was sore and pain in my lower back, about a five or six pain scale. The pain was dull. I probably took some kind of pain relief.

The following morning, I went to work, my back was still sore, about three on the pain scale. I was able to work with the pain I had.

The new carpet was installed on the 24th and 25th of January and once that had been completed we then had to shift all the furniture back into the house over the next five days. My back was sore doing so.

[92] In his final report of 5 November 2019, Mr Hodgson says:

While I accept that Mr Kinney had indeed suffered from degenerative problems in his lumbar spine, which had been clearly defined over the past 20 years, something acute had happened at the lumbosacral disc with his accident , firstly in December and then exacerbated by the incident in January by pulling up carpet. This, in my opinion, was the lumbosacral protrusion that led to the onset of irritation of the right S1 nerve root and the 'so called' symptoms of hamstring pain he suffered from.

[93] Mr Hodgson has the advantage of having treated the appellant for back issues since 1997. In this case, therefore, because of his longitudinal specialist knowledge of the appellant I conclude that his views are to be preferred to those of the Panel.

[94] I find in this case that what the appellant describes was, in combination, sufficiently traumatic to have caused at least a minor lumbosacral protrusion, over and above the degenerative changes, and that what then occurred was a personal injury for the purposes of s 20 (2) (g), being a personal injury caused by infection consequential on a personal injury suffered by a person for which the person has cover.

[95] Accordingly, the appeal is allowed and the respondent's decision of 22 February 2017 declining to cover lumbosacral discitis is reversed.

[96] Should there be any issue as to costs, counsel have leave to file memoranda in respect thereof.



Judge C J McGuire
District Court Judge

Solicitors: Peter Sara, Barrister and Solicitor, Dunedin for the appellant
Young Hunter, Christchurch, for the respondent