IN THE DISTRICT COURT
AT WELLINGTON

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

[2021] NZACC 66 **ACR 69/20**

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

Hearing: 15 March 2021
Heard at: Wellington/Te Whanganui-A-Tara

Appearances: Ms H Armstrong for the appellant
Mr M Clarke-Parker and Mr S Kinsler for the respondent

Judgment: 27 April 2021

RESERVED JUDGMENT OF JUDGE C J McGUIRE
[Entitlement to surgery Section 64 and Schedule 1
Accident Compensation Act 2001]

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[1] The issue on this appeal is whether on 5 March 2018 the appellant suffered a back injury when he lifted two 20 litre containers of milk at his work, causing a condition that now requires treatment.

Background

[2] On 5 March 2018 the appellant had an accident at his work. He attended his GP, Dr Lay, the following day, 6 March 2018. Dr Lay completed an ACC injury claim form. The description of the accident was:

> Lifted 2 x 20 l containers, hurt my back, felt something crack.

[3] The diagnosis was lumbar sprain. He was deemed unfit for work until 20 March 2018, which was subsequently extended until 10 April 2018.

[4] The appellant was referred to orthopaedic surgeon, Mr Castens, who saw him and reported on 23 May 2018.

[5] Mr Castens noted that prior to the injury there were no problems in relation to the appellant's back. He referred to x-rays taken on 27 March 2018 that showed a mild list towards the right-hand side but:

> Otherwise he has satisfactory disc height and no evidence of spondylolisthesis.

[6] Findings in an MRI undertaken on 21 June 2018 included:

> L4/L5: diffuse posterior disc bulge accentuated right posterolateral. No canal stenosis, mild narrowing of the right lateral recess without nerve root compromise. No foraminal stenosis. Facet joints normal.
>
> …
>
> Comment:
>
> Spondylosis at L4-5 including Schmorl's node endplate changes and associated endplate oedema. Diffuse posterior disc bulge at this level but no canal stenosis or nerve root compromise.

[7] Mr Castens commented on the findings, stating:

> … the scan has shown satisfactory changes overall. There is evidence of Schmorl endplate changes at the L4/5 level. There is mild disc dehydration here and generalised disc bulge. There is no obvious evidence of nerve compression or entrapment here and the exit foramen looks to be satisfactory in the spine as well.

[8] Mr Castens also commented that the appellant does not have a reliable surgical target to be accomplished with an operation and it would be reasonable to consider undertaking a corticosteroid injection to try and help settle down the symptoms.

[9] The appellant subsequently undertook a CT guided corticosteroid injection on 3 July 2018, which provided temporary improvement. Mr Castens further recommended the appellant undertaken a strengthening programme of either swimming or walking combined with physiotherapy.

[10] In his discharge report of 17 August 2018, Mr Castens commented:

> The only abnormality evident on his MRI scan has been simple Schmorl's node changes at L4/5 interspace.

[11] In an Assessment Report and Treatment Plan (ARTP) dated 9 May 2019 Mr Castens, under the heading "causal medical link between proposed treatment and covered injury", said:

> Direct. Lifting 20 kilogram containers on the 5th of March 2018 sustained an injury to his lower back. He heard something crack within his lower back likely on the basis of a L4/5 disc protrusion and resultant discogenic lower back pain.

[12] On 11 July 2019, in a clinical comment on behalf of ACC, Mr Peter Hunter, Principle Clinical Advisor – Orthopaedic Surgery said:

> The condition to be treated is primarily gradual process degenerative disc disease which has become symptomatic.

[13] In their decision of 12 July 2019, ACC turned down the appellant's request for surgery to treat his L4/5 discogenic lower back pain.

[14] Prior to the review hearing, Mr Castens provided a further report on 9 February 2020, in which he said:

> The neighbouring levels L3/4, L5/S1 as well as proximal sites show satisfactory hydration of the discs and if one was to assume that there is a degenerate condition at play then you would expect these levels would also be affected on his imaging studies.
>
> …

> The modic endplate changes that are seen on the MRI scan sagittal images with oedema in endplates can result from post injury changes as well at three months from the injury as seen on the June 2018 scan. There is no oedema seen in neighbouring levels of the lumbar spine although there is a small Schmorl node inferior endplate L3 with no oedema.

[15] Mr Hunter added the following additional clinical comment on 17 February 2020:

> Can the above MOI (mechanism of injury) cause L4/5 discogenic pain? does the medical evidence support the MOI has caused this condition?
>
> Possibly but the general practitioner notes the day after the injury are of a simple lumbar sprain which resolved in 3-4 weeks.

[16] Following the review decision, the appellant was referred to Dr Ian Bell, Musculoskeletal Medicine Physician.

[17] In his report of 7 August 2020, Dr Bell described the mechanism of injury and said:

> The injury mechanism discussed above and the pathology that it has caused is recognised to be associated with the initiation of a process of disc degradation.
>
> …
>
> However taking into consideration all of the circumstances regarding the development of back pain and the injury itself, it is more likely than not that these changes particularly inflammatory endplate change, were not present prior.

[18] On 6 October 2020, ACC's Clinical Advisory Panel (the CAP) reported. Included in its report is the following:

> The CAP explained that lumbar spondylosis and disc degeneration are almost universal. They have complex, multifactorial causes. The tissue weakening occurs gradually, related to genetic inheritance, aging, nutritional compromise, and loading history.
>
> The CAP noted that gradual onset degenerative disc disease can weaken disc to such an extent that structural failure occurs during the activities of daily living. The predominant cause of such failure, however, is not lifting 2 x 20 L containers, but rather, the tissue weakening that has come before. The structural failure is the endpoint of the progressive spondylitic process. Naturally, the lower lumbar levels, such as Mr Rhodes' L4/5 level, are affected first because they deal with the greatest daily stresses at the bottom of the spinal column. The absence of significant interval change between the 20/06/2018 and 08/03/2019 MRI scans is clinically accepted and within expected parameters.

> Mr Rhodes' L4/5 disc bulge and signal changes was not caused by a single episode of trauma. With increasing age, disc water content decreases, especially in the nucleus, and the nucleus tends to bulge into the intervertebral bodies. Nucleus pressure is reduced. Increased vertical loading of the annulus causes it to bulge radially and outward, and sometimes inward. The disc behaves like a "flat tyre". No inference about causation can be drawn on the presence of any part of the spectrum of this disc disease which is commonly unrelated to trauma.
>
> …
>
> The CAP commented that people develop low back pain all the time. Back pain is one of the most common causes for patients to seek medical care in both primary care and emergency setting. Some estimates of life time prevalence are as high as 84% in the adult population. Dr Bell did not explain why he does not believe this common clinical scenario. We all get back pain at some time in our lifetime. This does not imply that the symptoms are not real; it is well recognised that not all pain, both acute and chronic, is due to actual physical injury. Pain does not always indicate actual physical injury or actual tissue damage.

[19] The CAP concluded:

> The CAP considered that Mr Rhodes had pre-existing degenerative disc disease of his lumbar spine and the symptomatic aggravation from lifting 2 x 20 L containers was the predominant cause of his symptoms on 05/03/2018.

[20] On 17 November 2020 radiologist, Dr Coates, reviewed the MRI scans and the CT discography images. He said:

> In answer to your specific question with such localised changes occurring after a defined history of trauma (in the absence of a study prior to the injury to confirm these are longstanding) it is definitely possible that the changes at the L4/5 level are at least in part contributed by trauma. So called modic 1 changes simply refer to oedema adjacent to the endplate and this can also be seen in endplate fractures, traumatic Schmorl's nodes. In summary it is definitely possible that trauma has played a significant part in the changes that have developed at the L4/5 level.

[21] Dr Bell provided a further report on 26 November 2020. Amongst other things, he said:

> … the CAP makes reference to my report discussing that one outcome of disc injury is annular tears. They comment that this was not reported on Mr Rhodes's imaging.
>
> My comment:
>
> I have discussed Mr Rhodes's imaging with Dr Mark Coates, specialist MSK radiologist at Pacific Radiology Christchurch. He has in fact indicated that there is evidence of an annulus fissure, most pronounced on the discography

> images. So although these are not reported on original imaging, Mr Rhodes does in fact have annular pathology noted on imaging studies performed to date.

[22] Dr Bell goes on to acknowledge degenerative disc disease is not recognised to be a cause of pain. However, he states:

> I have indicated that imaging findings, specifically the endplate change noted on MRI, when taken together with the mechanism of injury and other circumstances surrounding onset of pain, is all entirely consistent with an injury having occurred at the time of the accident.

[23] Dr Bell continues:

> I am uncertain what evidence the CAP has to support that a sudden onset of back pain in association with lifting a combined weight of 40 kg in a flexed position is a typical history for aggravation of an underlying condition.
>
> …
>
> He does have longstanding gradual onset changes within the lumbar spine including disc narrowing, dehydration, and minor bulging. Such changes are recognised as a normal part of aging and do not constitute "pathology". However the endplate change on MRI, consisting of oedema/inflammatory change on MRI and which can be traumatic in origin related to a single episode of trauma (including heavy lifting), was not necessarily present prior to the accident in question. The only way one could ever be certain of this would have been to have performed an MRI prior to March 2018.

[24] In conclusion, Dr Bell states:

> I believe that the CAP is in error in concluding that the endplate changes noted on Mr Rhodes' MRI cannot be traumatic in origin and I have obtained a specialist musculoskeletal radiologist's opinion that differs from this view. CAP also highlights the fact that normal age related degenerative change is generally asymptomatic, and I agree. Yet at the same time they have stated that the accident has in some way caused it to become symptomatic and yet they have not stated how.
>
> …
>
> I remain firmly of the view that it is more likely than not that Mr Rhodes has sustained a physical injury to the L4/5 disc specifically the endplate, as a result of lifting a combined weight of 40 kgs, and that this injury continues to give rise to pain.

The Appellant's Submissions

[25] Ms Armstrong submits that there is a consensus of medical opinion from Mr Hunter, Mr Castens, Dr Coates, Dr Bell and the CAP that there are inflammatory

changes at the vertebral endplates at L4/5 and these are visible on MRI. Dr Bell describes these changes as a physical injury.

[26] Additionally, Dr Coates and Dr Bell identify an annular fissure at L4/5 and Mr Carstens identifies a disc bulge.

[27] On the issue of causation Ms Armstrong submits that the complete clinical picture must be considered, and the following factors are relevant:

- The mechanism of injury: a heavy lift in a flexed position applies force through the intervertebral discs causing bulging of the endplate and microfractures.
- The sudden onset of pain.
- A popping sensation at the time of the lift.
- No prior pain in the lower back.
- Persistent pain following the accident event.
- Endplate changes can be associated with accident events.
- Changes are isolated to L4/5.
- No oedema at other levels.

[28] She submits that flaws arise in the CAP analysis on account of the absence of a radiologist or musculoskeletal physician.

[29] She disagrees with the CAP's theory that there was no traumatic cause of the injury to the disc. She refers to the analysis of Dr Bell, Dr Coates and Mr Castens that the forces on the discs were considerable when the appellant lifted 40 kgs while in flexion.

[30] She notes that the CAP does not explain why the underlying back pathology is isolated to L4/5.

[31] She notes that the CAP relied on general statements about causation including that the appellant had an active degenerative disease without providing specific evidence.

[32] She also notes the absence of pain prior to the lifting event; the sudden onset of pain at the time of the lifting event; the popping sensation at the time of the lifting event; and the persistence of pain following the lifting event, provide a picture of a traumatic event that caused injury.

The Respondent's Submissions

[33] Mr Clarke-Parker refers to the Court of Appeal decision in *ACC v Ambros*[1] where the Court said:

> [67] … however, a Court may only draw a valid inference based on facts supported by the evidence and not on the basis of supposition or conjecture … Judges should ground their assessment of causation on their view of what constitutes a normal course of events, which should be based on the whole of the lay, medical and statistical evidence, and not be limited to expert witness evidence.
>
> …
>
> It must however always be borne in mind that there must be sufficient material pointing to proof of causation on the balance of probabilities for a Court to draw even a robust inference on causation. Risk of causation does not suffice.

[34] Mr Clarke-Parker also refers to other relevant principles that:

[a] Although a temporal link is insufficient to establish causation, it may be a significant piece of evidence.[2]

[35] He also refers to *Gordon v ACC*[3] where the Court said that care should be taken in assessing evidence where it is claimed that a minor injury has had major or chronic consequences, where there is another apparent cause for those consequences.

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1 *Accident Compensation Corporation v Ambros.* [2007] NZCA 304; [2008] 1 NZLR 340.
2 *Accident Compensation Corporation v Mehrtens* [2012] NZACC 250 at paragraph [48].
3 *Gordon v Accident Compensation Corporation* [2013] NZACC 154 at paragraph [68].

[36] He also refers to the statement of principle in *McDonald v ARCIC*[4] where the High Court said the fact that it is the event of an accident which renders symptomatic that which previously was asymptomatic does not alter the basic principle that pre-existing degenerative changes are not covered.

[37] He submits that the evidence does not establish that Mr Rhodes' ongoing back pain is eligible for cover:

[a] Mr Rhodes' condition is unrelated to the lumbar sprain, for which he has cover;

[b] Imaging shows features that the CAP considered were degenerative change, not traumatic, in Mr Rhodes's back and Mr Bell goes no further to identify as possibly traumatic;

[c] Mr Rhodes's position is the accident caused harm to the L4/5 level of his lumbar spine, however it is not clear this is the source of the pain;

[d] Back pain is common, does not always arise from trauma, and commonly arises from gradual degeneration of the back, sometimes brought on by ordinary activities of daily living;

[e] The pattern of pain (onset after the accident) is equally consistent with aggravation of a pre-existing condition as it is with a traumatic injury;

[f] The mechanism of injury – an action Mr Rhodes repeatedly carried out is consistent with a pre-existing degeneration becoming symptomatic rather than a one-off traumatic injury causing lasting damage.

[38] Mr Clarke-Parker notes that the CAP found there was no evidence of any distinct actual damage or abnormal functioning arising from the March 2018 accident that supports the diagnosis of a long-term physical injury.

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[4] *McDonald v Accident Rehabilitation and Compensation Insurance Corporation* [2002] NZAR 970.

[39] The CAP said that lower lumbar levels, such as L4/5, are affected first because they deal with the greatest daily stresses at the bottom of the spinal column and there are also subtle changes at the L3/4 disc level i.e. illustrating wider degeneration.

[40] Mr Clarke-Parker notes that while the injury could be caused by the accident event, that does not resolve the issue in dispute. As *Ambros* says, "risk of causation does not suffice".

[41] He also notes that the CAP considered that the results of the CT discogram cast doubt on whether the L4/5 level was the pain generator. He also notes that Dr Bell appears to agree that the discogram is not an accurate representation of whether or not the L4/5 disc is the source of the pain.

[42] Mr Clarke-Parker refers to the fact that disc degeneration is common in the absence of trauma due to genetic, mechanical or lifestyle factors and the CAP confirms that up to 35% of 20 to 39 year olds have disc degenerative changes on their MRI scans without any symptoms at all.

[43] Mr Clarke-Parker also refers to the CAP statement:

> Mr Rhodes' history recorded in the contemporary notes indicates that he had longstanding intermittent low back pain and that he developed a particularly bad episode on 05/03/2018 which led to him seeking medical assistance.

[44] He further submits the CAP statement, that the likelihood of an accident of the nature described by Mr Rhodes causing long term injury resulting in ongoing low back pain is low, is consistent with Mr Rhodes' evidence that he carried out this task many times before.

[45] Mr Clarke-Parker also refers to the evidence of radiologist, Dr Coates, which identifies only the possibility of a causal relationship.

[46] Ultimately, Mr Clarke-Parker submits that even the appellant's expert agrees that some features of the appellant's back show degenerative rather than traumatic changes and goes no further than asserting that the changes could be traumatic.

[47] He submits that:

> The careful and thoroughly referenced CAP report shows there is insufficient evidence to establish, on the balance of probabilities, that the condition for which treatment is sought was caused by the March 2018 accident.

Decision

[48] On 5 March 2018 the appellant lifted two 20 kg containers of liquid and as the GP's notes of the following day note:

> Like has done 100's times, but felt more than heard a crack like something go in lower back – pain, unable to lift – tried work this am but can't lift cardboard. Pain in low Lx spine and to R side but not L. Gets pain when sitting.

[49] The GP note also includes:

> Back injury few months ago but was better – ok.

[50] On the same day, 5 March 2018, a claim form was completed and lodged. The appellant was 37 years old at the time. According to the CAP report, that would place him as having a 35% chance of disc degeneration not caused by trauma.

[51] On 9 March the appellant again saw his GP. The GP note says:

> Not much better if any. Still sore, can't do much, slow taking pain meds … pain just at low Lx spine.
>
> O/E – sore esp getting up out of chair, and also if tries to squat, has difficulty getting up again. … tender sore at L5/S1.
>
> Little better – slow to improve … back looks weak.

[52] A further consultation on 22 March 2018 records:

> LBP started to improve last Thu. …
>
> O/E sore back like before pain on transferring sit/stand, sore to move … imp – relapse acute LBP (lower back pain).

[53] On 3 April 2018 his GP noted that the appellant's lower back pain feels quite a lot better.

[54] On 21 June 2018 some three months after the injuring event, an MRI was performed. The report concluded:

> Spondylosis at L4-5 including Schmorl's node endplate changes and associated endplate oedema. Diffuse posterior disc bulge at this level but no canal stenosis or nerve root compromise.
>
> The rest of the appellant's discs were normal.

[55] Orthopaedic Surgeon Mr Carstens in his Assessment Report and Treatment Plan of 9 May 2019 said that there was a direct causal medical link between the lifting event and the appellant's back injury now requiring surgery.

[56] In clinical opinion dated 11 July 2019, Mr Hunter said that the medical evidence did not support a causal link between the appellant's accident and the condition requiring surgery and "the condition to be treated is primarily gradual process degenerative disc disease which has become symptomatic". This is commented on by Mr Carstens in his report of 9 February 2020 where he says:

> The neighbouring levels L3/4, L5/S1 as well as proximal sites show satisfactory hydration of the disc and if one was to assume that there is a degenerative condition at play then you would expect these levels would also be affected on his imaging studies.

[57] Mr Hunter comments again on 17 February 2020 and alters his position somewhat, acknowledging that the injury "possibly" caused the appellant's condition.

[58] The appellant then sought the opinion of Dr Bell, who in a report of 7 August 2020, noted that the mechanism of injury described by the appellant is recognised as a potential cause of injury to a lumbar and vertebral disc. He also noted that studies have demonstrated that disc failure can be associated with a sudden, "popping" sensation as experienced by the appellant.

[59] Dr Bell states:

> However taking into account all of the circumstances regarding the development of back pain and the injury itself, it is more likely than not that these changes particularly inflammatory endplate change were not present prior.

[60] In response the Clinical Advisory Panel, in its report of 14 October 2020 explained:

> ….. lumbar spondylosis and disc degeneration are almost universal. They have complex, multifactorial causes. The tissue weakening occurs gradually, related to genetic inheritance, aging, nutritional compromise, and loading history.
>
> …
>
> The predominant cause of such failure, however is not lifting 2 x 20 litre containers, but rather the tissue weakening that has come before.

[61] The Clinical Advisory Panel went on to say that the prevalence of low back pain is found in 84% of the adult population. Such a broad statistic is of little value given that elsewhere the Clinical Advisory Panel acknowledges that those in the appellant's age group have a 35% chance of disc degeneration not caused by trauma.

[62] The Clinical Advisory Panel also noted:

> Mr Rhodes's history recorded in the contemporary notes indicates that he had longstanding intermittent low back pain.

[63] However, I was unable to find, in the agreed bundle of documents, any reference to long standing intermittent low back pain.

[64] I note there was a brief radiological report from 2 August 2017, following an accident where the appellant slipped on a hard floor and fell and had ongoing central lumbosacral spine pain. Stated under the heading 'findings':

> No bony or joint abnormality is seen.

[65] This contrasts with the findings of the MRI relating to the L4/5 disc on 21 June 2018, some three months after the accident. It is also noted that an initial x-ray taken on 27 March 2018 noted satisfactory preservation of vertebral disc space heights and vertebral body heights.

[66] The CAP takes a view that the inflammatory changes at the vertebral endplates were most likely due to Mr Rhodes's active degenerative disease. In essence therefore ACC's position, informed by Mr Hunter and the CAP, is that the appellant's condition was degenerative. However, both Mr Hunter and the CAP are

less than categorical, with Mr Hunter acknowledging a possibility of traumatic cause and the CAP being of the opinion that degeneration was the "predominant cause" of the appellant's condition.

[67] The final report is from Dr Bell, who took the further step of discussing the appellant's imaging with Dr Coates. Dr Coates indicated that there is evidence of an annulus fissure which the Clinical Advisory Panel acknowledges would be the outcome of disc injury.

[68] While Dr Bell acknowledges that the appellant does have longstanding gradual onset changes with the lumbar spine, he says that the endplate change on the MRI, consisting of oedema/inflammatory change, which can be traumatic in origin, "was not necessarily present prior to the accident in question".

[69] I therefore conclude that the report of the Clinical Advisory Panel as well as the report of Mr Hunter, are less than categorical as to the cause of the appellant's L4/5 condition.

[70] The matter is finely balanced. What, in my view, tips that balance in favour of a traumatic origin of the appellant's L4/5 condition, is the further comment from Dr Coates, referred to in Dr Bell's report of 26 November 2020, that there was evidence of an annulus fissure. The absence of such was referred to by the CAP as contraindicating a disc injury.

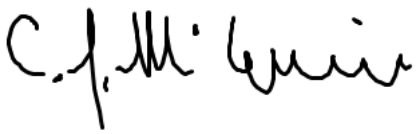
[71] Therefore the balance tips in favour of a traumatic origin of the appellant's L4/5 condition. I therefore conclude that the appellant has proved on the balance of probabilities, that the accident of 5 March 2018 caused the injury to his L4/5 disc.

[72] Accordingly, the appeal is allowed and the decision of the respondent of 12 July 2019 declining to pay for surgery is reversed.

[73] I note that the appellant also seeks a direction that the respondent assess his eligibility for weekly compensation arising from the injury of 5 March 2018. That is outside the parameters of his appeal. It is a matter that needs to be taken up with the

Corporation by the appellant. That way any decisions would be amenable to review and appeal.

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



Judge C J McGuire
District Court Judge

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