

5

FORM 15

RECORD OF APPEAL
THE STATE OF SOUTH CAROLINA
IN THE COURT OF APPEALS
[IN THE SUPREME COURT]

APPEAL FROM SOUTH CAROLINA WORKER'S
COMPENSTATION COMMISSION

COMMISSIONERS: Melody James, Chair, Aisha Taylor, Avery Wilkerson

Case No. 2017-001027

RECEIVED

Seeahray Brailsford, Employee, Claimant,.....Appellant, FEB 05 2018

v.

Court of Appeals

Piggly Wiggly Carolina Company, Inc., Employer, and Constitution
State Service Company as TPA for Greenbax Enterprise Inc., Carrier, Respondents.

RECORD ON APPEAL

Seeahray Brailsford
322 Rice Road
Newberry, S. C. 29108
(803) 276-0001
Employee, Claimant, Appellant
Self Represent

Michael E. Chase
Carmlo Barone Sammataro
P. O. Box 1473
Columbia, S. C. 29202
(803) 254-2200
Attorney for Respondent

I N D E X

Copy of Transcript of Proceeding, January 23, 2017, p. 1-22

Decision and order, p. 23-31

Story of Why and Picture, p. 32-38

Rotator Cuff Tears, p. 39-43

What is Primary Biliary Cirrhosis, p. 44-53

Autoimmune Diseases, p. 54-59

Depression – Older Adults, p. 56-59

SOUTH CAROLINA WORKERS' COMPENSATION COMMISSION

W.C.C. FILE #1019167

RECEIVED

MAY 25 2017

SC Court of Appeals

SEEAHRAY BRAILSFORD)
CLAIMANT,)
VS.)
PIGGLY WIGGLY)
EMPLOYER,)
AND)
GREENBAX ENTERPRISES)
CARRIER.)

SC WORKERS' COMPENSATION COMMISSION

FULL BOARD HEARING OF

SEEAHRAY BRAILSFORD

VS

PIGGLY WIGGLY

This is the Transcript of the South Carolina Workers' Compensation Full Board Hearing of Seeahray Brailsford versus Piggly wiggly, taken before Gloria Davis, a Court Reporter and Notary Public in and for the State of South Carolina, commencing at the hour of 1:32 P.M., Monday, January 23, 2017, at South Carolina Workers' Compensation Commission, 1333 Main Street Columbia, South Carolina.

COPY

REPORTED

BY

GLORIA DAVIS

PALMETTO COURT REPORTING
321 Mac Circle Lexington, SC 29073
(803) 358-0515
www.PalmettoCourtReporting.com

APPEARANCES

FOR THE APPELLANT:
Pro se

FOR THE RESPONDENT:
Michael E. Chase, Esquire
Turner Padgett Graham & Laney, PA
1901 Main Street, 17th Floor
P.O. Box 1473
Columbia, SC 29202

ALSO PRESENT:

Mr. George Rogers

COMMISSIONERS PRESENT:

Melody James, Chair
Aisha Taylor
Avery Wilkerson

*Reporter's Note: -- Indicates incomplete thought or sentence, trailing off or interruption by speakers.

**This is an Official Transcript governed by 25A S.C. Code ANN. Regs 67-615 D (Supp. 2000). The unauthorized copying of this document is deemed to be conversion of an asset belonging to the Court Reporter.

I N D E X

	Page
Argument for the Appellant by Ms. Brailsford	6:6
Argument for the Respondent by Mr. Chase	14:5
Reply for the Appellant by Ms. Brailsford	17:19
Certificate	22

STIPULATIONS

Commissioner James: All right. Madame Court Reporter, if you will call the case.

Court Reporter: Today is January 23rd, 2017. This is South Carolina Workers Compensation case number 1019167. This is the case of Seeahray Brailsford, the Claimant, versus Piggly wiggly, the Employer, Greenbax Enterprises is the Carrier. The Appellant is the pro se Claimant. The Respondent is represented by Michael E. Chase. Each side is allowed ten (10) minutes for argument and the Appellant three (3) minutes in reply. You are requested to argue the grounds of exception and stay within the record. The Single Hearing Commissioner was Commissioner Beck.

Commissioner James: All right. And, Ms. Brailsford, we had a discussion before we went on the record about your right to an attorney and also about the time that would be allotted for -- for the argument today. You responded to me that you do not wish to exercise your right to an attorney; is that correct?

Ms. Brailsford: Correct.

Commissioner James: Okay. So, you're going to be representing yourself in today's hearing,

1 correct?

2 Ms. Brailsford: Yes.

3 Commissioner James: And I also advised you
4 there is always some danger to
5 self-representation; you understand that?

6 Ms. Brailsford: Yes.

7 Commissioner James: All right. And you
8 still wish to proceed on your own?

9 Ms. Brailsford: Yes.

10 Commissioner James: All right. Then, you
11 can begin when you're ready, ma'am.

12 Ms. Brailsford: Can I ask a question?

13 Commissioner James: Yes, ma'am.

14 Ms. Brailsford: The question I have is, why
15 do you send people to a Workers' Comp doctor if
16 the doctor tells you what to do and then it's not
17 followed through?

18 Commissioner James: All right. Ma'am, you
19 can present that as part of your argument and your
20 position.

21 Ms. Brailsford: Okay.

22 Commissioner James: I can answer questions
23 on procedure but as to what actually happened in
24 this particular case, the two (2) of you all will
25 have to present that to us and what your -- what

1 your -- what your relative positions are, okay?

2 Ms. Brailsford: Okay.

3 Commissioner James: So, you can begin with
4 your ten (10) minutes, ma'am.

5 ARGUMENT FOR THE APPELLANT

6 By Ms. Brailsford:

7 well, I feel that -- wait a minute.

8 Mr. Rogers: She's not feeling well today;
9 today is one of her bad days.

10 Commissioner James: Okay.

11 Mr. Rogers: Would I be allowed to speak for
12 her? I'm her husband.

13 Commissioner James: No, sir. We can't allow
14 that. The Courts have ruled that if we allowed
15 you to do that it would be the unauthorized
16 practice of law; so, we can't do that. If she is
17 feeling physically unable today, then we can
18 discuss whether or not there needs to be a
19 continuance for today's hearing.

20 Mr. Rogers: Well, this goes on most of the
21 time. She is very, very, very, very sick.

22 Commissioner James: All right. Well --

23 Mr. Rogers: It's a liver disease and she
24 went from two hundred and fourteen (214) pounds --

25 Commissioner James: -- all right. Well --

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21
22
23
24
25

all right, this --

Mr. Rogers: -- to ninety-four (94) pounds.

Commissioner James: -- all right. Mr. Brailsford, just one minute. Ms. Brailsford, do you feel like -- like you can proceed today or do you need -- do you need a moment, do you need a --

Mr. Rogers: If you can talk, talk.

Ms. Brailsford: Okay..

Commissioner James: -- that's -- it's up to you. I don't want you to proceed if you're not feeling well today. We can --

Ms. Brailsford: No.

Commissioner James: -- we can talk about continuing it.

Ms. Brailsford: No. I'd just like to get it done.

Commissioner James: That -- that --

Mr. Rogers: One of the problems she has is she can't talk very long.

Commissioner James: -- all right. Then we'll adjust the time some for her in that case, okay?

Ms. Brailsford: Okay.

Commissioner James: So -- but it's standard is ten (10) minutes but what we'll do is we'll --

1 we'll adjust a little bit. I can't -- I can't
2 adjust it a ton but, again, we'll -- we'll
3 compensate for that some but if you are not
4 feeling up to this physically or emotionally, then
5 you let us know.

6 Ms. Brailsford: Okay.

7 Commissioner James: But we're going to
8 proceed then unless you tell me otherwise.

9 Ms. Brailsford: Okay.

10 Commissioner James: All right. You can go
11 ahead.

12 Ms. Brailsford: Well, I feel like it's unjust
13 because I am -- I can't even put -- I can't think.
14 Hold on a minute.

15 Mr. Rogers: Just tell them that when Dr.
16 Lovelace ordered the MM -- MRI they said no down
17 here and you want to know why and -- and --

18 Ms. Brailsford: What do I do, tell you about
19 what happened or why I am appealing it?

20 Commissioner James: Yes, ma'am, why you're
21 appealing.

22 Ms. Brailsford: Why I'm appealing it? Okay.
23 Because -- like the depression, I was never depressed
24 before that and then the thing I got back it says
25 depression was part of my illness; I got that after I

1 got injured and was out so long. And then I'd have to
2 fight for everything to get done; there was a wait for
3 everything. And then, I have a crack in my bone up
4 here (indicating) and that is related to the injury
5 because in Dr. Lee's notes, he's answering Workers'
6 Comp, if this crack in the bone is part of the original
7 injury and he said yes and it -- it was ruled that it's
8 not included.

9 Mr. Rogers: And your liver disease.

10 Ms. Brailsford: And then, I got the liver disease
11 from all this worry. And the -- the big bruise I had
12 on my arm, they -- the doctor explained it to us that
13 this is all part of it because I've had three (3)
14 surgeries, plus all the physical therapy, plus all the
15 medications, plus the constant pressure on me for
16 stress. And he -- they said that that's what led to
17 the liver disease and they're saying that's not part of
18 it either. All they want to take care of is the
19 shoulder. But if I fall and this bone cracks, it's
20 still not all the way healed, I still have a small
21 crack but there's nothing I can do; I have to let it
22 go. But if this -- if I should fall and land here
23 it'll break that and there's nothing they can do.

24 Mr. Rogers: Tell them about your injury.

25 Ms. Brailsford: I -- what happened was, I fell

1 and I broke my femur in three (3) spots. And then, I
2 ended up with having osteoporosis, which they said is
3 caused from all the other problems I've had but just
4 like cascaded down from this injury. But the -- the
5 main thing is, taking care of my shoulder and my arm,
6 which was ruled as part of it and the --

7 Mr. Rogers: The liver.

8 Ms. Brailsford: -- the liver and also, you -- you
9 send paperwork for the physical therapy to fill out and
10 test you and see how you perform and stuff and on the
11 back of that, the very last page, he says I can not
12 work. And they were asking me if I had looked for a
13 job. Well, he states right there I can't look -- I
14 can't work; I'm not workable. I have no other income
15 but Social Security.

16 Commissioner Taylor: Ms. Brailsford, at the
17 hearing below did you present any evidence, like
18 any reports that associated your liver disease to
19 the -- to the shoulder accident?

20 Mr. Rogers: We can't hear -- we can't hear
21 you.

22 Ms. Brailsford: I'm sorry?

23 Commissioner Taylor: Oh. At the hearing
24 below, did you present any evidence, any medical
25 records that tie in the liver disease to the

1 shoulder injury?

2 Mr. Rogers: We have them.

3 Commissioner Taylor: Did --

4 Ms. Brailsford: No, not --

5 Commissioner Taylor: -- did you present it
6 at the hearing before?

7 Ms. Brailsford: -- no.

8 Mr. Rogers: No.

9 Commissioner Taylor: Okay.

10 Ms. Brailsford: We -- we --

11 Mr. Rogers: We have all the evidence --

12 Commissioner Taylor: Okay.

13 Mr. Rogers: -- regarding --

14 Commissioner Taylor: And sir, I've got to
15 tell you you can't speak; I need her to answer my
16 questions, okay?

17 Ms. Brailsford: -- okay.

18 Mr. Rogers: -- well --

19 Commissioner Taylor: And she can -- she can
20 understand me.

21 Mr. Rogers: -- I'm the one that compiled it;
22 so, she doesn't know everything I've got.

23 Commissioner Taylor: I understand that but
24 that's --

25 Ms. Brailsford: Okay.

1 Commissioner Taylor: -- okay. It's her
2 claim. It's her workers' compensation claim.

3 Ms. Brailsford: We got -- went on the Internet
4 and we found all this information about this liver
5 disease I have and they all say the same thing, I have
6 --

7 Mr. Rogers: In the Comp.

8 Ms. Brailsford: -- to be a trigger to start it
9 and the trigger is this big bruise and my shoulder.
10 And what it says is that the T cells will run to the
11 shoulder. And they can't fix it; they don't know
12 what's wrong. So, they get confused. They don't
13 understand why yet but they get confused. And then
14 they'll go and they'll think, well, that's not it and
15 they'll go somewhere else. But since I was taking so
16 much medicine and the anesthesia and everything, they
17 went right to the liver assuming that that was the
18 problem. And what they do is they close the duct and
19 that's how I got the sclerosis of the liver, we think.
20 But they all say it transpires from all of this stress
21 on the shoulder. And the tracking is from this -- from
22 the stress. I had three (3) surgeries, all the therapy
23 and we're -- Dr. Lee said that the weight of the muscle
24 pulled down on the shoulder and cracked it. He said it
25 is kind of rare but it does happen. He said since I

1 had three (3), like, right in a row it was a lot of
2 stress on the shoulder and that's what cracked it. But
3 it's in their own notes where he replies to them and
4 tells them but yet they're saying no.

5 Commissioner James: You have four (4)
6 minutes left; do you have anything else you want
7 to tell us, ma'am? And we have your -- we have
8 your written brief and all of the records from the
9 hearing below.

10 Ms. Brailsford: Okay.

11 Commissioner James: So, we do have your
12 written position --

13 Ms. Brailsford: Okay.

14 Commissioner James: -- and we -- we will be
15 reviewing that.

16 Ms. Brailsford: All right.

17 Commissioner Wilkerson: Thank you.

18 Ms. Brailsford: I have all kinds of
19 information if you need anything else.

20 Commissioner James: We -- we have -- we are
21 restricted to whatever the record is below.

22 Ms. Brailsford: Okay.

23 Commissioner James: So, we have -- we have
24 all of that --

25 Ms. Brailsford: Okay.

1 Commissioner James: -- okay? All right.

2 Mr. Chase.

3 Mr. Chase: Thank you, Commissioner.

4 ARGUMENT FROM THE RESPONDENT

5 By Mr. Chase:

6 May it please the Commission. This is a -- this
7 was a how much case as far as permanent partial
8 disability to a single scheduled member, the left
9 shoulder, the -- at the hearing with Commissioner Beck.
10 Under Section 42-9-30 it's our position today that the
11 ruling by Commissioner Beck is supported by a
12 preponderance of evidence and that there was no error
13 of law in his decision. Before I make just a few
14 comments, and I -- I really don't have many to make,
15 Commissioners, I did want to point out a little bit of
16 explanation with the award that Commissioner Beck made
17 in this case. And as the Commissioners have seen in
18 the order it is seventy percent (70%) permanent partial
19 disability to the Claimant's shoulder, subject to the
20 five hundred (500) week limitation. And what I wanted
21 to point out was at the time of the hearing
22 approximately two hundred and ninety (290) weeks of
23 temporary total disability had been paid at that point
24 in time. So, to do that math for you Commissioners,
25 five hundred (500) weeks, the maximum, less the two

1 ninety (290), comes out to two hundred and ten (210)
2 weeks. So, if you work that on to the shoulder, that's
3 seventy percent (70%) of the shoulder that was left on
4 the balance of the maximum that she could get under the
5 Workers' Compensation Act of five hundred (500) weeks.
6 So, we're here today on an appeal of a case that was --
7 the issue was the amount of permanency to that body
8 part, the left shoulder. So, I have just a few
9 comments to make; really it's all in the record. Dr.
10 Lee was the Claimant's final surgeon. She was with Dr.
11 Holmes to start with. Dr. Lee gave a forty-six point
12 seven percent (46.7%) rating in the case, impairment
13 rating. We were there on a stop pay application. We
14 were seeking credit for overpayment. We were given
15 some credit for overpayment but only back to the date
16 that we filed our stop pay hearing request, our Form
17 21; that filing date was July of 2016, July 25th. The
18 hearing in this case was August 1st of 2016. Now, we
19 were seeking credit all the way back to when Dr. Lee
20 released the Claimant, we didn't get that at the single
21 Commissioner Hearing, we got from when we filed for our
22 stop pay hearing request; we have not appealed that
23 issue. The Claimant was also given, in addition to the
24 seventy percent (70%) permanent partial disability to
25 the shoulder, lifetime causally related medical

1 treatment for her left shoulder and that does include
2 the hardware that she has in her shoulder. Just a
3 couple of points from the Single Commissioner Hearing
4 that are in the record of this case. The Claimant is
5 no longer taking any prescription medication at all for
6 the shoulder; that was introduced. She has not been
7 back to Dr. Lee, the surgeon, since he released her,
8 that was all the back in -- on July the 29th of 2014;
9 so, she has not been back to see him since then. The
10 Claimant does not know -- owe any medical bills,
11 whatsoever, from the left shoulder treatment provided
12 by my clients and those include medical bills and
13 treatment from Dr. Holmes, who I've mention already,
14 Dr. Lee, who was final -- her final surgeon. But in --
15 in addition to that, Commissioners, my clients paid for
16 her to see Dr. Fowble for a second opinion and also a
17 third opinion with a Dr. Hess in her case, as well as
18 substantial physical therapy with Newberry Therapists.
19 At the time of the Single Commissioner Hearing, the
20 Claimant was not seeking benefits for any depression,
21 which she is discussing today; in fact, that's on page
22 20 of the record or the hearing transcript and she was
23 asked specifically by Commissioner Beck whether or not
24 she was claiming depression as part of her claim and
25 she indicated she was not at that time. There was no

1 evidence introduced, anything with the liver or
2 anything with the depression at the time of the
3 hearing. So, with all respect, we ask that you affirm
4 the decision below. There has been no error of law and
5 the findings of fact are supported by a preponderance
6 of the evidence. Thank you.

7 Commissioner James: Ms. Brailsford, you have
8 three (3) minutes to reply if you have anything
9 you wish to say, ma'am.

10 Mr. Rogers: I didn't under -- I couldn't
11 hear it.

12 Commissioner James: You have three (3)
13 minutes to reply if you have --

14 Mr. Rogers: Tell them that -- that it's
15 wrong. That -- it -- it -- the liver was formed
16 by your shoulder not being cared for.

17 Commissioner James: All right.

18 REPLY FOR THE APPELLANT

19 By Ms. Brailsford:

20 We don't understand because the liver came about
21 because of the shoulder and I didn't -- we didn't know
22 to bring evidence the first time so we brought it all
23 this time because we thought it would be given a
24 different sentence. But we just don't -- it's kind of
25 a waste of time.

1 Mr. Rogers: That's what the shoulder looked
2 like when you guys down here stopped the MRI.
3 Nobody understands why you stopped the MRI. We
4 have contacted Tim Scott in Washington, Senator
5 Cromer --

6 Commissioner James: Sir, I don't mean to --
7 to stop you because I know you want to talk and --
8 but I have to because you can talk to her and
9 you've done it a couple of times.

10 Mr. Rogers: -- okay.

11 Commissioner James: But I can't --

12 Mr. Rogers: Ask them why they stopped the
13 MRI --

14 Commissioner James: -- I can't --

15 Mr. Rogers: -- ask them why they did it.

16 Ms. Brailsford: Okay. why did --

17 Commissioner James: -- let -- let me explain
18 to you why you have to stop you. I can't let you
19 advocate for her because it's the unauthorized
20 practice of law. You can talk to her just like
21 you've done a couple of times if you want to
22 suggest some things to her but --

23 Mr. Rogers: Ask them why they stopped the
24 MRI.

25 Ms. Brailsford: Okay. why didn't -- why

1 didn't they do it from the beginning, give me the
2 MRI?

3 Commissioner James: -- all right. Ma'am,
4 and I'm assuming you're talking about the Carrier.

5 Ms. Brailsford: Right.

6 Commissioner James: So -- all right. And if
7 that issue was in front of Commissioner Beck, then
8 we have that issue in front of us, okay?

9 Ms. Brailsford: Okay.

10 Mr. Rogers: Well, tell them about the States
11 Attorney's Office, that you went there and they
12 said you had been terribly wronged.

13 Ms. Brailsford: Well, we -- like I've said, we've
14 called -- we've talked to everybody and they don't
15 understand this either. And the VA's Office, Mrs.
16 Rogers, she said that they feel that I was justly
17 mistreated but, of course, there was nothing they
18 could do. So, they would pass this to other
19 people and they all say the same thing but nobody
20 wants to do anything about it.

21 Commissioner James: All right. Thank you
22 all. This concludes the hearing.

23 Court Reporter: Mr. Brailsford, could I get
24 your full name, please, since you were a speaker?

25 Ms. Brailsford: He can't hear you.

1 Court Reporter: Could I get your full name
2 please since you were a speaker?

3 Mr. Rogers: Mine?

4 Court Reporter: Yes, sir.

5 Mr. Rogers: Why do you want my name?

6 Court Reporter: Because you spoke I have to
7 --

8 Mr. Rogers: George Rogers.

9 Court Reporter: -- I thought you were Mr.
10 Brailsford?

11 Mr. Rogers: I'm George Rogers.

12 Commissioner James: All right. You will get
13 a written set of instructions when a decision has
14 been made; it will be made in the next ten (10)
15 days and, of course, it may take a little longer
16 for it to be sent out but you'll get a copy of
17 that and then should you prevail then it -- it'll
18 be assigned to probably the staff attorney to
19 draft and should Mr. Chase prevail it will be
20 assigned to him but you'll get something in the
21 next couple of weeks, okay?

22 Mr. Rogers: May I just say why we don't have
23 an attorney?

24 Commissioner James: No, sir. You're not
25 allowed to speak for her.

1 Mr. Rogers: Tell them why.

2 Commissioner James: All right.

3 Ms. Brailsford: The reason I don't have an
4 attorney is because they all say the same thing,
5 there's nothing you can do.

6 Commissioner James: All right.

7 Ms. Brailsford: The law needs to be changed,
8 period; it's not fair to the person that's
9 injured.

10 Commissioner James: All right. And -- and

11 --

12 Mr. Chase: Thank you, Commissioner.

13 Commissioner James: -- thank you all.

14 Ms. Brailsford: Thank you.

15 (THERE BEING NO FURTHER ARGUMENTS, THE HEARING CONCLUDED AT
16 1:52 P.M.)

1 STATE OF SOUTH CAROLINA)

2 COUNTY OF RICHLAND)

CERTIFICATE

3
4 Be it known that I took the foregoing South Carolina
5 Workers' Compensation Commission Full Board Hearing of
6 Seeahray Brailsford, vs. Piggly Wiggly;

7 That I was then and there a Notary Public in and for
8 the State of South Carolina-At-Large;

9 That by virtue thereof I was duly authorized to
10 administer an oath;

11 That the witness was first duly sworn to testify the
12 truth, the whole truth, and nothing but the truth,
13 concerning the matter in controversy aforesaid;

14 The foregoing transcript of 21 typewritten pages
15 represents a true, accurate and complete transcription of
16 the testimony so given at the time and place aforesaid to
17 the best of my skill and ability;

18 That I am not related to nor an employee of any of the
19 parties hereto, nor a relative or employee of any attorney
20 or counsel employed by the parties hereto, nor interested in
21 the outcome of this action;

22 witness my hand and seal this 18th Day of February,
23 2017.

24 Gloria G. Davis

25 Gloria G. Davis

Notary Public for South Carolina

My Commission Expires 12/21/21

*This Transcript may contain quoted material, such material
is reproduced as read or quoted by the speaker.

**This is an Official Transcript governed by 25A S.C. Code
Ann. Regs. 67-615 D (Supp. 2000). The unauthorized copying
of this document is deemed to be conversion of an asset
belonging to the Court Reporter.

**APPELLATE PANEL
DECISION AND ORDER
OF THE
SOUTH CAROLINA WORKERS' COMPENSATION COMMISSION**

SCWCC FILE NO. 1019167

Seeahray Brailsford, Employee,

Claimant/Appellant,

v.

Piggly Wiggly Carolina Company, Inc., Employer,

and

Constitution State Service Company
as TPA for Greenbax Enterprise Inc., Carrier

Defendants/Respondents.

Appellate Panel Review held in Columbia,
South Carolina on January 23, 2017 per
notices timely and properly served on all
parties of interest.

Appellate Panel Decision and Order filed
March ____, 2017

ARGUMENTS BRIEFED BY:

Seeahray Brailsford representing herself *pro se*
for Claimant/Appellant

Michael E. Chase, Esquire, of Columbia, South Carolina
for Defendants/Respondents

STATEMENT OF THE CASE

The parties were heard by Commissioner T. Scott Beck, Hearing Commissioner, in Columbia, South Carolina on August 1, 2016. On October 11, 2016, Commissioner Beck issued the following Findings of Fact, Conclusions of Law, and Order:

FINDINGS OF FACT

1. The Claimant sustained an accidental injury to her left shoulder only on November 23, 2010, which arose out of and in the course of employment with Piggly Wiggly. This finding is based upon the pleadings, the testimony rendered, and the medical evidence submitted.
2. The Claimant treated with Dr. Wendell Holmes, and he performed shoulder surgery on March 9, 2011 (Def. APA 56-57). The Claimant continued to have shoulder issues and received a second opinion, paid for by the Defendants, with Dr. David Lee, who performed a second shoulder surgery on February 15, 2012 (Def. APA 181-184). The Claimant continued to have ongoing shoulder issues and eventually had a reverse total shoulder arthroplasty (Def. APA 188-190). As a result of this last procedure, the Claimant has prosthetics in her shoulder. This finding is based upon the testimony rendered and the medical evidence submitted.
3. The Claimant did not sustain any other injuries by accident arising out of and in the course of employment on the date in question, including but not limited to, her left arm, left elbow, and depression. In addition, the Claimant failed to show a causal connection between her left shoulder injury and subsequent treatment for her liver issue and other claimed infections she contends that she suffered from while in the hospital. This finding is based upon the testimony rendered and the medical evidence submitted.
4. Per stipulations made by the parties, the Claimant earned an average weekly wage of \$537.18, and, therefore, the applicable compensation rate is \$358.14.

5. The Claimant reached maximum medical improvement for her single scheduled member shoulder injury on July 29, 2014 (DEF APA 229). She was assigned a converted impairment rating of 46.7% to the left shoulder (DEF APA 229). She did not receive any other impairment ratings as a result of this accident.
6. Defendants are entitled to stop payment of temporary total disability benefits and are entitled to a credit for temporary total disability benefits paid beyond July 25, 2016, the date which the Defendants filed their Form 21.
7. The Claimant is entitled to 70% permanent partial disability to the left shoulder, less credit for TTD overpayment, and subject to the maximum total benefits of 500 weeks compensation under the Act, which may limit her net recovery.
8. The Claimant is awarded future lifetime medical care limited to the repair, maintenance, or replacement of causally related medical hardware for the left shoulder only pursuant to S.C. Code Ann. Section 42-15-60.

CONCLUSIONS OF LAW

1. The Claimant suffered an admitted compensable injury to the left shoulder only on November 23, 2010. This conclusion is based upon findings of fact 1-5, and South Carolina Code Ann. Section 42-1-160.
2. The Claimant reached maximum medical improvement for the left shoulder on July 29, 2014. This conclusion is based upon findings of fact 1-5.
3. The Defendants are entitled to stop payment of temporary total disability benefits and are entitled to a credit for overpayment of temporary total disability benefits back to the date of filing the Form 21 on July 25, 2016 pursuant to South Carolina Code Ann. Section 42-9-10 and the regulations.

4. The Claimant is entitled to an award of 70% to the left shoulder. This conclusion is based upon finding of facts 1-7, and South Carolina Code Ann. Section 42-9-30 for a single scheduled member injury.
5. The award of 70% to the shoulder in this case for permanent partial disability is subject to, and may be limited by, the maximum period of compensation of 500 weeks allowable under the Act pursuant to section 42-9-10 and other sections of the Act. Therefore, claimant's net award may be less than 70% of the shoulder depending on the number of weeks of temporary total disability benefits paid.
6. The Claimant is entitled to lifetime causally related medical expenses limited to repair, maintenance or replacement of the hardware in the Claimant's left shoulder only. This conclusion is based upon South Carolina Code Ann. Section 42-15-60.

AWARD

IT IS HEREBY ORDERED AND ADJUDGED that the Claimant sustained a compensable injury to the left shoulder only on November 23, 2010 and reached maximum medical improvement on July 29, 2014;

IT IS FURTHER ORDERED AND ADJUDGED that Defendants are entitled to stop payment of temporary total disability benefits and that Defendants are entitled to a credit for temporary total disability benefits paid beyond July 25, 2016, the date of filing the Form 21.

IT IS FURTHER ORDERED AND ADJUDGED that the Claimant is awarded 70% permanent partial disability benefits to the left shoulder, less credit for overpayment of TTD, and subject to the 500 week limitation of maximum compensation benefits.

IT IS FURTHER ORDERED AND ADJUDGED that the Claimant is awarded future medical care limited to repair, maintenance, or replacement of causally related medical hardware in the left shoulder.

IT IS SO ORDERED.

Within the statutory period, the Claimant filed an Application for Review in this case, setting forth the reasons for an appeal. A copy of this Application was furnished to all interested parties prior to Appellate Review.

All proper testimony has been taken. Together with all documentary evidence and a transcript of the hearing, appellate briefs were delivered to the individual members of the South Carolina Workers' Compensation Appellate Panel. Oral Argument has been provided to the individual members of the Full Commission and the case has since been under study and consideration.

By appeal, Claimant/Appellant submits the following:

1. The Single Commissioner erred as a matter of fact and law in finding that the claim was limited to a single schedule member injury.
2. The Single Commissioner erred as a matter of fact and law in determining that the Claimant did not suffer from depression resulting from this claim.
3. The Single Commissioner erred as a matter of fact and law in determining that the Claimant did not suffer from a compensable liver disease.

On the other hand, Defendants/Respondents contend that the Appellant only sustained a left shoulder injury on the date in question. Respondents deny that the Appellant sustained compensable injuries to her liver or psyche. Respondents relied on the medical evidence which released the Appellant with a converted 46.7 percent impairment rating to the left shoulder on

July 29, 2014 by Dr. Lee. (Hearing Tr. p. 15). Respondents further contended that the Appellant was sent to two additional doctors for additional opinions. (Hearing Tr. p. 16).

In addition, Respondents contended that the depression claim was not raised and that the Appellant specifically denied claiming depression as part of her claim to the Single Commissioner. (Hearing Tr. p. 16). Lastly, Respondents contended that the Appellant did not produce any evidence regarding her liver to the Single Commissioner. (Hearing Tr. p. 17). Therefore, the Respondents requested that the Full Commission affirm the decision below and find that there has been no error of law and that the findings of fact were supported by the preponderance of the evidence.

Based upon a review of the Record, Briefs, and oral argument, the Panel, by a **UNANIMOUS** vote, **AFFIRMS** the Decision and Order of the Hearing Commissioner dated April 2, 2012 on the basis that it is supported by a preponderance of the evidence, and contains no error of law. Accordingly, the Findings of Fact and Conclusions of Law set forth below shall become and hereby are the law of the case:

FINDINGS OF FACT

1. The Claimant sustained an accidental injury to her left shoulder only on November 23, 2010, which arose out of and in the course of employment with Piggly Wiggly. This finding is based upon the pleadings, the testimony rendered, and the medical evidence submitted.
2. The Claimant treated with Dr. Wendell Holmes, and he performed shoulder surgery on March 9, 2011 (Def. APA 56-57). The Claimant continued to have shoulder issues and received a second opinion, paid for by the Defendants, with Dr. David Lee, who performed a second shoulder surgery on February 15, 2012 (Def. APA 181-184). The Claimant continued to have ongoing shoulder issues and eventually had a reverse total shoulder arthroplasty (Def. APA 188-

190). As a result of this last procedure, the Claimant has prosthetics in her shoulder. This finding is based upon the testimony rendered and the medical evidence submitted.

3. The Claimant did not sustain any other injuries by accident arising out of and in the course of employment on the date in question, including but not limited to, her left arm, left elbow, and depression. In addition, the Claimant failed to show a causal connection between her left shoulder injury and subsequent treatment for her liver issue and other claimed infections she contends that she suffered from while in the hospital. This finding is based upon the testimony rendered and the medical evidence submitted.

4. Per stipulations made by the parties, the Claimant earned an average weekly wage of \$537.18, and, therefore, the applicable compensation rate is \$358.14.

5. The Claimant reached maximum medical improvement for her single scheduled member shoulder injury on July 29, 2014 (DEF APA 229). She was assigned a converted impairment rating of 46.7% to the left shoulder (DEF APA 229). She did not receive any other impairment ratings as a result of this accident.

6. Defendants are entitled to stop payment of temporary total disability benefits and are entitled to a credit for temporary total disability benefits paid beyond July 25, 2016, the date which the Defendants filed their Form 21.

7. The Claimant is entitled to 70% permanent partial disability to the left shoulder, less credit for TTD overpayment, and subject to the maximum total benefits of 500 weeks compensation under the Act, which may limit her net recovery.

8. The Claimant is awarded future lifetime medical care limited to the repair, maintenance, or replacement of causally related medical hardware for the left shoulder only pursuant to S.C. Code Ann. Section 42-15-60.

CONCLUSIONS OF LAW

1. The Claimant suffered an admitted compensable injury to the left shoulder only on November 23, 2010. This conclusion is based upon findings of fact 1-5, and South Carolina Code Ann. Section 42-1-160.
2. The Claimant reached maximum medical improvement for the left shoulder on July 29, 2014. This conclusion is based upon findings of fact 1-5.
3. The Defendants are entitled to stop payment of temporary total disability benefits and are entitled to a credit for overpayment of temporary total disability benefits back to the date of filing the Form 21 on July 25, 2016 pursuant to South Carolina Code Ann. Section 42-9-10 and the regulations.
4. The Claimant is entitled to an award of 70% to the left shoulder. This conclusion is based upon finding of facts 1-7, and South Carolina Code Ann. Section 42-9-30 for a single scheduled member injury.
5. The award of 70% to the shoulder in this case for permanent partial disability is subject to, and may be limited by, the maximum period of compensation of 500 weeks allowable under the Act pursuant to section 42-9-10 and other sections of the Act. Therefore, claimant's net award may be less than 70% of the shoulder depending on the number of weeks of temporary total disability benefits paid.
6. The Claimant is entitled to lifetime causally related medical expenses limited to repair, maintenance or replacement of the hardware in the Claimant's left shoulder only. This conclusion is based upon South Carolina Code Ann. Section 42-15-60.

ORDER

Based on the foregoing Findings of Fact and Conclusions of Law:

IT IS HEREBY ORDERED that the Order of the Hearing Commissioner filed in the above-captioned matter on October 11, 2016, is hereby affirmed by the Full Commission Appellate Panel on the basis that it is supported by a preponderance of evidence and contains no error of law. The claim for a compensable psychological issue and liver disease is denied in its entirety.

AND IT IS SO ORDERED.

**SOUTH CAROLINA WORKERS'
COMPENSATION COMMISSION**

Melody L. James, Commissioner
On Behalf of the Appellate Panel

FULL AFFIRMATION

Concur:

Avery B. Wilkerson, Jr., Commissioner

Aisha Taylor, Commissioner

Why?

My name is Seeahray Brailsford. I was the deli/bakery manager for the Piggly Wiggly store in Prosperity SC for 7 years and in November of 2010, while at work, I injured my left shoulder by tearing the rotator cuff. I reported the injury and was sent to Dr. Oscar Lovelace who was the workers compensation doctor for Piggly Wiggly at the time. Dr. Lovelace noted my shoulder had a rotator cuff tear but he did not know how extensive the tear was. He also ordered an MRI because my arm was swollen, I was bruised from the shoulder to past my elbow, and my fingers were cold and blue. He scheduled the MRI for the next day.

The next day Dr. Lovelace's office called and told me that Travelers Insurance (workers comp) had said no to the MRI. I went back to see Dr. Lovelace and he wanted me to start physical therapy so the arm would not get stiff, but once again Travelers Insurance said no to the physical therapy. I went back to Dr. Lovelace a week later and he then told me he wanted me to see an orthopedic surgeon in Newberry, he reordered the MRI, and physical therapy. Travelers Insurance said no again. Why?

On one visit to Dr. Lovelace's office a nurse with Travelers Insurance came in with me to the office. Dr. Lovelace let the nurse know that he was not happy with Travelers Insurance, he did not understand why every time he requested an order Travelers Insurance would refuse the request. He was so upset that he told the nurse that the proper procedure was xrays or other tests, physical therapy and then orthopedic surgery. He told her that Travelers Insurance did not follow protocol and asked her to leave.

Finally late in December of 2010 about 40 days after the accident Dr. Lovelace's office called me to set up an appointment for an MRI. I was working with such arm pain that I could not lift anything and my fingers were still swollen and blue. I continued to work because I did not want to lose my job. After a couple of weeks I went back to get the results of the MRI. Dr. Lovelace told me at that time that my rotator cuff was torn very badly in the left shoulder. He then reordered me to see an orthopedic surgeon. Travelers Insurance said no again. And the entire time I was working and trying to save my job. Why?

In January 2011 I finally was able to see the orthopedic surgeon. Travelers Insurance sent me to see a Dr. Holmes. Dr. Holmes read the MRI and also said the rotator cuff was torn

2

very badly. At first he did not want to do the surgery because too much time had passed since my accident.

I did not get a surgery date until February 2011. Dr. Holmes office called and stated that the earliest date was in March 2011. I asked why so long and that I was hurting very badly. Dr. Holmes nurse asked me when the accident occurred and when I told them they agreed it had been a long time and that if they had a cancelation that they would get me in earlier. I called the Mayo clinic and was informed that someone my age, 63, that the repair should be done within 7 to 10 days. But I am still working in terrible pain, swollen and blue fingers. I did not want to lose my job or position.

I finally got a surgery date in March 2011. It is 107 days after my accident. I started physical therapy the next day after surgery. I kept my appointments through all the pain, I could not lift my arm, it was still very swollen and my fingers were still blue. Every time I went back to see Dr. Holmes I told him about my concerns with blue fingers, numbness and my arm still being swollen. On the last visit with Dr. Holmes told me to stop worrying about the arm and that he didn't know what else to do with me.

I decided to get a second opinion. I went to see Dr. David Lee in January 2012. He asked for post op xrays but none were taken. Dr. Lee took them in his office. He told me the results were not good. He would need to redo the surgery but we needed to wait for approval from Travelers Insurance. I finally got the ok in February 2012 for surgery. Meanwhile I was sent for another MRI with dye. As I was lying on the table I heard the doctor and the nurse talking and heard him say that he could not see where anything had been done at all or it had completely come undone.

I was in the OR when the doctor informed my husband that he could not do the micro surgery, that it would take major surgery because there was so much scare tissue and damage that he would have to clean up first and that he could not guarantee that the surgery would hold because too much time and damage had passed. After 4 weeks I started physical therapy. I was still having a very hard time, still in a lot of pain, fingers are still swollen and blue. Dr. Lee ordered another xray and informed me that the surgery did not hold and that the only thing left to do was shoulder replacement. Meanwhile, I am still working. Travelers Insurance agreed to the

surgery and it was set up for October 2012. Now, I am almost 2 years from the date of my accident in November of 2010.

From the time of the 3rd surgery I could feel myself not well. My energy level was failing. I went to see Dr. Lovelace and he treated me for depression. I had the 3rd surgery but was unable to start physical therapy for 6 to 8 weeks. I restarted physical therapy for the third time. One day I had a very sharp pain in my shoulder. I called Dr. Lee and made an appointment. He took xrays and said nothing showed up but that he was treating this like a fracture. He told me to come back in 2 weeks. After 2 weeks I went back for more xrays and at this time he said there was a crack in the shoulder. He sent me home again to return in 1 week. I then took yet another xray and Dr. Lee informed me that the crack went all the way through the bone and that it would take a very long time to heal. He told me that this happens due to the shoulder bones being very weak from 3 surgeries and the weight of the muscle. He also told me that if it did not heal that he would have to put screws in my shoulder to keep the bones together. Dr. Lee told me at this time he would order a bone stimulator to help heal the bone. It took over 6 months to get the stimulator. I even hired an attorney but Mr. Pope couldn't even get the stimulator any faster. I did not understand how it could take so long. Why?

In the meantime I was getting even sicker. It could have been from depression, but my body could not physically function. I cried a lot. I was unable to do normal things around my house. I did not understand why I felt so bad.

After several months of physical therapy I went back to see Dr. Lee and at this time he said he unfortunately could not do anything else for me. The damage from the start of waiting too long and too much time had passed before getting the first surgery.

I was then fired by letter from Piggly Wiggly. I was never called or received one letter or card to wish me a speedy recovery or even to see how I was doing after 7 years of work and dedication. That included hours of improving the department, going to classes and I really enjoyed my job. It was all over Travelers Insurance not giving me the ok for the MRI back in November of 2010. The outcome is a total of 91% disability in my left shoulder and arm.

After several months, my attorney Mr. Pope, Travelers Insurance's attorney, Mr. Chase, and Mr. Merchant (mediator) all met with me to try to settle this matter before we went in front

of the insurance commissioner. I told them I was too sick to come in but Mr. Pope insisted that I had to be there. I arrived at 11:30a, I was very sick and my skin was a yellow green color. I was scared and I didn't know what was wrong with me. We met altogether the first few minutes. Mr. Chase (Piggly Wiggly attorney) apologized to me on behalf of Piggly Wiggly for the treatment from and neglect that I have suffered.

I was still at the office at 4:30p and was still very ill. I told them repeatedly that I had to go home that I was sick. My husband came through the door and was shocked that I was still there and of my appearance. There was no way I was going to take \$73,400 for what they did to me. I told my husband I was not happy with this and he took me home.

The next day I went to the doctor and he put me into the hospital immediately. I had surgery the next day. Dr. Jones asked my husband what happened. My husband explained everything to him from the accident in 2010 all the way to the meeting that lasted 5 hours. Dr. Jones wondered how they couldn't see how sick I was. I was in ICU for 4 days. I had feeding tubes, tubes draining fluids from my liver, saline, antibiotics, pain meds and oxygen. I came very close to dying. The doctor told my husband it would be touch and go for few days.

I was to go to a meeting on March 23, 2015. I was still in the hospital and they wanted a letter from Dr. Jones stating why I couldn't be there. I was on my death bed and they wanted a letter. They wanted me to sign for a settlement and I was still in the hospital. My husband kept telling them that I was sick. Finally Dr. Jones said NO visitors unless approved. I lost 110 lbs in 4 months. Due to the surgery they found out that I got a liver disease. Primary bilateral cirrhosis in the hospital. The most embarrassing part was that Mr. Pope (my attorney) sent me the letter and the contract for me to sign. I would have thought that he would have taken up for me.

I went home from the hospital at the end of March 2015 with a feeding tube. I could not stand or walk. I was in terrible shape and pain. They kept wanting me to sign the contract. I was very sick and on a lot of medications. A nurse came by 3 times a week and a physical therapist to start. The nurse would take vitals, check the feeding tubes, clean me up and changed dressing.

The attorneys continuously tried to get me to sign the contract. My husband read the contract, we let several people we know read the contract including other attorneys and even law

(5)

enforcement officials and they have all said that it was the worst thing they had ever read. I would not sign it and I still have not signed the contract.

I was told by a doctor that it may be a good idea to write to our Governor Nikki Hailey, for she may not be aware of what has happened with workers compensation or the actions of Travelers Insurance. I wrote a letter much like this one explaining my situation and the events since November of 2010 until now. I have not heard from her, no letter, nothing acknowledging that they had received it at all. I really needed some advice so I went to the DA's office. Mrs. Rogers stated that I was terribly wronged and told me to contact the Lt. Governor. I called at least 14 times. No letter, no return phone call or any kind of response.

At this time I contacted Mrs. Valentine who is over the workers compensation. Her reply to me was that it takes time for treatment. Why? I did not get a sorry for any problems I am having and she didn't even take the time to answer any of my questions. Next I called Senator Cromer's office. He told me that workers comp on Assembly St in Columbia hung up on him.

You don't have to be a high graduate to understand why big companies want to come to SC. As a right to work state, they can treat employees like dirt. They can fire you for no reason. Workers comp can treat a hurt employee any way they want and obviously nothing can be done about it. I intend to ask the public to sign a petition for the way I was treated. I say let's get rid of big businesses who don't give a damn about their injured employees. Like Senator Cromer said nothing can be done for me but my story may help someone else.

It amazes me that everyone I have spoken to including the DA's office, Mr. Rogers, Mr. Pope, Piggly Wiggly and their attorney Mr. Chase, Dr. Lee, Senator Cromer and other attorneys all say the same thing, I was treated awful!!! But no one knows what to do to help me.

Because of the cost of an MRI that Travelers Insurance was contracted to fulfill by Piggly Wiggly, I have lost my job which most importantly includes being able to care for myself, I lost my health, I lost my life and health insurance, and I am missing out on my 401k which is my retirement that I will not ever be able to enjoy now. This is all due to Travelers Insurance not doing what they were supposed to do. The quality of my life now is very poor and I still have no one to help me. Why? I loved my job, took great pride in working and now I can't. I wonder as

6

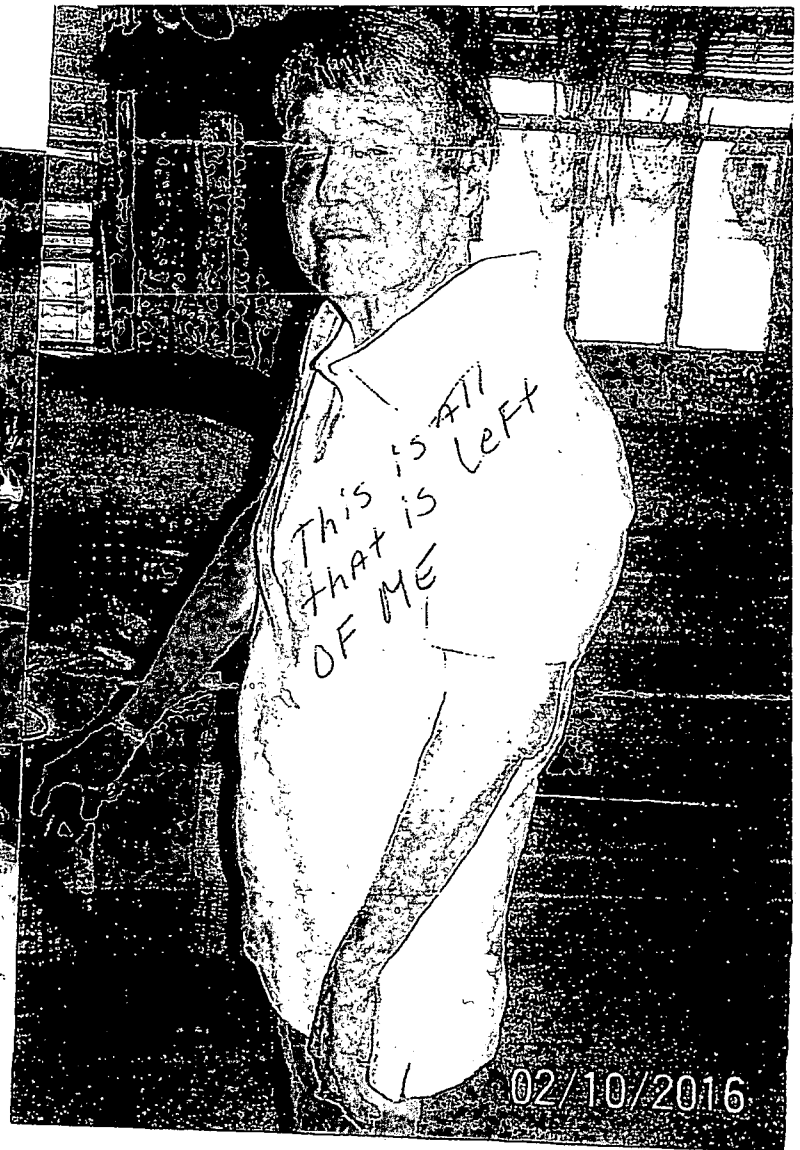
I lay here if anyone else has went through this. Do firemen, police officers or teachers or their families would have to wait 107 days for treatment or an operation. Why? Again just why?

Sincerely,

Seeahray Brailsford



-This is before Accident when I WAS manager of two depts.



02/10/2016



30/11/2010

This is what happened and workers comp did not give me medical care for 107 days and did not get a MRI for 33 days after accident

OrthoInfo

Your connection to expert orthopaedic information

Rotator Cuff Tears

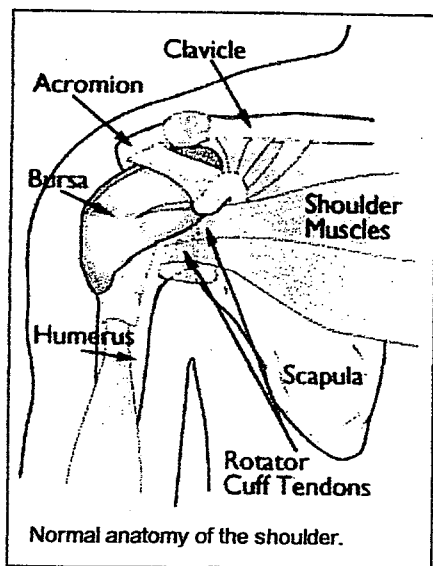
Information on rotator cuff tears is also available in Spanish: *Desgarres del manguito de los rotadores* ([topic.cfm?topic=A00604](http://orthoinfo.aaos.org/topic.cfm?topic=A00604)).

A rotator cuff tear is a common cause of pain and disability among adults. In 2008, close to 2 million people in the United States went to their doctors because of a rotator cuff problem.

A torn rotator cuff will weaken your shoulder. This means that many daily activities, like combing your hair or getting dressed, may become painful and difficult to do.

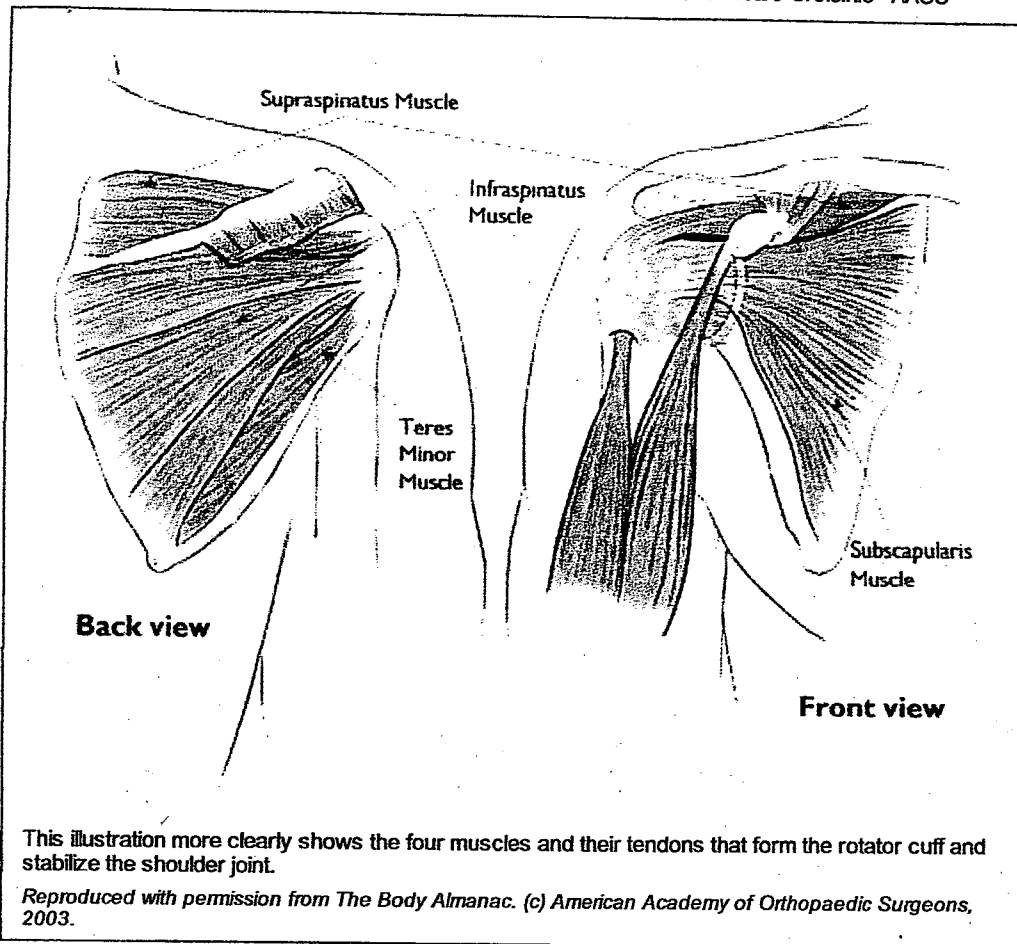
Anatomy

Your shoulder is made up of three bones: your upper arm bone (humerus), your shoulder blade (scapula), and your collarbone (clavicle). The shoulder is a ball-and-socket joint: The ball, or head, of your upper arm bone fits into a shallow socket in your shoulder blade.



Your arm is kept in your shoulder socket by your rotator cuff. The rotator cuff is a network of four muscles that come together as tendons to form a covering around the head of the humerus. The rotator cuff attaches the humerus to the shoulder blade and helps to lift and rotate your arm.

There is a lubricating sac called a bursa between the rotator cuff and the bone on top of your shoulder (acromion). The bursa allows the rotator cuff tendons to glide freely when you move your arm. When the rotator cuff tendons are injured or damaged, this bursa can also become inflamed and painful.



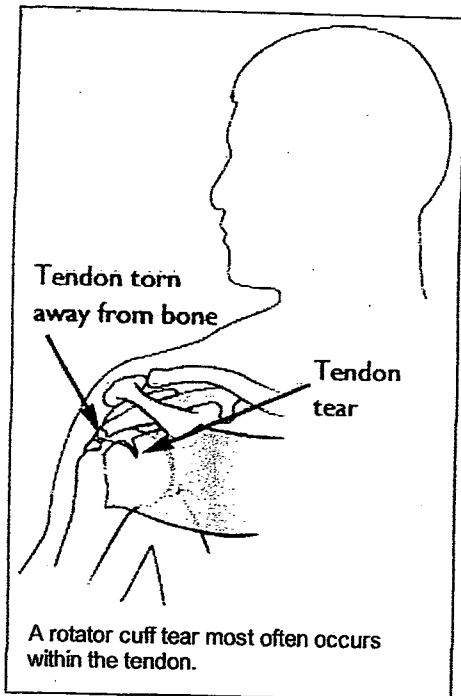
Description

When one or more of the rotator cuff tendons is torn, the tendon no longer fully attaches to the head of the humerus. Most tears occur in the supraspinatus muscle and tendon, but other parts of the rotator cuff may also be involved.

In many cases, torn tendons begin by fraying. As the damage progresses, the tendon can completely tear, sometimes with lifting a heavy object.

There are different types of tears.

- **Partial Tear.** This type of tear damages the soft tissue, but does not completely sever it.
- **Full-Thickness Tear.** This type of tear is also called a complete tear. It splits the soft tissue into two pieces. In many cases, tendons tear off where they attach to the head of the humerus. With a full-thickness tear, there is basically a hole in the tendon.



Cause

There are two main causes of rotator cuff tears: injury and degeneration.

Acute Tear

If you fall down on your outstretched arm or lift something too heavy with a jerking motion, you can tear your rotator cuff. This type of tear can occur with other shoulder injuries, such as a broken collarbone or dislocated shoulder.

Degenerative Tear

Most tears are the result of a wearing down of the tendon that occurs slowly over time. This degeneration naturally occurs as we age. Rotator cuff tears are more common in the dominant arm. If you have a degenerative tear in one shoulder, there is a greater risk for a rotator cuff tear in the opposite shoulder — even if you have no pain in that shoulder.

Several factors contribute to degenerative, or chronic, rotator cuff tears.

- **Repetitive stress.** Repeating the same shoulder motions again and again can stress your rotator cuff muscles and tendons. Baseball, tennis, rowing, and weightlifting are examples of sports activities that can put you at risk for overuse tears. Many jobs and routine chores can cause overuse tears, as well.
- **Lack of blood supply.** As we get older, the blood supply in our rotator cuff tendons lessens. Without a good blood supply, the body's natural ability to repair tendon damage is impaired. This can ultimately lead to a tendon tear.
- **Bone spurs.** As we age, bone spurs (bone overgrowth) often develop on the underside of the acromion bone. When we lift our arms, the spurs rub on the rotator cuff tendon. This condition is called shoulder impingement, and over time will weaken the tendon and make it more likely to tear.

Risk Factors

Because most rotator cuff tears are largely caused by the normal wear and tear that goes along with aging, people over 40 are at greater risk.

People who do repetitive lifting or overhead activities are also at risk for rotator cuff tears. Athletes are especially vulnerable to overuse tears, particularly tennis players and baseball pitchers. Painters, carpenters, and others who do overhead work also have a greater chance for tears.

Although overuse tears caused by sports activity or overhead work also occur in younger people, most tears in young adults are caused by a traumatic injury, like a fall.

Symptoms

The most common symptoms of a rotator cuff tear include:

- Pain at rest and at night, particularly if lying on the affected shoulder
- Pain when lifting and lowering your arm or with specific movements
- Weakness when lifting or rotating your arm
- Crepitus or crackling sensation when moving your shoulder in certain positions

Tears that happen suddenly, such as from a fall, usually cause intense pain. There may be a snapping sensation and immediate weakness in your upper arm.

Tears that develop slowly due to overuse also cause pain and arm weakness. You may have pain in the shoulder when you lift your arm to the side, or pain that moves down your arm. At first, the pain may be mild and only present when lifting your arm over your head, such as reaching into a cupboard. Over-the-counter medication, such as aspirin or ibuprofen, may relieve the pain at first.

Over time, the pain may become more noticeable at rest, and no longer goes away with medications. You may have pain when you lie on the painful side at night. The pain and weakness in the shoulder may make routine activities such as combing your hair or reaching behind your back more difficult.



Doctor Examination

Medical History and Physical Examination

After discussing your symptoms and medical history, your doctor will examine your shoulder. He or she will check to see whether it is tender in any area or whether there is a deformity. To measure the range of motion of your shoulder, your doctor will have you move your arm in several different directions. He or she will also test your arm strength.

Your doctor will check for other problems with your shoulder joint. He or she may also examine your neck to make sure that the pain is not coming from a "pinched nerve," and to rule out other conditions, such as arthritis.

Imaging Tests

Other tests which may help your doctor confirm your diagnosis include:

- **X-rays.** The first imaging tests performed are usually x-rays. Because x-rays do not show the soft tissues of your shoulder like the rotator cuff, plain x-rays of a shoulder with rotator cuff pain are usually normal or may show a small bone spur.
- **Magnetic resonance imaging (MRI) or ultrasound.** These studies can better show soft tissues like the rotator cuff tendons. They can show the rotator cuff tear, as well as where the tear is located within the tendon and the size of the tear. An MRI can also give your doctor a better idea of how "old" or "new" a tear is because it can show the quality of the rotator cuff muscles.



Reproduced with permission from JF Sarwark, ed: *Essentials of Musculoskeletal Care*, ed 4. Rosemont, IL, American Academy of Orthopaedic Surgeons, 2010.

Treatment

If you have a rotator cuff tear and you keep using it despite increasing pain, you may cause further damage. A rotator cuff tear can get larger over time.

Chronic shoulder and arm pain are good reasons to see your doctor. Early treatment can prevent your symptoms from getting worse. It will also get you back to your normal routine that much quicker.

The goal of any treatment is to reduce pain and restore function. There are several treatment options for a rotator cuff tear, and the best option is different for every person. In planning your treatment, your doctor will consider your age, activity level, general health, and the type of tear you have.

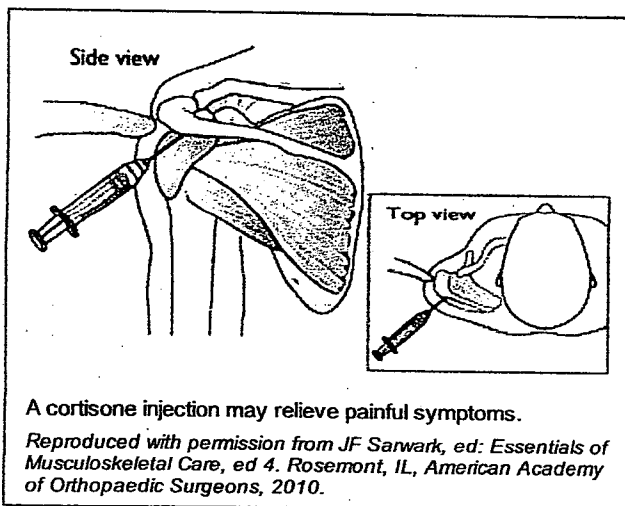
There is no evidence of better results from surgery performed near the time of injury versus later on. For this reason, many doctors first recommend nonsurgical management of rotator cuff tears.

Nonsurgical Treatment

In about 50% of patients, nonsurgical treatment relieves pain and improves function in the shoulder. Shoulder strength, however, does not usually improve without surgery.

Nonsurgical treatment options may include:

- **Rest.** Your doctor may suggest rest and limiting overhead activities. He or she may also prescribe a sling to help protect your shoulder and keep it still.
- **Activity modification.** Avoid activities that cause shoulder pain.
- **Non-steroidal anti-inflammatory medication.** Drugs like ibuprofen and naproxen reduce pain and swelling.
- **Strengthening exercises and physical therapy.** Specific exercises will restore movement and strengthen your shoulder. Your exercise program will include stretches to improve flexibility and range of motion. Strengthening the muscles that support your shoulder can relieve pain and prevent further injury.
- **Steroid injection.** If rest, medications, and physical therapy do not relieve your pain, an injection of a local anesthetic and a cortisone preparation may be helpful. Cortisone is a very effective anti-inflammatory medicine.



The chief advantage of nonsurgical treatment is that it avoids the major risks of surgery, such as:

- Infection
- Permanent stiffness
- Anesthesia complications
- Sometimes lengthy recovery time

The disadvantages of nonsurgical treatment are:

Print View

« Back to regular page view (/what-is-primary-biliary-cirrhosis-pbc/)

Your printed page will look *something* like this.

<https://www.texasliver.com/what-is-primary-biliary-cirrhosis-pbc/>

What is Primary Biliary Cirrhosis (PBC)?

Primary biliary cirrhosis (PBC) is a disease characterized by inflammatory destruction of the small bile ducts within the liver. PBC eventually leads to cirrhosis of the liver. The cause of PBC is unknown, but because of the presence of autoantibodies, it is generally thought to be an autoimmune disease. Other etiologies, such as infectious agents, have not been completely excluded. PBC has a worldwide prevalence of approximately 5/100,000 and an annual incidence of approximately 6/1,000,000. The prevalence and incidence appear to be similar in different regions of the world. About 90% of patients with PBC are women. Most commonly, the disease is diagnosed in patients between the ages of 40 and 60 years. Most patients with PBC present with pruritus (itching). After pruritus, jaundice (yellow skin caused by bilirubin retention) is the most common presenting symptom. Several patients also present with complaints related to chronic portal hypertension (increased blood pressure in the veins that go to the liver that can lead to symptoms such as bleeding in the esophagus or fluid retention in the abdomen). Some patients are discovered to have PBC during workup of another illness. Since the widespread use of routine serum biochemical analysis, many patients present for evaluation of an elevated serum alkaline phosphatase activity that was detected on laboratory examination.

Patients with PBC have abnormalities in several blood tests. In essentially all patients, the serum alkaline phosphatase and gamma-glutamyltranspeptidase activities are markedly elevated (these are enzymes present in the bile ducts). Serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) activities are usually moderately elevated (these are enzymes made by hepatocytes, the predominant liver cell type). The serum bilirubin concentration is normal early in the disease and rises as the disease progresses (which causes jaundice or yellow skin). Most patients have an elevated serum cholesterol concentration, which is largely contained in an abnormal lipoprotein, termed lipoprotein X, which is produced in patients with bile duct obstruction. The total gamma-globulin concentration is usually normal until late in the disease when cirrhosis develops. Almost all patients with PBC have an elevated serum IgM concentration (a type of antibody). The prothrombin time (a measure of blood clotting) and serum albumin concentration (the major protein in blood that is made in the liver) are normal until cirrhosis develops later in the course of disease.

Serum autoantibodies are of primary importance in the diagnosis of PBC. Antibodies against mitochondria are characteristic of PBC and found in about 90% of patients. About 50% of patients with PBC also have antinuclear antibodies. Antibodies against mitochondrial and nuclear proteins are found in several diseases besides PBC, but the cDNA cloning of several mitochondrial proteins has shown that antibodies against specific antigens are virtually diagnostic of PBC. The same has been shown to be true for antibodies against nuclear proteins in about 25% of patients. Because of the presence of specific autoantibodies, PBC is thought to be an autoimmune disease. Several laboratories around the world are actively involved in determining how the immune response relates to the bile ducts destruction characteristic of the disease. In addition to clinical and laboratory abnormalities and the presence of specific autoantibodies, histological examination (looking at tissue under the microscope) of liver tissue is of central importance in the diagnosis of PBC. Tissue for this purpose is obtained by liver biopsy, which is generally an outpatient procedure. Histologically, PBC is classified into four stages. Stage I is referred to as the florid duct lesion or nonsuppurative destructive cholangitis and is characterized by mononuclear inflammatory cells surrounding a small bile duct. In stage II, there is proliferation of small bile ductules. Stage III is characterized by fibrosis or scarring. Stage IV is cirrhosis. These histological stages demonstrate the progression of the disease

from destruction of the intrahepatic bile duct to fibrosis and cirrhosis. Histological features of more than one stage can be seen on one liver biopsy. Because of sampling differences, the stages can also vary in liver biopsies done at different times on the same patient. In general, however, there is a gradual progression over years from the histological features of stage I to stage IV.

The diagnosis of PBC must be based on a combination of historical, laboratory, serological and histological criteria. In general, patients are middle-aged women who present with pruritus early and jaundice late. Patients that present late in the course of disease may also have signs and symptoms of cirrhosis and hepatic failure. Many patients are referred for evaluation of an isolated elevated serum alkaline phosphatase activity on laboratory testing for other purposes. Essentially all patients have elevated serum alkaline phosphatase and gamma-glutamyltranspeptidase activities. The serum IgM concentration is almost always elevated. About 90% of patients have autoantibodies against specific mitochondrial proteins (the E2 subunits of the oxo-acid dehydrogenase complexes). Approximately 50% of patients have antinuclear antibodies, sometimes against very specific proteins (nuclear pore membrane protein gp210, transcriptional activator Sp100, inner nuclear membrane protein LBR). The absence of an elevated serum IgM concentration and/or specific autoantibodies should place the diagnosis of PBC in doubt. Patients with PBC must have a consistent liver biopsy. The histological findings alone are frequently not diagnostic as the florid duct lesion is often not seen and other features, such as ductular proliferation, fibrosis and biliary cirrhosis, can be seen in other liver diseases.

PBC is a progressive disease that leads to cirrhosis and liver failure. The time from diagnosis to end-stage liver disease can range from a few months to 20 years depending upon when the diagnosis is first made. Several mathematical models based on clinical, laboratory and histological criteria have been devised to predict disease progression. In general, the development of portal hypertension indicates a poor prognosis. The serum bilirubin concentration is the best prognostic indicator of all laboratory values. Once the serum bilirubin concentration reaches 6 mg/dl, the average life expectancy is about 2 years. At this time, patients should be evaluated for possible liver transplantation.

Despite extensive studies, medical therapy has not been shown to have a significant impact in slowing the progression of PBC. Patients with PBC should take vitamins and calcium to help prevent osteoporosis (loss of bone), a common complication of this disease. Colchicine may play a role in inhibiting liver fibrosis and improves laboratory values but not signs or symptoms. D-penicillamine has been studied in several series but the results have shown it to be ineffective and possibly toxic. Various immunosuppressive agents have been studied in patients with PBC. Corticosteroids are probably not effective and may aggravate the osteoporosis commonly present in patients with PBC. Azathioprine (Imuran), methotrexate and cyclosporin A have been examined in several studies and are still being investigated, but these agents will not likely produce radical improvements in clinical course. Ursodiol (Actigall or Urso), a bile acid, has been shown to improve the laboratory and clinical parameters in patients with PBC and the results of one study suggest that it may slow the progression of the disease. Orthotopic liver transplantation is highly successful in patients with end-stage liver disease resulting from PBC.

The eMedicineHealth doctors ask about Primary Biliary Cirrhosis:

Primary Biliary Cirrhosis (PBC) - Diagnosis

How was your case of PBC diagnosed?

Show Your Story

(Page 1 of 6)

Comment from: Teri, 55-64 Female (Patient)

Published: June 24

I was diagnosed with primary biliary cirrhosis (PBC) in 2012 at age 57 when I had my gall bladder removed. Biopsy showed scarring not PBC. Eight months later my primary care physician (PCP) had an ultrasound and it showed cirrhosis. In 8 months, wow! I have bad itching to my head, arms and legs. PCP referred me to a specialist and said for me not to worry. I would probably live for 20 years. I do monthly blood work and am taking vitamin D and hope for the best.

Prz Gastroenterol. 2014; 9(2): 82–87.

Published online 2014 May 5. doi: [10.5114/pg.2014.42502](https://doi.org/10.5114/pg.2014.42502)

PMCID: PMC4108749

Osteoporosis in primary biliary cirrhosis of the liver

Joanna Raszeja-Wyszomirska¹ and Tomasz Miazgowski²¹Department of General, Transplant and Liver Surgery, Medical University of Warsaw, Poland²Department of Hypertension and Internal Medicine, Pomeranian Medical University, Szczecin, Poland

✉ Corresponding author.

Address for correspondence: Prof. Tomasz Miazgowski, Department of Hypertension and Internal Medicine, Pomeranian Medical University, 1 Unii Lubelskiej St, 71-252 Szczecin, Poland. phone/fax: +48 91 425 35 50. e-mail: miazgowski@interia.pl

Received 2012 Jan 8; Revised 2012 Mar 15; Accepted 2012 Apr 2.

Copyright © 2014 Termedia

This is an Open Access article distributed under the terms of the Creative Commons Attribution-Noncommercial 3.0 Unported License, permitting all non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

This article has been cited by other articles in PMC.

Abstract

Go to:

Osteoporosis is a metabolic bone disease associated with a reduction in bone mass and deterioration of bone architecture, leading to increased fragility and subsequent low-trauma fractures in the vertebral column, hip, forearm and other bones. In literature, metabolic bone diseases such as osteoporosis and osteomalacia have been recognised as a complication of chronic liver disease, although the mechanisms of this association remain unclear. An increasing body of research data indicates a strong relationship between osteoporosis and primary biliary cirrhosis (PBC), which mainly results from early diagnosis of the disease, usually when it is still asymptomatic. The incidence of osteoporosis in PBC ranges from 20% to 44% and increases with the progression of the disease. Similarly, the incidence of bone fractures is high in this group of patients (10–20%). In this article, current knowledge on risk factors, pathogenesis, diagnosis and treatment of osteoporosis in PBC is reviewed.

Keywords: primary biliary cirrhosis, osteoporosis

Primary biliary cirrhosis

Go to:

Primary biliary cirrhosis (PBC) is a chronic autoimmune liver disease in which the inflammatory process occurs in the intrahepatic bile ducts. Its incidence is estimated to be 5 : 100,000 persons/year, while the prevalence is 50–250 per million [1]. Primary biliary cirrhosis represents approximately 1% of all cases of cirrhosis; it occurs only in adults, mostly in women and with a peak in the 5th and 6th decades of life. Primary biliary cirrhosis is relatively often found among family members, which is commonly associated with the presence of HLA-DR3, HLA-DR8 and HLA-DR52a antigens. The number of cases of primary biliary cirrhosis is gradually increasing, and nowadays it is usually diagnosed in earlier stages, before fully developed cirrhosis and liver failure. Although the progression of the disease can be significantly inhibited by treatment with ursodeoxycholic acid, a number of patients develop end-stage liver failure requiring an organ transplant. Up to 60% of patients may have no clinical symptoms of PBC; the remainder usually experience chronic fatigue, itching, dryness of the mouth and eyes, and symptoms of associated diseases, often with autoimmune aetiology. Advances in the diagnosis and treatment of patients with PBC are being accompanied by increasing recognition of the related problems of reduced bone mineral density (BMD) and low-trauma osteoporotic fractures.

Hepatic osteodystrophy

Go to:

The term 'hepatic osteodystrophy' was first introduced in 1960 and refers to a metabolic bone disease (MBD) in patients with chronic liver diseases. It used to be believed that MBD occurs only in patients with chronic cholestasis, but it is now known that MBD can also occur in other chronic liver diseases, with a severity dependent on the duration and severity of the liver disease. Hepatic osteodystrophy comprises two types of change in the bone: 1) osteoporosis, which is similar to primary osteoporosis but affects the trabecular bone more often than the cortical bone, and 2) osteomalacia, which predominates in advanced liver disease, particularly those caused by malnutrition [2]. In advanced stages of PBC, both these conditions may overlap, although osteomalacia ("soft" bone) is difficult to diagnose (only through invasive bone biopsy). There are also discrepancies in the assessment of the importance of osteomalacia and osteoporosis as factors leading to hepatic osteodystrophy [2].

Osteoporosis

Go to:

Osteoporosis is a systemic skeletal disease associated with low bone mineral density and impaired spatial structure which increases the risk of low-trauma fractures, especially in the vertebral column, proximal femur and forearm bones. As defined by the World Health Organisation [3], osteoporosis is diagnosed on the basis of a densitometry test result in which bone mineral density (BMD) is lower by at least 2.5 standard deviations (SD) from the peak bone density of healthy young subjects (*T*-score). The term osteopaenia refers to persons with bone mineral density (BMD) between -1 SD and -2.5 SD of the *T*-score.

The commonly recognised risk factors for osteoporosis in the general population include age, female sex, previous fractures, treatment with steroids, smoking and alcohol abuse, low physical activity, low intake of vitamin D and low body weight. For example, the average risk of bone fracture in a 50-year-old Caucasian woman is about 40% compared to only 13% in men of the same age. In addition, a fracture of the femoral epiphysis is associated with a 20% risk of death within 6 months mainly due to pneumonia, venous thrombosis and pulmonary embolism. Low-trauma fractures are the most serious consequence of osteoporosis; regardless of the cause of osteoporosis, vertebral fractures in the lower thoracic and lumbar spine are the most frequent [4]. The systematic increase in the incidence of osteoporosis in the world is associated not only with longer human life, but also with a sedentary lifestyle and low-calcium diet.

Osteoporosis in primary biliary cirrhosis

Go to:

Osteoporosis is a disease that often accompanies PBC. Its prevalence in recent years has increased, which is mainly associated with the increasingly early diagnosis of this liver disease. In cross-sectional studies on patients with PBC, osteoporosis incidence has been diagnosed with densitometry ranges from 20% to 44% (Table I) and increases with the progress of the disease [10, 12]. It is estimated that in the advanced stages of PBC, only about 20% of patients may have normal BMD [11]. However, precise determination of the prevalence of osteoporosis/osteopaenia is difficult because the aforementioned studies evaluate populations differently in terms of gender, race, age or severity of disease, as well as using different diagnostic criteria for the diagnosis of osteoporosis (*t*-score or *z*-score). There are also discrepancies in the literature as to whether osteoporosis occurs in patients with PBC more frequently than in the general population. Guañabens *et al.* observed a greater incidence of osteoporosis in patients with PBC in large groups tested in two studies [10, 13], similar to Mounach *et al.* [14], but contrary to Newton *et al.*, who observed no such relationship [8]. Despite these discrepancies, as well as methodological limitations, studies performed to date suggest that patients with PBC display abnormal densitometry results more often than in the general population. The incidence of bone fractures is equally high in this group of patients (10–20%), although the severity of the disease seems to have no significant effect there [12, 15].

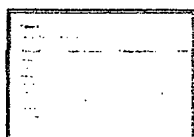


Table I

The incidence of osteoporosis in PBC

Just as in the general population, risk factors for osteoporosis in PBC include age, female gender, smoking, excessive alcohol consumption, underweight (body mass index in adulthood $< 19.0 \text{ kg/m}^2$), early menopause (before 45 years of age), positive family history and treatment with steroids. In addition, patients with PBC often have low physical activity and are more likely to fall [9], and also generally have lower levels of vitamin D than healthy people [16, 17]. Some studies indicate that in contrast to the general population, women with PBC are less predisposed to osteoporosis by menopause than by increased cholestasis and the severity of histological changes in the liver [10].

The pathogenesis of osteoporosis in PBC is suggested to include the influence of both the severity of bone resorption and a slowdown in internal modelling. Bone remodelling is largely controlled by the system: osteoprotegerin (OPG) – receptor activator of nuclear factor $\text{NF-}\kappa\text{B}$ – ligand (RANKL), the role of which has yet to be explained in disorders of bone metabolism in PBC. The RANKL is a transmembrane ligand present on stromal cells and osteoblasts; it binds to RANK, forming a complex that strongly stimulates osteoclasts. Osteoprotegerin, produced for example in the liver, is capable of binding to RANKL as its soluble receptor. This combination prevents the binding of RANKL to RANK, which slows down the pathway of osteoclast maturation and consequently inhibits osteoclastic bone resorption.

In studies on healthy individuals OPG expression increases with age, while other dependencies are less distinct. Patients with osteoporosis are usually observed to have increased OPG, although its low levels have also been found in postmenopausal women with vertebral fractures [16]. Other studies report an increased risk of fractures of the forearm and hip in women with high concentrations of OPG [10]. Some researchers even believe that low RANKL and high OPG levels are good markers of increased risk of all low-trauma fractures [14].

In a few studies, patients with PBC also had elevated levels of OPG and reduced RANKL [18, 19]. It is not known whether that is a primary phenomenon, or rather a consequence of increased bone resorption in the early stages of PBC. It is possible that it is not PBC-specific and can only be regarded as a marker of chronic inflammation [19]. Furthermore, in PBC no link has been found between increased levels of OPG and low RANKL and the risk of osteoporosis or changes in markers of bone remodelling activity [18].

Most studies indicate that the development of osteoporosis in PBC is less associated with a slow down in bone formation than with the severity of bone resorption [13, 20]. The impaired function of osteoblasts may be the effect of cirrhosis-related (i.e. not solely PBC-related) reduction in the production of certain growth factors (especially IGF-1), increased synthesis of oncofoetal fibronectin, or the direct toxic effect of unconjugated bilirubin and lithocholic acid on precursors and osteoblasts [4, 21]. Slowing down of the remodelling process due to impaired bone formation is illustrated in a few studies on PBC that show low levels of osteocalcin [22], a non-collagen protein which is a biochemical marker of osteoclast function. On the other hand, some histomorphometric studies suggest that PBC does not impair bone formation, but, on the contrary, it increases the rate of remodelling and bone resorption [23].

Other factors predisposing to bone metabolism disorders in PBC include malnutrition and vitamin deficiencies, especially vitamins D and K. Reduced hepatic synthesis of vitamin D binding proteins, reduced activity of 25-hydroxylase activity and reduced concentrations of the vitamin D receptor, inducing peripheral resistance to the hormone, may cause secondary hyperparathyroidism, which increases bone resorption and deepens the deficit of calcium ions. In turn, vitamin K deficiency, frequently observed in cholestasis, impairs osteoclast maturation and function. This vitamin inhibits the expression of RANKL [4] and is necessary for the synthesis of osteocalcin. Supplementation with vitamin K prevents the loss of bone in PBC [4].

The importance of genetic factors in disorders of bone metabolism in PBC remains uncertain. Springer *et al.* [14] evaluated the effect of BsmI polymorphism of the vitamin D receptor gene on BMD in patients with PBC. They found only a weak negative correlation with BMD of the lumbar spine, and those results have not yet been confirmed. Other studies in this group of patients tried to establish a relationship between BMD and polymorphism of the insulin growth factor IGF-1 gene and collagen type I gene. However, the results of those studies are inconclusive [15].

Osteoporosis and adipokines

Go to:

Body weight is an important predictor of BMD at different skeletal sites, influencing bone density, for example by mechanical stress (concerning especially cortical bone elements), as well as via hormones and adipose tissue cytokines. Adipokines not only modulate the processes related to diet, energy expenditure and glucose metabolism, insulin sensitivity, free fatty acid oxidation, or reproductive function and regulation of the cardiovascular system, but they also affect bone metabolism. The best known is the effect of leptin on bone mass, which occurs via a specialised population of hypothalamic neurons as well as by receptors of β_2 -adrenergic osteoblasts. Leptin inhibits osteoclastogenesis by reducing the synthesis of RANK and RANKL, and increasing levels of OPG. In one study, leptin levels were significantly lower in women with PBC than in healthy subjects, with no significant correlation found between the risk of osteoporosis and the concentration of leptin, its soluble receptor, liver function test results or the histological severity of liver disease in women with PBC [24]. Similarly reduced leptin levels in the serum of women with PBC and malnutrition were reported by Ben-Ari *et al.* [25], although the leptin levels did not correlate with the severity of the liver disease. García-Suárez *et al.* also reported decreased levels of leptin in PBC, with the degree of reduction significantly associated with the severity of histological changes in the liver [26]. In contrast to these reports, one study showed higher levels of leptin in patients with PBC (class A Child-Pugh score) than in healthy subjects [27]. In view of these conflicting reports, it remains difficult to assess the significance of changes in leptin concentration for the pathogenesis of osteoporosis in PBC.

Adiponectin is another adipokine with a potential effect on bone metabolism; it has an anti-inflammatory effect, enhances insulin sensitivity and increases fatty acid oxidation. In bone, adiponectin stimulates the production of RANKL and inhibits OPG expression in osteoblasts, thereby indirectly promoting osteoclastogenesis. It is postulated that in healthy persons adiponectin influences bone through three primary mechanisms: 1) positive, para/autocrine, 2) negative, direct endocrine and 3) positive, by enhancing the anabolic effect of insulin on bone tissue. The concentration of circulating adiponectin is negatively correlated with BMD and seems to be an independent predictor of low BMD [28–31]. The impact of adiponectin has not yet been assessed with regard to the risk of osteoporosis in PBC. In the only experiment on a group of patients, significantly elevated levels of the hormone were demonstrated regardless of age and body weight [32]. Given the negative relationship between adiponectin and BMD in healthy people, it can be expected that the share of adipokines may be significant in the pathogenesis of osteoporosis in PBC. There is a clear need for further research in this area.

Diagnosis of osteoporosis in primary biliary cirrhosis

Go to:

The primary diagnostic tool for early detection of osteoporosis in PBC is dual X-ray absorptiometry (DXA) of the lumbar spine and proximal femur. In accordance with the recommendations of the European Association for the Study of the Liver, tests should be performed at diagnosis, and then at 1-year intervals [33]. Indications for DXA in PBC are summarised in [Table II](#).

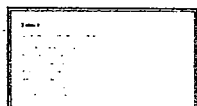


Table II

Indications for densitometric examination in PBC

Treatment

Go to:

Treatment of osteoporosis in PBC poses two major difficulties. Firstly, for several years patients have been qualified for the treatment of primary osteoporosis according to a FRAX calculation of absolute 10-year risk of bone fracture (<http://www.shef.ac.uk/FRAX/tool.jsp>). In addition to the anthropometric and DXA test results (which are not absolutely required), FRAX computes information in which only one element is associated with PBC (questions about diseases predisposing to secondary osteoporosis, including chronic liver diseases). The usefulness of FRAX in PBC has not yet been evaluated. However, regardless of the result of the FRAX calculations, it seems that the use of an absolute risk of fracture > 20% as a threshold for the implementation of therapeutic intervention (established for the general population) is too high in this group of patients. Given the much higher risk of

osteoporosis in PBC than the risk of primary and secondary osteoporosis not accompanied by PBC, it seems more reasonable to perform individual assessment of patients based on the age of onset, duration and severity of a disease, previous treatment with steroids, severity of cholestasis and qualification for a liver transplant. In practice, this means that in PBC diagnosis based on DXA results, osteoporosis therapy should be initiated at an early stage, i.e. in most patients with BMD between -1 SD and -2.5 SD, irrespective of the measurement site. In addition, studies show that the occurrence of fragility fractures is a risk factor for subsequent bone fractures, which that is independent of BMD. This means that after a fracture in the vertebral column, proximal epiphysis of the femoral neck or forearm loco typico fractures, even with an appropriate DXA test result, the patient should be subject to pharmacological treatment of osteoporosis.

Secondly, there is no specific method of treating osteoporosis in PBC, and therefore it is carried out in accordance with general rules. As far as possible, all patients should modify their lifestyle and eating habits; increase physical activity, withdraw stimulants and alcohol, avoid situations with a risk of falls, and increase the supply of calcium in the diet. Unless there are specific contraindications, most patients should introduce calcium and vitamin D supplementation so that the daily intake of calcium (including calcium contained in the diet) is 1000–1500 mg and vitamin D is 400–800 IU per day, preferably monitoring serum concentrations in order to achieve a target value > 30 ng/ml).

To date there have been no well-documented results of clinical trials evaluating fracture-risk efficacy and safety of pharmacological treatments of osteoporosis in PBC. The most frequent treatment includes bisphosphonates, administered orally or intravenously [33–35]. In this group of drugs, the most frequently analysed were etidronate and alendronate in several months of therapy. Alendronate (at 70 mg/week) significantly improves bone mineral density after 1 year of use, with minimal side effects [4]. There is no data on the effect of zoledronic or ibandronic acid on osteoporosis in PBC.

In the only (and pilot) study on raloxifene in PBC patients by Levy *et al.* [36], 9 patients who were given raloxifene exhibited a slight increase in BMD in the lumbar spine. Anabolic therapy with strontium ranelate and recombinant human parahormone (rPTH 1-34) has not yet been used in PBC, although preliminary results of animal studies are promising [37]. Similarly, denosumab – IgG2 monoclonal antibody directed against RANKL – has not yet been used in PBC. Denosumab, similarly to natural immunoglobulin, consists exclusively of amino acids and carbohydrates, and so is not eliminated from the body via the hepatic pathway. Furthermore, because of convenience in drug administration (subcutaneous injection every 6 months), denosumab may be an attractive alternative to bisphosphonate therapy in the prevention and treatment of osteoporosis in PBC [38].

References

Go to:

1. Mazur W. Pierwotna marskość żółciowa wątroby. In: Antczak, Myśliwicz, Pruszczyk, editors. Wielka Interna. Gastroenterologia. 2010. pp. 442–50. [Polish], Medical Tribune Polska.
2. Goel V, Kar P. Hepatic osteodystrophy. Trop Gastroenterol. 2010;31:82–6. [PubMed]
3. Report of a WHO Scientific Group. Prevention and Management of Osteoporosis; 2003. WHO Technical Report Series 921.
4. Parés A, Guanabens N. Osteoporosis in primary biliary cirrhosis: pathogenesis and treatment. Clin Liver Dis. 2008;12:407–24. [PubMed]
5. Springer JE, Cole DE, Rubin LA, et al. Vitamin D-receptor genotypes as independent genetic predictors of decreased bone mineral density in primary biliary cirrhosis. Gastroenterology. 2000;118:145–51. [PubMed]
6. Pares A, Guañabens N, Rodes J. Gene polymorphism as predictors of decreased bone mineral density and osteoporosis in primary biliary cirrhosis. Eur J Gastroenterol Hepatol. 2005;17:311–5. [PubMed]
7. Menon KV, Angulo P, Weston S, et al. Bone disease in primary biliary cirrhosis: independent indicators and rate of progression. J Hepatol. 2001;35:316–23. [PubMed]

8. Newton J, Francis R, Prince M, et al. Osteoporosis in primary biliary cirrhosis revisited. *Gut*. 2001;49:282-7. [PMC free article] [PubMed]
9. Solerio E, Isaia G, Innarella R, et al. Osteoporosis: still a typical complication of primary biliary cirrhosis? *Dig Liver Dis*. 2003;35:339-46. [PubMed]
10. Guañabens N, Pares A, Ros I, et al. Severity of cholestasis and advanced histological stage but not menopausal status are the major risk factors for osteoporosis in primary biliary cirrhosis. *J Hepatol*. 2005;42:573-7. [PubMed]
11. Guichelaar MM, Kendall R, Malinchoc M, Hay JE. Bone mineral density before and after OLT: long-term follow-up and predictive factors. *Liver Transpl*. 2006;12:1390-402. [PubMed]
12. Guañabens N, Cerda D, Monegal A, et al. Low bone mass and severity of cholestasis affect fracture risk in patients with primary biliary cirrhosis. *Gastroenterology*. 2010;138:2348-56. [PubMed]
13. Guañabens N, Parés A, Mariñoso L, et al. Factors influencing the development of metabolic bone disease in primary biliary cirrhosis. *Am J Gastroenterol*. 1990;85:1356-62. [PubMed]
14. Mounach A, Ouzzif Z, Wariaghli G, et al. Primary biliary cirrhosis and osteoporosis: a case-control study. *J Bone Miner Metab*. 2008;26:379-84. [PubMed]
15. Solaymani-Dodaran M, Card TR, Aithal GP, et al. Fracture risk in people with primary biliary cirrhosis: a population based cohort study. *Gastroenterology*. 2006;131:1752-7. [PubMed]
16. Frith J, Kerr S, Robinson L, et al. Primary biliary cirrhosis is associated with falls and significant fall related injury. *QJM*. 2010;103:153-61. [PubMed]
17. Malham M, Jørgensen SP, Ott P, et al. Vitamin D deficiency in cirrhosis relates to liver dysfunction rather than aetiology. *World J Gastroenterol*. 2011;17:922-5. [PMC free article] [PubMed]
18. Guanabens N, Enjuanes A, Alvarez L, et al. High osteoprotegerin serum levels in primary biliary cirrhosis are associated with disease severity but not with *thmRNA* gene expression in liver tissue. *J Bone Miner Metab*. 2009;27:347-54. [PubMed]
19. Szalay F, Hegedus D, Lakatos PL, et al. High serum osteoprotegerin and low RANKL in primary biliary cirrhosis. *J Hepatol*. 2003;38:395-400. [PubMed]
20. Stellon AJ, Webb A, Compston J, Williams R. Low bone turnover state in primary biliary cirrhosis. *Hepatology*. 1987;7:137-42. [PubMed]
21. Guañabens N, Parés A. Management of osteoporosis in liver disease. *Clin Res Hepatol Gastroenterol*. 2011;35:438-45. [PubMed]
22. Guañabens N, Parés A, Alvarez L, et al. Collagen-related markers of bone turnover reflect the severity of liver fibrosis in patients with primary biliary cirrhosis. *J Bone Miner Res*. 1998;13:731-8. [PubMed]
23. Cuthbert JA, Pak CY, Zerwekh JE, et al. Bone disease in primary biliary cirrhosis: increased bone resorption and turnover in the absence of osteoporosis or osteomalacia. *Hepatology*. 1984;4:1-8. [PubMed]
24. Szalay F, Fohoffer A, Horvath A, et al. Serum leptin, soluble leptin receptor, free leptin index and bone mineral density in patients with primary biliary cirrhosis. *Eur J Gastroenterol Hepatol*. 2005;17:923-8. [PubMed]
25. Ben-Ari Z, Schafer Z, Sulkes J, et al. Alterations in serum leptin in chronic liver disease. *Dig Dis Sci*. 2002;47:183-9. [PubMed]
26. García-Suárez C, Crespo J, Fernández-Gil PL, et al. Plasma leptin levels in patients with primary biliary cirrhosis and their relationship with degree of fibrosis. *Gastroenterol Hepatol*. 2004;27:47-50. [PubMed]

27. Breidert M, Zimmermann TF, Schneider R, et al. Ghrelin/leptin-imbalance in patients with primary biliary cirrhosis. *Exp Clin Endocrinol Diabetes*. 2004;112:123–6. [PubMed]
28. Gonnelli S, Caffarelli C, Del Santo K, et al. The relationship of ghrelin and adiponectin with bone mineral density and bone turnover markers in elderly men. *Calcif Tissue Int*. 2008;83:55–60. [PubMed]
29. Peng YM, Lei SF, Guo Y, et al. Sex-specific association of the glucocorticoid receptor gene with extreme BMD. *J Bone Miner Res*. 2008;23:247–52. [PMC free article] [PubMed]
30. Kanazawa I, Yamaguchi T, Yamamoto M, et al. Relationships between serum adiponectin levels versus bone mineral density, bone metabolic markers, and vertebral fractures in type 2 diabetes mellitus. *Eur J Endocrinol*. 2009;160:265–73. [PubMed]
31. Kontogianni MD, Dafni UG, Routsias JG, Skopouli FN. Blood leptin and adiponectin as possible mediators of the relation between fat mass and BMD in perimenopausal women. *J Bone Miner Res*. 2004;19:546–51. [PubMed]
32. Floreani A, Variola A, Niro G, et al. Plasma adiponectin levels in primary biliary cirrhosis: a novel perspective for link between hypercholesterolemia and protection against atherosclerosis. *Am J Gastroenterol*. 2008;103:1959–65. [PubMed]
33. EASL Clinical Practice Guidelines: Management of cholestatic liver diseases. *J Hepatol*. 2009;51:237–67. [PubMed]
34. Musialik J, Petelenz M, Gonciarz Z. Effects of alendronate on bone mass in patients with primary biliary cirrhosis and osteoporosis. Preliminary results after 1 year. *Scand J Gastroenterol*. 2005;40:873–4. [PubMed]
35. Zein CO, Jorgensen RA, Clarke B, et al. Alendronate improves bone mineral density in primary biliary cirrhosis: a randomized placebo-controlled trial. *Hepatology*. 2005;42:762–71. [PubMed]
36. Levy C, Harnois DM, Angulo P, et al. Raloxifen improves bone mass in osteoporotic women with primary biliary cirrhosis: result of pilot study. *Liver Int*. 2005;25:117–21. [PubMed]
37. Dresner-Pollak R, Gabet Y, Steimatzky A, et al. Human parathyroid hormone 1–34 prevents bone loss in experimental biliary cirrhosis in rats. *Gastroenterology*. 2008;134:259–67. [PubMed]
38. Dempster DW, Lambing CL, Kostenuik PJ, Grauer A. Role of RANK ligand and denosumab, a targeted RANK ligand inhibitor, in bone health and osteoporosis: a review of preclinical and clinical data. *Clin Ther*. 2012;34:521–36. [PubMed]

Articles from Przegląd Gastroenterologiczny are provided here courtesy of Termedia Publishing



Autoimmune Diseases: Overview

<http://www.womenshealth.gov>

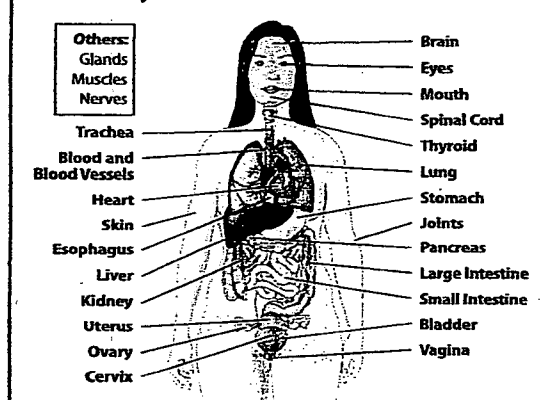
1-800-994-9662

TDD: 1-888-220-5446

Q: What are autoimmune diseases?

A: Our bodies have an immune system, which is a complex network of special cells and organs that defends the body from germs and other foreign invaders. At the core of the immune system is the ability to tell the difference between self and nonself: what's you and what's foreign. A flaw can make the body unable to tell the difference between self and nonself. When this happens, the body makes autoantibodies (AW-toh-AN-teye-bah-deez) that attack normal cells by mistake. At the same time special cells called regulatory T cells fail to do their job of keeping the immune system in line. The result is a misguided attack on your own body. This causes the damage we know as autoimmune disease. The body parts that are affected depend on the type of autoimmune disease. There are more than 80 known types.

Body Parts That Can Be Affected by Autoimmune Diseases



Q: How common are autoimmune diseases?

A: Overall, autoimmune diseases are common, affecting more than 23.5 million Americans. They are a leading cause of death and disability. Yet some autoimmune diseases are rare, while others, such as Hashimoto's thyroiditis, affect many people.

Q: Who gets autoimmune diseases?

A: Autoimmune diseases can affect anyone. Yet certain people are at greater risk, including:

- **Women of childbearing age** — More women than men have autoimmune diseases, which often start during their childbearing years.
- **People with a family history** — Some autoimmune diseases run in families, such as lupus and multiple sclerosis. It is also common for different types of autoimmune diseases to affect different members of a single family. Inheriting certain genes can make it more likely to get an autoimmune disease. But a combination of genes and other factors may trigger the disease to start.
- **People who are around certain things in the environment** — Certain events or environmental exposures may cause some autoimmune diseases, or make them worse. Sunlight, chemicals called solvents, and viral and bacterial infections are linked to many autoimmune diseases.
- **People of certain races or ethnic backgrounds** — Some autoimmune diseases are more common or more severely affect certain groups of people more than others. For instance, type 1 diabetes is more common in white people. Lupus is most severe for African-American and Hispanic people.

FREQUENTLY ASKED QUESTIONS



<http://www.womenshealth.gov>

1-800-994-9662

TDD: 1-888-220-5446

Types of Autoimmune Diseases & Their Symptoms	
Disease	Symptoms
Multiple sclerosis (MUHL-tip-uhl sklur-OH-suhss) (MS) A disease in which the immune system attacks the protective coating around the nerves. The damage affects the brain and spinal cord.	• Weakness and trouble with coordination, balance, speaking, and walking • Paralysis • Tremors • Numbness and tingling feeling in arms, legs, hands, and feet • Symptoms vary because the location and extent of each attack vary
Myasthenia gravis (meye-uhss-THEEN-ee-uh GRAV-uhss) (MG) A disease in which the immune system attacks the nerves and muscles throughout the body.	• Double vision, trouble keeping a steady gaze, and drooping eyelids • Trouble swallowing, with frequent gagging or choking • Weakness or paralysis • Muscles that work better after rest • Drooping head • Trouble climbing stairs or lifting things • Trouble talking
Primary biliary cirrhosis (BIL-ee-air-ee sur-ROH-suhss) The immune system slowly destroys the liver's bile ducts. Bile is a substance made in the liver. It travels through the bile ducts to help with digestion. When the ducts are destroyed, the bile builds up in the liver and hurts it. The damage causes the liver to harden and scar, and eventually stop working.	• Fatigue • Itchy skin • Dry eyes and mouth • Yellowing of skin and whites of eyes
Psoriasis (suh-REYE-uh-suhss) A disease that causes new skin cells that grow deep in your skin to rise too fast and pile up on the skin surface.	• Thick red patches, covered with scales, usually appearing on the head, elbows, and knees • Itching and pain, which can make it hard to sleep, walk, and care for yourself May have: • A form of arthritis that often affects the joints and the ends of the fingers and toes. Back pain can occur if the spine is involved.



[Home](#) → [Medical Encyclopedia](#) → Depression - older adults

URL of this page: [//medlineplus.gov/ency/article/001521.htm](https://medlineplus.gov/ency/article/001521.htm)

Depression - older adults

Depression is a mental health condition. It is a mood disorder in which feelings of sadness, loss, anger, or frustration interfere with daily life for weeks or longer.

Depression in older adults is a widespread problem, but it is not a normal part of aging. It is often not recognized or treated.

Causes

In older adults, life changes can increase the risk for depression or make existing depression worse. Some of these changes are:

- A move from home, such as to a retirement facility
- Chronic illness or pain
- Children moving away
- Spouse or close friends passing away
- Loss of independence (for example, problems getting around or caring for oneself)

Depression can also be related to a physical illness, such as:

- Thyroid disorders
- Parkinson disease
- Heart disease
- Cancer
- Stroke
- Dementia (such as Alzheimer disease)

Overuse of alcohol or certain medicines (such as sleep aids) can make depression worse.

Symptoms

Many of the usual symptoms of depression may be seen. However, depression in older adults may be hard to detect. Common symptoms such as fatigue, appetite loss, and trouble sleeping can be part of the aging process or a physical illness. As a result, early depression may be ignored, or confused with other conditions that are common in older adults.

Exams and Tests

The doctor or nurse will examine you and ask questions about your medical history and symptoms.

Blood and urine tests may be done to look for a physical illness.

You may be referred to a mental health specialist to help with diagnosis and treatment.

Treatment

The first steps of treatment are to:

- Treat any illness that may be causing the symptoms
- Stop taking any medications that may be making symptoms worse
- Avoid alcohol and sleep aids

If these steps do not help, medicines to treat depression and talk therapy often help.

Doctors often prescribe lower doses of antidepressants to older people, and increase the dose more slowly than in younger adults.

To better manage depression at home, you should:

- Exercise regularly, if your doctor says it is ok.
- Surround yourself with caring, positive people and do fun activities.
- Learn good sleep habits.
- Learn to watch for the early signs of depression, and know how to react if these occur.
- Drink less alcohol and do not use illegal drugs.
- Talk about your feelings with someone you trust.
- Take medications correctly and discuss any side effects with your doctor.

Outlook (Prognosis)

Depression often responds to treatment. The outcome is usually better for people who have access to social services, family, and friends who can help them stay active and engaged.

The most worrisome complication of depression is suicide. Men make up most suicides among older adults. Divorced or widowed men are at the highest risk.

Families should pay close attention to elderly relatives who are depressed and who live alone.

When to Contact a Medical Professional

Call your health care provider if you keep feeling sad, worthless, or hopeless, or if you cry often. Also call if you are having trouble coping with stresses in your life and want to be referred for talk therapy.

Go to the nearest emergency room or call your local emergency number (such as 911) if you are thinking about suicide (taking your own life).

If you are caring for an aging family member and think they may have depression, contact their health care provider.

Alternative Names

Depression in the elderly

References

Abbasi O, Burke WJ. Depression. In: Ham RJ Jr., Sloane PD, Warshaw GA, et al., eds. *Ham's Primary Care Geriatrics: A Case-Based Approach*. 6th ed. Philadelphia, PA: Saunders Elsevier; 2013:chap 18.

Ismail Z, Fischer C, McCall WV. What characterizes late-life depression? [<https://www.ncbi.nlm.nih.gov/pubmed/24229652>]

Psychiatr Clin N Am

Review Date 9/2/2014

Updated by: Timothy Rogge, MD, Medical Director, Family Medical Psychiatry Center, Kirkland, WA. Also reviewed by David Zieve, MD, MHA, Isla Ogilvie, PhD, and the A.D.A.M. Editorial team.



A.D.A.M., Inc. is accredited by URAC, also known as the American Accreditation HealthCare Commission (www.urac.org).

URAC's [accreditation program](#) is an independent audit to verify that A.D.A.M. follows rigorous standards of quality and

accountability. A.D.A.M. is among the first to achieve this important distinction for online health information and services. Learn

more about A.D.A.M.'s [editorial policy](#), [editorial process](#) and [privacy policy](#). A.D.A.M. is also a founding member of Hi-Ethics and subscribes to the principles of the Health on the Net Foundation (www.hon.ch).

The information provided herein should not be used during any medical emergency or for the diagnosis or treatment of any medical condition. A licensed physician should be consulted for diagnosis and treatment of any and all medical conditions. Call 911 for all medical emergencies. Links to other sites are provided for information only -- they do not constitute endorsements of those other sites. Copyright 1997-2016, A.D.A.M., Inc.

Duplication for commercial use must be authorized in writing by ADAM Health Solutions.

ADAM

U.S. National Library of Medicine 8600 Rockville Pike, Bethesda, MD 20894

U.S. Department of Health and Human Services National Institutes of Health

Page last updated: 02 December 2016

FORM 16

CERTIFICATE OF COUNSEL FOR RECORD ON APPEAL
THE STATE OF SOUTH CAROLINA
IN THE COURT OF APPEALS
[IN THE SUPREME COURT]

APPEAL FROM SOUTH CAROLINA WORKERS'S
COMPENSTATION COMMISSION

COMMISSIONERS: Melody James, Chair, Aisha Taylor, Avery Wilkerson

Case No. 2017-001027

Seeahray Brailsford
Employee, Claimant, Appellant

Appellant

V

Piggly Wiggly, INC., Employer
Constitution State Service Company
AS TPA for Greenbax Enterprise, INC., Carrier

Respondent

CERTIFICATE OF COUNSEL

The undersigned certified that this Record on Appeals complies with Rule 211 (b), SCACR

Seeahray Brailsford
322 Rice Road
Newberry, S. C. 29108
(803) 276-0001
Employee, Claimant, Appellant
Self Represent