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DEPARTMENT OF VETERANS AFFAIRS

CLAIMS INTAKE CENTER

PO BOX 4444

JANESVILLE, WI 53547-4444

Dear Claims Administrator,

I am a medical doctor specializing in internal medicine: my National Provider Identifier (NPI) number is 5555555555. This medical opinion is offered in support of Mr. Jim Doe’s (SSN last four 5555) claim for VA disability compensation benefits in reference to his gout disability. In rendering this opinion, I have reviewed the relevant documentation from service medical records, post service medical records, Mr. Doe’s personal statement dated 10/10/2021, various treatise and other documents referenced in Mr. Doe’s personal statement, witness statements dated 9/9/2021 and 8/8/2021, VA provided DBQ dated 6/6/2021, VA provided medical opinion dated 6/6/2021, and the VA decision notification dated 11/11/2021.

I have also conducted a video interview with Mr. Doe where we discussed the aforementioned documentation. My conclusions are based solely on the evidence of record and the references cited on this document. Three opinions are contained on this document: opinion (1) establishes the onset of the gout disease during service; opinion (2) establishes a likely gout attack during service and opinion (3) establishes gout being caused and/or aggravated secondary to service-connected osteoarthritis at the big toe joints. I have also provided a rebuttal of the VA medical opinion dated 6/6/2021.

Gout Current Diagnosis

Post service medical records note that Mr. Doe was diagnosed and treated for gout on five separate occasions: 2008, 2010, 2012, 2015, and 2017. The 2015 and 2017 episodes are supported by x-ray findings denoting gouty changes. The symptoms noted during these episodes were absolutely typical of gout attacks; hence, by history I concur with the diagnosis of gout on all five occasions. There is no doubt that Mr. Doe currently has gout.

Nature and Course of Gout

Gout is a chronic heterogeneous disorder of urate metabolism causing a common form of inflammatory arthritis that is very painful. Gout is one of the many forms of arthritis. It usually affects one joint at a time (often the big toe joint). There are times when symptoms get worse, known as flares, and times when there are no symptoms, known as remission. Repeated bouts of gout can lead to gouty arthritis, a worsening form of arthritis. There is no cure for gout; however, gout can often be controlled with proper medications and a gout diet.

Gout flares start suddenly and can last days or weeks. These flares are followed by long periods of remission—weeks, months, or years—without symptoms before another flare begins. Gout usually occurs in only one joint at a time. It is often found in the big toe. Along with the big toe, joints that are commonly affected are the lesser toe joints, the ankle, and the knee. Symptoms in the affected joint(s) may include: pain (usually intense), swelling, redness, and heat.

Gout is caused by a condition known as hyperuricemia, where there is too much uric acid (UA) in the body. The body makes UA when it breaks down purines, which are found in your body and the foods you eat. If UA reaches a level in the blood of 6.2 -6.8 mg/dL or higher,3 it becomes insoluble and needlelike crystals of a salt called monosodium urate (MSU) may form. In lower temperature areas of the body (such as the joints in the feet) crystals can form at lower concentrations.

Typically treating doctors will want UA levels reduced below 6 mg/dL to control gout episodes. Hyperuricemia does not always cause gout, and hyperuricemia without gout symptoms does not need to be treated. Left untreated gout will likely progress through four stages:3 asymptomatic hyperuricemia, acute gout, intercritical gout, and chronic tophaceous gout. Mr. Doe’s gout is currently at stage 3; thus, I will not discuss stage 4 as it is not relevant at this time.

In asymptomatic hyperuricemia (stage 1) there are no physical symptoms, but there is an increase in blood UA levels capable of producing crystals (onset of disease). This stage may last 20-30 years or more.3, 5 During this period, crystals build up in the joints that eventually trigger inflammation, redness, swelling, and pain. Long term sustained increased levels of UA in the blood is a prerequisite for acute gouty arthritis.

Acute gouty arthritis (stage 2) is when the first physical symptoms of gout appear. Sometimes the first signs of gout are brief twinges of pain (petit attacks)3 in an affected joint. These attacks can occur for several years before the full-blown condition occurs.

Intercritical gout (stage 3) is the term used to describe the periods between attacks. The first attack is usually followed by a complete disappearance (remission) of symptoms. But, untreated, gout nearly always returns. Over two-thirds of patients have at least one more attack within 2 years of the first attack. By 10 years, over 90% of patients who had one attack are likely to have more attacks.

The following make it more likely that an individual will develop hyperuricemia, which causes gout: being male, being obese, having certain health conditions, including; congestive heart failure, hypertension, insulin resistance, metabolic syndrome, diabetes, poor kidney function, eating or drinking food and drinks high in fructose (a type of sugar), drinking alcohol, and having a diet high in purines which the body breaks down into UA.  Purine-rich foods include red meat, organ meat, and some kinds of seafood, such as anchovies, sardines, mussels, scallops, trout, and tuna.

Direct Relationship to Service Due to the Onset of Gout During Service

The first key document in Mr. Doe’s service treatment records is a 08 June 1987 blood test noting a uric acid (UA) level of 6.7mg/dL, which is well within the range to produce crystals (stage 1 of gout). The second key document is a post service treatment record noting the first diagnosed gout attack on 13 August of 2008 (stage 2 of gout). From 1987 to 2008 is 21 years; thus, considering that gout typically takes 20 years or more to progress from stage 1 to stage 2 it is likely that the onset of gout was in 1987 during Mr. Doe’s service years. The temporal relationship from stage 1 to stage 2 is undeniable and exactly matches the course and nature of gout. The temporal relationship is supported by other documents in service medical records and Mr. Doe’s personal statement as follows:

The 1974 enlistment examination notes Mr. Doe’s weight as 122 pounds, height as 65 inches, and his blood pressure of 110/70: all normal. A routine physical in 1987 notes a weight of 145 and a lipid profile, blood glucose, and metabolic panel that are well within the normal ranges. Even the UA level of 6.7mg/dL noted at this time was normal albeit at the high end. Mr. Doe’s personal statement notes that his weight increased substantially to the low to mid 140s within his first 3 years of service and stayed that way for the next ten years; however, that later in 1987 he hurt his back resulting in a decrease in his ability to exercise at his previous intensity levels resulting in weight gain. He also noted that by his retirement his waist size increased from 36 to 39 inches. Visceral fat (belly fat) is a risk factor for gout.

Service medical records note a 22 November 1987 back injury event with severe left sided sciatica and five back pain and/or sciatica complaints from 1987 until his 1995 retirement from service. Service medical records also note a 12 September 1992 lumbar spine MRI that cited several damaged lumbar vertebrae, nerve impingement, and traumatic arthritis. These injuries led to a dramatic increase in Mr. Doe’s weight: his 1994 retirement physical recorded a weight of 170, a 25-pound increase in just seven years: for a total weight gain of 48-pounds during service. The end of service BMI was 28.3 which is substantially overweight. This weight is a risk factor for and comorbidity of gout. The recorded values noted during the retirement exam demonstrate a body system that is completely out of control.

His blood pressure was abnormal at 130/80 which is considered Stage 1 hypertension. The fasting lipid profile notes total cholesterol at 249, triglycerides at 255, LDL cholesterol 161, and an HDL cholesterol at 37: all abnormal. For numerous health reasons these results are alarming to say the least. These results establish the presents of metabolic syndrome which is a key risk factor for and comorbidity of gout. Metabolic syndrome is simply a series of traits. If you have the traits, you have the syndrome. In this case, we have a blood pressure systolic reading of 130 mm Hg, triglycerides at 255, and HDL cholesterol at 37 which clearly establish a diagnosis of metabolic syndrome during service per National Heart, Lung, and Blood Institute guidelines.4

***Opinion (1):***

Given the fact that UA levels were within the range to produce crystals (stage 1 of gout) during service and the temporal relationship from stage 1 to stage 2 of gout: coupled with the in-service risk factors/comorbidities of being overweight, visceral fat, a purine-rich diet (discussed below), daily alcohol use (discussed below), and metabolic syndrome it is likely (more than a 50% likeliness) that gout had its onset in service; thus, a nexus to service is established.

Direct Relationship to Service Based on an In-Service Event

It should be noted that when gout was first officially diagnosed in 2008 the treating physician recorded that the “patient reports similar episodes of less severity in the past.” This is important in that a 25 June 1992 service treatment record notes a complaint of foot pain, but no diagnosis or exact location of the pain was given. Mr. Doe’s personal statement clarifies the 1992 report as extreme pain, redness, and swelling at the big toe joint that only lasted 6-8 hours. His statement goes on to note that he has had many unreported short-term (hours long) pain episodes at the big toe joints between 1992 and 2008. Several facts of record support gout attacks during service as follows:

1. The short-term big toe joint pain episodes described by Mr. Doe in his personal statement and to the treating physician in 2008 are consistent with gout “petit” attacks.3 These short-term big toe joint pain events during and post service establishes that gout stage 2 was likely present long before gout was first diagnosed in 2008.
2. Most gout attacks are at the big toe joints.3 All of Mr. Doe’s gout attacks have been at the big toe joints.
3. If we consider the 1992 service treatment record report of foot pain (a gout petit attack) as the beginning of stage 2 of gout (acute gouty arthritis), I would expect to see increased levels of UA (gout stage 1) dating back approximately 20 years to be consistent with the course and nature of gout; unfortunately, the June 1987 blood test noting a UA level of 6.7mg/dL is the only UA test in service treatment records. In lieu of an actual UA test, evidence of record indicates that Mr. Doe’s UA levels were likely elevated during service long before 1987.

Generally, UA concentration increases gradually with age from birth to puberty. Body weight and UA levels plateau during late puberty because of the effects of sex hormones. By the end of puberty, for young adults within their normal BMI, UA levels are at the low end of the normal range 3.5 to 4.5 mg/dL. Since Mr. Doe entered service just days after his 17th birthday, shortly after the end of puberty, I would expect his UA levels to be at the low end of the normal range; however, once he entered service Mr. Doe’s personal statement notes a significant shift in diet.

He describes his before service diet as high in fruits and vegetables while being low in red meats, seafood, and no alcohol use. Once he entered the service (1974) his diet fully reversed as high in red meats, seafood, and daily alcohol use. The army dining menu of record is full of examples of purine-rich foods with no low purine diet option capable of sustaining the caloric demands of military service.

This change in diet undoubtedly led to an immediate and sustained raise in Mr. Doe’s UA level over his years of military service as demonstrated by the 1987 blood test noting a UA level of 6.7mg/dL. By the 1992 report of foot pain at the big toe joint Mr. Doe’s UA level had likely been at a level capable of producing crystals (gout stage 1) for 18 years (1974-1992). The temporal relationship of 18 years from stage 1 to stage 2 is consistent with the course and nature of gout.

1. Mr. Doe’s personal statement cites nearly daily alcohol use, when not on tactical assignments, during his 20 years of active duty. He describes his drinking as limited to beer and he typically drank two per day. Alcohol, especially beer, is very high in purines making it a risk factor for gout; additionally, beer is a well know trigger for gout attacks.
2. Service treatments records cite the following comorbidities during service: being overweight and metabolic syndrome (per retirement blood test lab results).
3. Mr. Doe’s personal statement cites the following gout risk factors present during service: visceral fat and purine-rich diet.
4. VA hospital lab testing results dated 12 October 2012 identify Mr. Doe with an inherited trait of UA underexcretion. In other words, his body does not adequately remove UA. This leads to higher levels UA in the blood: 90% of gout suffers have this trait. Undersecretion disorders are autosomal dominantly inherited: meaning he inherited a gene from one of his parents that caused the UA underexcretion trait. This inherited trait further adds evidence supporting that sustained higher levels of UA were present during service years.

*Opinion (2):*

Given Mr. Doe’s reports of short-term foot pain events at the big toe joints during service, the fact that UA levels were within the range to produce crystals (stage 1 of gout) during service, the temporal relationship from stage 1 to stage 2 of gout, the gout risk factors present in service, the gout comorbidities present in service, the alcohol use while in service, and the UA underexcretion trait it is at least as likely as not (a 50% likeliness) that the 25 June 1992 service treatment record noting a complaint of foot pain was a petit gout attack; thus, a nexus to service is established.

Secondary Relationship to service: Gout related to Big Toe Osteoarthritis

A 2019 study determined that the osteoarthritic process releases small cartilage particulates into joint spaces that promote MSU crystal formation.6 The study also noted that the crystals in osteoarthritic joints were smaller, which have greater inflammatory potential. A 2014 study concluded that osteoarthritic joints are at great risk for gout.7 Of particular importance is a 2007 study that found MSU crystal formation and deposition will be further potentiated in the osteoarthritic first MTPJ (the big toe joint) by impaired urate solubility and enhanced crystal nucleation arising from factors relating to the anatomical location of the first MTPJ namely lower distal temperature and physical stress.8

Opinion (3):

Given the fact that osteoarthritic joints promote MSU crystal formation increasing the risk for and severity of gout and the fact that an osteoarthritic big toe joint is at a very high risk of developing gout coupled with the fact that all five gout episodes of record occurred at Mr. Doe’s service-connected osteoarthritic big toe joints, it is at least as likely as not (a 50% likeliness) that gout developed as a direct result of the osteoarthritic big toe joints; thus, a nexus to service is established. Also, it is likely (more than a 50% likeliness) that gout is aggravated beyond the natural progression of the disease by the osteoarthritic big toe joints causing increased attack frequency, longer lasting attacks, and heightened pain levels; thus, a nexus to service is established.

VA Medical Opinion Rebuttal

The VA medical opinion dated 6/6/2021 is flawed or misleading on nearly every conclusion or statement made by the VA medical examiner as follows:

1. The examiner concluded that Mr. Doe currently does not have gout nor has he ever had gout. She bases her conclusion on the lack of a joint fluid test which would conclusively confirm the presence of UA crystals. She also challenges the 2008 gout diagnosis, stating that the big toe joint could be inflamed for any number of reasons. It’s worth mentioning that during the 2015 and 2017 episodes the record notes that Mr. Doe requested a joint fluid test, but the examining VA treatment provider refused to conduct the testing. It’s illogical for VA to deny a claim for lack of a test, when VA refuses to do the test when the opportunity presents itself. Joint aspiration is only done during a gout flare.

It is true that a joint fluid test is the gold standard for diagnosing gout; however, in practice joint fluid is typically not drawn. The American College of Rheumatology concluded:2

While this gold standard has high specificity, its feasibility and sensitivity may be inadequate, because of difficulty with aspiration of joints (particularly small ones) and/or examination of the sample under polarising microscopy. Thus, although MSU crystal results are extremely helpful when positive, they are not a feasible universal standard…

Also, joint aspiration introduces the possibility of infection to the joint; thus, aspiration is only done when a gout diagnosis is in question. In Mr. Doe case he has been diagnosed with gout on five different occasions by five different doctors with several different specialties including rheumatology, podiatry, and internal medicine. Each doctor did a thorough in person examination during a joint pain episode and noted symptoms of warmth, pain, swelling, and extreme tenderness at the big toe joint. This is a classic gout attack called “podagra.”

They also note that the symptoms came on suddenly with intense pain within the first 12-24 hours which is another classic gout trait. There is simply little doubt here that these episodes are gout, which is the likely reason why a joint fluid test was not done. The Mayo Clinic states that “Doctors usually diagnose gout based on your symptoms and the appearance of the affected joint.”1 The American College of Rheumatology (ACR) and the European League Against Rheumatism (EULAR) jointly published guidelines for diagnosing gout in 2015.2 The symptoms noted on each of Mr. Doe’s episodes meet the criteria for a gout diagnosis. I see no reasonable basis for challenging Mr. Doe’s 2008 gout diagnosis or any of the five.

The VA opinion makes no mention of the 2010, 2012, 2015, or 2017 gout events of record; thus, it’s clear that the examiner did not adequately review the record. The VA opinion is silent in regard of the x-ray evidence noting gouty changes in 2015 and 2017; for this reason alone, the opinion is inadequate. The x-ray evidence combined with the reported symptoms make a gout diagnosis conclusive.

1. The VA examiner concluded that even if the 2008 event was gout, that there is no temporal relationship to service since that acute event was 13 years after service. This conclusion is not consistent with the course and nature of gout. The first stage of gout is asymptomatic hyperuricemia and, to be clear, this stage must happen. There will be no acute gout attack without a long period (approximately 20 years) of sustained UA levels in the crystal producing range. As noted previously in this document, UA levels in the crystal producing range were noted during service in 1987; thus, from 1987 to 2008 is 21 years which exactly matches the temporal relationship from stage 1 to stage 2 of gout.
2. The examiner notes Mr. Doe’s UA level, at the time of examination, as 7.0 mg/dL and states that this level is within the normal range. An adjudicator is likely to interpret the examiners conclusion that the level was normal as a point weighting against a gout finding, when in fact the reverse is true. The examiner omits the most important fact about gout, which is that 7.0 mg/dL is hyperuricemia and well above the range to produce MSU crystals which are the cause of gout. The 7.0 mg/dL UA level is a fact weighing in favor of a gout finding.

It’s worth noting that there are 11 UA blood test results noted in post-service medical records (starting in 1998-present) and all are in the hyperuricemia range; thus, there has been long standing elevated UA levels capable of producing MSU crystals evident in the record which is a key fact supporting a gout finding. Clearly a thorough examination of post service medical records was not conducted by the VA medical examiner.

The information, opinions, and rebuttal contained on this document are based on my medical training, my 14 years of practical medical experience, the referenced medical treatise, and the known medical science of gout.

Sign: Date:

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