

Table 1. Illustrative Provider and Patient Quotations from Source Studies

Analytical Theme	Quotation	Reference
Provider Barriers		
Knowledge gaps and management approaches	"I think that there is lack of knowledge by both patients and health professionals. I just thought you just had gout flare ups and then it just went away, so there is definitely a need for education and better training."	Spencer <i>et al.</i>
Perceptions and beliefs about gout patients	"Adherence to uric acid-lowering therapy is not a problem in patients with gout, since they are well aware of the fact they will get new gout attacks if they do not take their medication."	Spaetgens <i>et al.</i>
System barriers to optimal gout care	"It's another thing, too, the time issue. Cause if you're really, really busy, you don't spend time to talk to the patient, you don't have time, if we're busy."	Humphrey <i>et al.</i>
Patient Barriers		
Limited gout knowledge	"I think I have accepted the fact that there is no cure. It is up to me just to minimize it, I think. I don't think there is any cure because I haven't talked to anybody who has had it and say they don't get it anymore. Is that possible?"	Lindsay <i>et al.</i>
Attitudes toward taking medication	"So I know gout's never going to kill me, right. So I don't want to be taking – I don't want to be rattling around full of tablets all the time"	Richardson <i>et al.</i>
Interactions with healthcare providers	"I don't think they gave you enough. It kind of wasn't even the basics. There were no follow-ups or anything and I was going regularly. There must have been time in there. I didn't know about uric acid levels or what I should aim for. In my mind, I never had it explained."	Te Karu <i>et al.</i>
Practical barriers to chronic medication use	"I'm frequently too busy with school pressures to worry about taking medication regularly."	Katz <i>et al.</i>

Conclusions: Our thematic synthesis identified several barriers to gout care, particularly knowledge gaps among both providers and patients as well as implementation barriers to the provision of optimal care. Knowledge translation initiatives emphasizing the "curable" nature of gout targeting both providers and patients as well as strategies to reduce system barriers and support regular medication use are urgently needed to improve gout care.

Acknowledgements: This study was supported in part by a grant from the Canadian Institutes of Health Research (PCS 146388). We wish to thank the Arthritis Patient Advisory Board of Arthritis Research Canada for providing their consumer input into this project.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.4298

THU0461 ACCURACY OF HUMASENS-PLUS POINT-OF-CARE URIC ACID METER USING CAPILLARY BLOOD OBTAINED BY FINGERTIP PUNCTURE

S. Fabre¹, P. Clerson², J.-M. Launay³, J.-F. Gautier⁴, T. Vidal-Trecan⁴, J.-P. Riveline⁴, A. Platt⁵, A. Abrahamsson⁶, J.N. Miner⁷, G. Hughes⁵, P. Richette¹, T. Bardin¹. ¹Rheumatology department, Hôpital Lariboisière, Paris; ²Soladis Clinical Studies, Roubaix; ³Biochemistry department; ⁴Diabetology department, Hôpital Lariboisière, Paris, France; ⁵Personalised Healthcare Unit, AstraZeneca, Cambridge, United Kingdom; ⁶Personalised Healthcare Unit, AstraZeneca, Mölndal, Sweden; ⁷Research and development, Ardea Biosciences Inc., San Diego, CA, United States

Background: A key factor in the success of gout management is the long-term lowering of uricemia below predetermined targets (300 or 360 μ mol/l). Monitoring of uricemia in gout patients is therefore important, and is presently done in the laboratory on plasma samples obtained after venous puncture. An accurate uric acid (UA) meter allowing rapid testing by the health care professionals and self-measurement by the patient should improve management of gout.

Objectives: This study aimed to assess the reliability of immediate UA measurement in capillary blood samples obtained from fingertip puncture using the HumaSens^{Plus} point-of-care meter (meter) compared with that of a standard laboratory assay (lab).

Methods: Capillary UA levels were measured from 236 consenting diabetic patients using the commercially available HumaSens^{Plus} UA meter (European Conformity marked and approved for EU market use). Each patient also had a plasma UA measurement in the biochemistry laboratory using an uricase automated colorimetric assay. Since the UA meter has a dynamic range of 180–1190 μ mol/l, when the values were out-ranged (meter reading LO or HI), they were individually compared to corresponding plasma measurements. Agreement between capillary and plasma UA levels was assessed by Intraclass Correlation Coefficient (ICC) and Bland-Altman graphic representation. Best capillary UA threshold for detection of hyperuricemia (plasma UA > 360 μ mol/l) was determined from a ROC curve, relationship between methods were identified by regression. Impact of potential confounding factors (biological parameters/treatments) was searched. A total of 206 paired measurements were required for estimation of an ICC of 0.80 with a precision of 0.10 at alpha risk of 0.05%. To better understand discrepancies between meter and lab, results were compared to reference plasma UA measurements by liquid chromatography-mass spectrometry (LC-MS) in a subgroup of 77 patients who gave complementary consent.

Results: Fourteen capillary samples were read LO by the meter: 11 were confirmed by lab to be below 180 μ mol/l and 3 were above (189, 206 and 428 μ mol/l). Two capillary samples were read HI and were measured at 303 and 213 μ mol/l by lab. In the remaining 222 samples with meter and lab values, ICC was 0.90 [0.87–0.92] and Bland-Altman curve showed acceptable agreement over all the tested values. Best meter threshold for detection of hyperuricemia by the HumaSens^{Plus} meter was 330 μ mol/l (sensitivity 0.89, specificity 0.89, area under the ROC curve 0.95). Based on regression, plasma uricase of

360 μ mol/l corresponded to 343 μ mol/l. Among the biological parameters tested, only hematocrit impacted capillary uric acid measurements, however negligibly. No medication appeared to significantly affect test results. Plasma uricase measurements were better correlated to LC-MS measurements ($r=0.98$ [0.96–0.99]) than capillary measurements ($r=0.84$ [0.75–0.90]).

Conclusions: Results of the HumaSens^{Plus} meter were reasonably comparable to those of the laboratory assay. It is easy to use and may be useful in clinic and in epidemiologic studies.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.2787

THU0462 MORTALITY IN PATIENTS WITH GOUT: A SYSTEMATIC REVIEW

S. Mitchell¹, H. Liedgens², E. Johannes³. ¹Decision Resources Group, Bicester, United Kingdom; ²Head Health Economics and Outcomes Research Market Access, SBU Europe; ³Head Market Access Europe, Grünenthal GmbH, Aachen, Germany

Background: Gout is a chronic, progressive, inflammatory disease characterised by elevated serum uric acid (sUA) levels (1). In Europe the prevalence of gout ranges from 0.9–2.5%, and is increasing (2). Published data indicate that gout is an independent risk factor for both all-cause and cardiovascular (CV)-related mortality (3, 4).

Objectives: To conduct a systematic review to identify studies reporting the association between gout and mortality (all-cause and CV-related).

Methods: Relevant publications were identified by interrogating electronic databases; Medline & MEDLINE In-Process, EMBASE and the Cochrane Library (accessed 3 May 2016). Eligibility criteria included adult patients with a definitive diagnosis of acute/chronic gout (self-reported/physician diagnosed), with no restriction on publication date, study design or geography.

Results: Nineteen studies met the pre-defined inclusion criteria and were reviewed. The studies were conducted in: the US (n=8); Taiwan (n=5); Canada (n=3); Spain (n=1); Singapore (n=1); and the UK (n=1). In addition to patients having a diagnosis of acute/chronic gout, 6 of the 19 studies were conducted in the following patient subgroups: renal transplant (n=1); chronic kidney disease (n=2); patients with a recent acute myocardial infarction (n=2); and patients with heart failure (n=1). There were several consistent findings across the 19 studies: (i) gout was associated with an increase in both all-cause mortality (reported hazard ratios [HR] ranged from 1.13 to 2.37) and CV-related mortality (reported HR ranged from 1.10 to 3.88) compared with patients without gout; (ii) the increased risk in all-cause mortality was primarily driven by an increase in CV-related mortality; (iii) the increased mortality risk was higher in females than males. One study reported that the presence of tophi was independently associated with a higher risk of all-cause mortality. Notably one study reported that patients who received urate-lowering therapy (ULT) have a statistically significant lower all-cause mortality and CV-related mortality risk relative to patients who do not receive ULT.

Conclusions: This systematic review confirms that gout is associated with an increased risk of all-cause and CV-related mortality; this was consistently reported across the eligible studies. The findings highlight the risk associated with gout and emphasise the need for appropriate treatment of this curable disease.

References:

- Keenan R, et al. Etiology and pathogenesis of hyperuricemia and gout. Kelley's textbook of rheumatology. Elsevier Saunders. 2013;94:1533–53.
- Richette P, et al. 2016 updated EULAR evidence-based recommendations for the management of gout. Ann Rheum Dis. 2016;annrheumdis-2016-209707.
- Lottmann K, et al. Association between gout and all-cause as well as cardiovascular mortality: a systematic review. Curr Rheumatol Rep. 2012;14(2):195–203.
- Clarson L, et al. Increased cardiovascular mortality associated with gout: a systematic review and meta-analysis. Eur J Prev Cardiol. 2015;22(2):335–43.

Disclosure of Interest: S. Mitchell: None declared, H. Liedgens Employee of: Grünenthal GmbH, E. Johannes Employee of: Grünenthal GmbH

DOI: 10.1136/annrheumdis-2017-eular.6062

THU0463 EPIDEMIOLOGY OF GOUT AND HYPERURICEMIA IN NEW CALEDONIA

T. Bardin¹, E. Magnat², P. Clerson³, P. Richette⁴, B. Rouchon². ¹Rheumatology, Hôpital Lariboisière, Paris, France; ²Agence Sanitaire et Sociale, Nouméa, New Caledonia; ³Soladis Clinical Studies, Roubaix; ⁴Rheumatology, Hôpital Lariboisière, Paris, France

Background: New Caledonia is a Pacific island of 270,000 inhabitants with mixed ethnicities, including Melanesians (39.1%) and Polynesians (10.2%) and people from European ancestry (27.2%).

Objectives: To determine the prevalence of gout and hyperuricemia in the various ethnicities and to characterize associated factors.

Methods: A 3-degree random sample of the population aged 18 to 60 years old was redressed according to the 2014 New Caledonia census. Face-to-face interviews were performed by trained nurses who used a predefined questionnaire along with planned physical measurements. All participants underwent capillary measurement of creatinine level (StatSensor) and all men and only women older