Pius X (1835-1914): the last gouty pope

Storia della nefrologia italiana

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ABSTRACT

Gout is a common, complex, systemic and well-studied form of chronic inflammatory arthritis in adults. It is due to the deposition of sodium monourate crystals in peripheral joints and periarticular tissues driven by hyperuricemia. Gout is the oldest recorded inflammatory arthritis to affect humankind, with roots stretching back to 2460 BC. It is known as "the rich man's disease", "the patrician malady", "a disease of plenty", "disease of kings", "disease of Western Society", and also "a life-style disease". Few studies have addressed the problem of gout among popes, affluent people who usually live longer than their contemporaries and are among the most scrutinized persons. Pius X (1835-1914) was the last pope with gout.

Gout seems to have affected 26 out of 265 popes (9.81%) from Saint Peter to Benedict XVI (34-2013 AD). The first was Gregory I Magnus, who was pope in the years 590-604, the last was Pius X, who reigned from 1903 to 1914 at age 79. Their age at death was 71.7 ± 9.2 years (Mean \pm SD). All popes were elderly men, some had voracious appetites and/or were wine drinkers. Several were sedentary and obese, while others were sober eaters, who took long walks or went riding. Chiragra (arthritic pain in the hands), podagra (arthritic pain in the big toe) and renal stone disease were among the most frequent disturbances.

The causes of death, due to CKD, strokes and infections are discussed along with the fact that gout disappeared from the Vatican Palace on August 22, 1914. However, in accordance with the Theory of Epidemiological Transition, gout seems likely to become a problem for the general population, increasingly adopting unhealthy lifestyle choices, in the absence of a correct education.

KEYWORDS: gout, popes, Pius X, renal death, death due to infection, death due to stroke

Introduction

Gout is a chronic, painful, non-infectious, non-lethal disease associated with crystal deposition of uric acid, when uric acid concentration exceeds 6.8 mg/dl plasma. The kidneys may cause hyperuricemia – the prevalence of which increases in the old and very old, – but are also the target of hyperuricemia (renal stones, renal disease and its progression). Hyperuricemias are due to either renal overload, renal underexcretion or a combination of both; renal overload may be due to overproduction by dietary purines, endogenous purine synthesis, purine breakdown and purine salvage [1–6]. Gout, known also as the "patrician malady" and the "disease of distinction" [7,8] is the oldest recorded inflammatory arthritis to affect humankind, with roots stretching back to 2640 BC [9].

Popes by definition belong to the most affluent class and their lifespan is longer than their contemporaries. In fact, a total of 51 pontiffs reigning in the years 1493 to 2005 lived to a mean age of 63.9 years and died an average of 10.0 years after being enthroned [10]. We have studied the narratives around popes, from Saint Peter to Benedict XVI [11–15], and demonstrated a high prevalence of gout. In a recent review [16] we identified a total of 25 gouty popes: 14 out of 25 (58%) had risk factors; 5 out of 25 (25%) had comorbidities; 21 out of 25 (84%) were unable to perform their duties; 8 out of 25 (32%) died of stroke; 12 of them (68%) had renal disease; 12 out of 17 (70.6%) underwent a renal death. Renal disease did not affect age at death [16].

This paper focuses on the last gouty pope, Pope Pius X. His death has been traditionally but wrongly attributed, even by us, to acute pneumonia. The present study now points out that his death was most likely linked to uremia, due to lasting gout, the final straw being acute pulmonary infection.

Historical case report – Pius X (1835-1914), Pope (8/4, 1903-8/22,1914)

Pius X (Figure 1), born Giuseppe Melchiorre Sarto on June 2, 1835 at Reise (Province of Treviso), was ordained priest in 1858 and, in the same year, became parish priest. Later he was nominated bishop of Mantua (1884), cardinal and patriarch of Venice (June 1893) and elected Pope on August 4, 1903; he reigned until August 22, 1914. A renowned orator, he is remembered for his expertise in sacred music and for hiring Lorenzo Perosi for the Choir of the Sistine Chapel, for his antimodernism and the refusal of science, for the letters sent to European powers to avoid the First World War, and for the wide pastoral care and the love for the poor. In his last will and testament wrote "born poor, lived poor, want to die poor". Roger Aubert, the Belgian historian Roger Aubert (1914-2009) has defined Pius X as the greatest reformer of the internal life of the Church after the Council of Trent [17].

His health has been described as good until the end of his days and his death ascribed to "acute tracheitis, bronchitis, infection-inflammation of the lower left lung lobe", a disease of acute onset followed by rapid worsening. He was under the care of Andrea Amici (1870-1920), archiater and chief of medical services in the Vatican, and of Ettore Marchiafava (1847-1935), professor of pathology at the University La Sapienza in Rome. His disease lasted from Saturday August 15 (he celebrated the last mass) to the night of August 20, 1920. The course was characterized by a worsening fever that, in his last hours, peaked at 40°C and was associated with dyspnea [18-20].

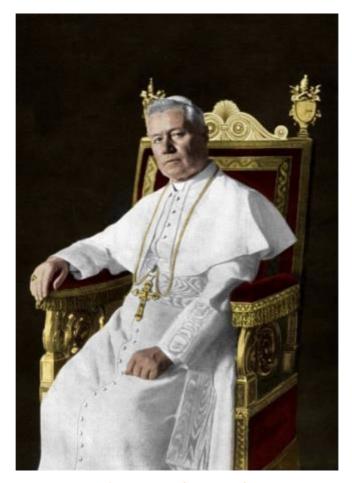


Figure 1: Picture of Pope Pius X (1835-1914), October 1903, from Herder Verlag, Freiburg im Breisgau: Die katholischen Missionen (digitally colored). Image in the public domain, https://commons.wikimedia.org/wiki/File:Pius_X,_by_Francesco_De_Federicis,_1903_(retouched,_colorized).tif

However, we now know that Giuseppe Sarto, since his early years of priesthood, had suffered from gout, which flared painfully from time to time and was tolerated by him. As a pope, for obvious state reasons, he was forced to frequent health checks and restrictive dietary impositions [21]. The disease flared up in August 1920 and was associated with chest pain, fever, nephritis (uncurable at that time). The disease extended to the bronchial tree and caused the pneumonia that killed him [22]. So, the diagnosis was pneumonia, heart failure, pericarditis and uremia due to gout.

He was beatified in 1951 by Pius XII. As far as we know, he was the last gouty pope and after him the disease was never again associated with the papacy.

Discussion

Recent studies have defined gout as a "papal disease" [16]. Pope Pius X is the last in the list of 26 gouty popes of the Catholic Church between the years 590-1914 (Table 1). Gout affected 9.77% of all popes and he was the 18th out of 26 (69.3%) gouty popes to die of a renal cause.

The disease left him, like 22 out of 26 (84.6%) other popes, unable to perform his duties.

No.	Popes	Family name	Start of pontificate	End of pontificate	Inhability to perform	Renal/non renal death**	Age of death
1	St Gregory I	Anici	9/3, 590	3/12, 604	yes	Non-renal	64
2	Sisinnius	NK	1/15, 708	2/4, 708	yes	Non-renal	58
3	Sergius II	Sergio	1/2 844	1/17 847	yes	Non-renal	57*
4	Boniface VI	NK	4/5, 896	4/20 896		Non-renal	NK
5	Honorius IV	Giacomo Savelli	4/2, 1285	4/3, 1297	yes	Non-renal	77*
6	Boniface VIII	Benedetto Caetani	12/24, 1294	10/11, 1303	yes	Non-renal	73
7	Clement VI	Pierre Roger	5/7, 1342	12/6, 1352		Non-renal	62
8	Nicholas V	Tommaso Parentucelli	3/6, 1447	3/24, 1455	yes	Renal	58
9	Callistus III	Alonso de Borja	4/8, 1455	8/6, 1458	yes	Renal	80
10	Pius II	Enea Silvio Piccolomini	8/19, 1458	8/15, 1464	yes	Renal	66
11	Sixtus IV	Francesco della Rovere	8/9, 1471	8/12, 1484	yes	Renal	70*
12	Pius III	Francesco Todeschini Piccolomini	9/22, 1503	10/18, 1503	yes	Renal	64
13	Julius II	Giuliano della Rovere	11/1, 1503	2/21, 1513		Non-renal	70
14	Julius III	Giovanni Maria del Monte	2/7, 1550	3/23, 1555	yes	Non-renal	68
15	Marcellus II	Marcello Cervini degli Spannoni	4/1, 1555	4/30, 1555	yes	Renal	54*
16	Pius IV	Giovanni Angelo Medici di Marignano	12/25, 1559	12/9, 1565	yes	Renal	66
17	Clement VIII	Ippolito Aldobrandini	1/30, 1592	3/3, 1605		Renal	70*
18	Gregory XV	Alessandro Ludovisi	2/9, 1621	7/8, 1623	yes	Renal	69
19	Innocent X	Camillo Pamphilj	10/4, 1644	1/7, 1655	yes	Non-renal	80
20	Clement X	Lorenzo Altieri	4/29, 1670	7/22, 1676	yes	Non-renal	86
21	Innocent XI	Benedetto Odescalchi	9/21, 1676	8/12, 1689	yes	Renal	78
22	Innocent XII	Antonio Pignatelli	7/12, 1691	9/28, 1700	yes	Non-renal	85*
23	Clement XII	Lorenzo Corsini	7/12, 1730	2/6, 1740	yes	Renal	88*
24	Benedict XIV	Prospero Lorenzo Lambertini	8/17, 1740	5/3, 1758	yes	Renal	83*
25	Pius VIII	Francesco Saverio Castiglioni	3/31, 1829	11/30, 1830	yes	Non-renal	69
26	Pius X	Giuseppe Melchiorre Sarto	8/4, 1903	8/20, 1914	yes	Renal	79
All po	ppes		84.6%	50% Renal	71.9 ±9.2#		

Table I: Gouty popes (no. 26). Data for popes nos. 1-25 in reference no.16. (* affected by stroke; ** presumed Renal/Non Renal death; # Mean ±SD; NK = not known).

The mean age at death of the 26 popes listed in Table I was 71.7 ±9.7 years and no difference was found between the age at death of popes who died of a renal cause and those who died of a non-renal cause. Pius X died from an acute infectious disease, which is always a risk for a gouty person. In fact, compared to the general population, gout patients have an increased association with all-cause disease mortality, especially attributed to cardiovascular diseases, cancer, and infectious diseases [23].

In a study by Vargas-Santos et al. [24] enrolling 19,497 people with a new diagnosis of gout and 194,947 controls, a strong association was found between gout and risk of death due to renal disease. Furthermore, a study by Spaetgen et at. [25] investigated the risk of various types of

infections (pneumonia and urinary tract infection), and infection-related mortality in patients with gout using data from the UK Clinical Practice Research Datalink. Their study was the first evaluating the risk of community-acquired infections in patients with gout versus matched controls. Gout was associated with a 34% increased risk of pneumonia. Also, in a national study across the United States [26], the most common infection was pneumonia (52%) in 1998-2000 and sepsis (52%) in 2015-2016. Older age was associated with a greater risk.

There is a strong suspicion, still to prove, of an association between lung infection and the lung dysfunction described in uremia for the first time in 1932 by Ehrich and McIntosh in 3 patients with Bright's disease [27]. They believed that some toxic or metabolic factor resulted in edema and congestion with "formation of an exudate which failed to resorb and then went on to organization" [27], a dysfunction that has been extensively studied in recent years. A restrictive dysfunction, associated with gravity of CKD, was disclosed by Mukai et al. [28], whereas Zoccali et al. [29] have shown, by systematically applying chest ultrasound in ESRD patients, that hidden or clinically manifest lung congestion is exceedingly frequent in this population an may be detected at a preclinical stage.

Gout, probably the first known non-communicable disease, might not represent in principle the best candidate to be discussed in terms of "Theory of Epidemiologic Transition". This theory was advanced in a landmark paper by Abdel R. Omran [30] after infectious diseases were conquered [31] after World War II and degenerative and "man-made diseases" started emerging. Using demographical tools, Omran analyzed the changing patterns of population age distribution in relation to changes in mortality, fertility, life expectancy, causes of death. He identified 3 ages in humankind: the age of famine and pestilence (life expectancy <30 years), the age of "receding pandemics" (life expectancy 30-50 years), and the "age of degenerative diseases and man-made disease" (life expectancy >50 year). The theory has been updated frequently, and finally poverty (initially neglected) has been taken into consideration along with incomes and education [30–36].

This is relevant and makes the theory suitable to explain the high prevalence of gout in popes and the low, but slightly increasing, prevalence in the general population. The data shall be discussed in terms of lifestyles, income and education. It has been shown that affluent and educated people also adopt immoderate lifestyles causing non-communicable diseases associated with morbidity and mortality [30–36]. However, these people, when made aware of the risks, often agree to modify their lifestyles choices, whereas poorer, uneducated people do not. Thus, the latter group tends to experience the morbidity and mortality of the disease (third transition phase) at the time when rich well-educated individuals achieve protection [36].

By applying the above concepts to gout (Figure 2), we can say that popes before 1915 had a high prevalence of gout due to lifestyles choices causing it. These were later corrected through education and gout disappeared. Thus, in 2021, gout has no room in the apostolic palaces. At the same time, poor people, because of undernutrition, working conditions, and frequent movements back and forth from the workplace, were "protected" from gout, and therefore, before 1915, the prevalence of gout was zero. After World War II the general population has become sedentary, while the availability of proteins and the abuse of spirits, wines and other alcoholic beverages, as well as beverages rich in glucose, has sharply increased. Therefore, in the USA, Italy and France, the blood concentration of uric acid has been slightly but steadily increasing; the prevalence of gout is still minimal, but increasing, and will continue to do as long as education fails to encourage healthier lifestyles.

	Lifestyles causing gout		1	tyles ing gout	Prevalence of gout	
	Before 1915	2021	Before 1915	2021	Before 1915	2021
Popes	↑	\	+	^	^	0
General Population	+	1	↑	+	0	†

Figure 2: Lifestyles causing and preventing gout, and trends in the prevalence of gout in popes and general population before 1915 and in 2021

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REFERENCES

- 1. Dalbeth N, Merriman TR, Stamp LK. Lancet 2016; 388(10055): 2039-52 https://doi.org/10.1016/s0140-6736(16)00346-9
- 2. Ragab G, Elshahaly M, Bardin T. Gout: An old disease in new perspective – A review. J Adv Res 2017: 8:495-511. https://doi.org/10.1016/j.jare.2017.04.008
- Igel TF, Krasnokutsky S, Pillinger MH. Recent advances in understanding and managing gout. F1000Res 2017; 6:247. https://doi.org/10.12688/f1000research.9402.1
- 4. Dalbeth N, Choi HK, Joosten LAB, Khanna PP, Matsuo H, Perez-Ruiz F, Stamp LK. Gout. Nature Reviews Disease Primers 2019; 5:69. https://doi.org/10.1038/s41572-019-0115-y
- Martinon F, Petrilli V, Mayor A, Tardivel A, Tschopp J. Gout-associated uric acid crystaks activate the NALP3 inflammasome. Nature 2006: 440:237-41. https://doi.org/10.1038/nature04516
- 6. McCarty DJ, Hollander JL. Identification of urate crystals in gouty synovial fluid. Ann Intern Med 1961; 54:45-64. https://doi.org/10.7326/0003-4819-54-3-452
- 7. Porter R, Rousseau GS. Gout: The Patrician Malady. New Haven, Yale University Press:
- 8. Savica V, Santoro D, Ricciardi B, Ricciardi CA, Calo LA, Bellinghieri G. Morbus dominorum: gout as the disease of lords. J Nephrol 2013; 26(S22):113-16. https://doi: 10.5301/jn.5000349
- 9. McKeown T. The origins of human disease. Oxford, Blackwell: 1988.
- 10. Retief FP, Cilliers L. Disease and causes of death among popes. Acta Theologica 2006: 26(2):S7. https://doi.org/10.4314/actat.v26i2.52576
- 11. De Santo NG, Bisaccia C, De Santo Causes of death due to disease of the genito-urinary system and of the heart among 264 popes in the years 65-2005 AD: First approach. Nephrol Dial Transplant 2019; 34(S1): gfz103.SP804. https://doi.org/10.1093/ndt/gfz103.SP804
- 12. De Santo NG, Bisaccia C, De Santo LS. Deaths caused by cardiorenal disease among 264 popes from St. Peter to St. John Paul II. Hellenic Nephrology 2019; 31:158.
- 13. De Santo NG, Bisaccia C, De Santo LS. Papal deaths caused by cardiorenal disease. First Approach. Arch Hell Med 2020; 37(S2):177-81.
- 14. Bisacccia C, De Santo LS, De Santo NG. Gout a papal disease: a study in 20 pontiffs (540-1830. Nephrol Dial Transplant 2020; 35(S3):gfaa144.P1836. https://doi.org/10.1093/ndt/gfaa144.P1836
- 15. De Santo N, Bisaccia C, De Santo (2021). Renal stone disease in 193 pontiffs from Vigilius to Pius VIII (537-1830). Nephrol Dial Transplant 2021; 36(S1):gfab105.001. https://doi.org/10.1093/ndt/gfab105.001

- 16. De Santo NG, Bisaccia C, De Santo LS. Gout: a papal disease-a historical review of 25 gouty popes (34-2005 AD). J Nephrol 2021; 34(5):1565-67. https://doi.org/10.1007/s40620-021-01117-8
- 17. Aubert R. Documents relatifs au movement catholique italien sous le pontificat de S. P. X. ibid., XII (1958), pp. 202-43, 304-70. In: Pius X, Enciclopedia Treccani online. Accessed on December 9, 2021.
- 18. Merry del Val R. San Pio X. Verona, Fede e Cultura: 2012.
- 19. Occelli P. Il beato Pio X. Roma, ed. Paoline: 1951, p. 237.
- 20. Siccardi C. San Pio X. Roma, San Paolo ed.: 2014. p. 369.
- 21. dal Gal G. Pio X il papa santo. Firenze, Libreria Editrice: 1940, p. 283.
- 22. Sanguinetti O. Pio X: Un pontefice santo alle soglie del secolo breve. Milano, Sugarco Edizioni: 2014, p. 283
- 23. Disveld IJM, Zoakman S, Jansen TLTA, Rongen GA, Kienhorst LBE, Janssens HJEM, Fransen J, Janssen M. Crystal-proven gout patients have an increased mortality due to cardiovascular diseases, cancer, and infectious diseases especially when having tophi and/or high serum uric acid levels: a prospective cohort study. Clin Rheumatol 2019; 38(5):1385-91. https://doi.org/10.1007/s10067-019-04520-6
- 24. Vargas-Santos AB, Neogi T, da Rocha Castelar-Pinheiro G, Kapetanovic MC, Turkiewicz A. Cause-Specific Mortality in Gout: Novel Findings of Elevated Risk of Non-Cardiovascular-Related Deaths. Arthritis Rheumatol 2019; 71(11):1935-42. https://doi.org/10.1002/art.41008
- 25. Spaetgens B, de Vries F, Driessen JHM, Leufkens HG, Souverein PC, Boonen A, van der Meer JWM, Joosten LAB. Risk of infections in patients with gout: a population-based cohort study. Scientific Reports 2017; 7:1429. https://doi.org/10.1038/s41598-017-01588-5
- 26. Singh JA, Cleveland JD. Serious Infections in Patients With Gout in the US: A National Study of Incidence, Time Trends, and Outcomes. Arthritis Care Res 2020; 73(6):898-908. https://doi.org/10.1002/acr.24201
- 27. Ehrich W, McIntosh JF. The pathogenesis of bronchiolitis obliterans. Arch Path 1932; 13:69-
- 28. Mukai H, Ming P, Lindholm B, Heimbürger O, Barany P, Anderstam B, Stenvinkel P, Qureshi AR. Restrictive lung disorder is common in patients with kidney failure and associates with protein-energy wasting, inflammation and cardiovascular disease. PLoS One 2018: 13(4):e0195585.
 - https://doi.org/10.1371/journal.pone.0195585

- 29. Zoccali C, Tripepi R, Torino C, Bellantoni M, Tripepi G, Mallamaci F. Lung congestion as a risk factor in end-stage renal disease. Blood Purif. 2013; 36(3-4):184-91. https://doi.org/10.1159/000356085
- Mc Keown R. The epidemiologic Transition: Changing Patterns of Mortality and Population Dynamics. Am J Lyfestyle Med 2009; 3(S1): 19S-26S. https://doi.org/10.1177/1559827609335350
- 31. Omran AR. The epidemiologic transition. A theory of the Epidemiology of Population Change. Milbank Memorial Fund Quarterly 1971; 49(4):509-38.
- 32. Caldwell JC. Population health in transition. Bull World Health Org 2001; 71(1):159-60.
- 33. Pearson TA. Education and income: double edged swords in the epidemiologic transition of cardiovascular disease. Ethnicity & Disease 2003; 13(S2):158-63.

- 34. Pearson TA. Socioeconomic status and cardiovascular disease in rural population. In Stamler J, Hazuda H (eds). Report on the conference on Socioeconomic Status and cardiovascular disease. Washingtoon DC, National Heart, Lung, and Blood Institute: 1995, pp. 101-08.
- Marmot MG, Smith GA, Stansfeld S, Patel C, et al. Health inequalities among British civil servants. Lancet 1991; 337:1387-93. https://doi.org/10.1016/0140-6736(91)93068-k
- 36. Kaplan G, Keil J. Socioeconomic factors and cardiovascular disease: a review of the literature. Circulation 1993; 88:1973-88. https://doi.org/10.1161/01.cir.88.4.1973