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What is the absolute risk of developing diabetes mellitus in patients with glucocorticoid-treated polymyalgia rheumatica and giant cell arteritis? a systematic review and meta-analysis

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WHAT IS THE ABSOLUTE RISK OF DEVELOPING DIABETES MELLITUS IN PATIENTS WITH GLUCOCORTICOID-TREATED POLYMYALGIA RHEUMATICA AND GIANT CELL ARTERITIS? A SYSTEMATIC REVIEW AND META-ANALYSIS
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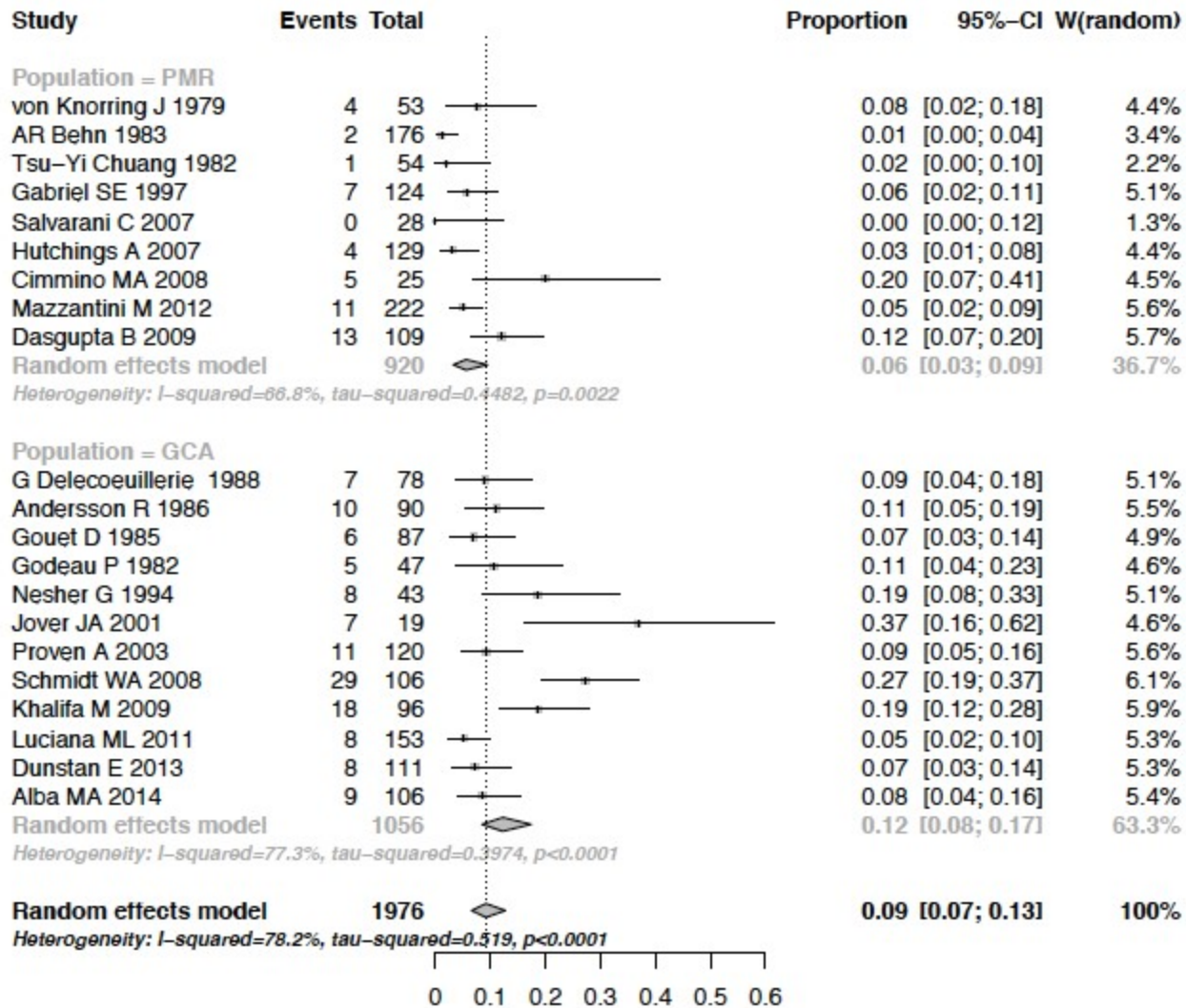
Background: Polymyalgia rheumatica (PMR) and giant cell arteritis (GCA) are treated with glucocorticoids (GCs) but long-term GC use is associated with diabetes mellitus (DM). The absolute incidence of this serious complication in this patient group remains unclear.

Objectives: To quantify the absolute risk of GC-induced DM in PMR and GCA in published literature.

Methods: We identified literature from inception to February 2016 reporting diabetes following exposure to oral GC in patients with PMR and/or GCA without pre-existing diabetes. A random-effects meta-analysis was performed to summarise the literature. Risk of bias was assessed using the Cochrane Collaboration tool.

Results: 21 eligible publications were identified. In studies of patients with GCA, mean cumulative GC dose was almost two times higher than in studies of PMR (8.9g vs 5.0g), with slightly longer treatment duration but much longer duration of follow-up (8.8years vs 4.4years). The incidence proportion (cumulative incidence) of patients who developed new-onset DM was 6% (95%CI: 3-9%) for PMR and 12% (95%CI: 8-17%) for GCA. Heterogeneity between studies was high (I^2=78.2%), as there were differences in study designs, patient population, geographical locations and treatment strategies. Based on UK data on incidence rate of DM in the general population[1], the expected background incidence rate of DM over 4.4 years in PMR patients and 8.8 years in GCA patients (the duration of follow-up) would be 4.8% and 9.7%, respectively. Very little information on predictors of DM in PMR or GCA patients was found. The overall risk of bias was high for many of the observational studies, especially relating to definition and recording of outcome and prognostic variables.

Image/graph:

Study	Events	Total	Proportion	95%–CI	W(random)
Population = PMR					
von Knorring J 1979	4	53	0.08	[0.02; 0.18]	4.4%
AR Behn 1983	2	176	0.01	[0.00; 0.04]	3.4%
Tsu–Yi Chuang 1982	1	54	0.02	[0.00; 0.10]	2.2%
Gabriel SE 1997	7	124	0.06	[0.02; 0.11]	5.1%
Salvarani C 2007	0	28	0.00	[0.00; 0.12]	1.3%
Hutchings A 2007	4	129	0.03	[0.01; 0.08]	4.4%
Cimmino MA 2008	5	25	0.20	[0.07; 0.41]	4.5%
Mazzantini M 2012	11	222	0.05	[0.02; 0.09]	5.6%
Dasgupta B 2009	13	109	0.12	[0.07; 0.20]	5.7%
Random effects model		920	0.06	[0.03; 0.09]	36.7%
Heterogeneity: I–squared=66.8%, tau–squared=0.4482, p=0.0022					
Population = GCA					
G Delecoeuillerie 1988	7	78	0.09	[0.04; 0.18]	5.1%
Andersson R 1986	10	90	0.11	[0.05; 0.19]	5.5%
Gouet D 1985	6	87	0.07	[0.03; 0.14]	4.9%
Godeau P 1982	5	47	0.11	[0.04; 0.23]	4.6%
Nesher G 1994	8	43	0.19	[0.08; 0.33]	5.1%
Jover JA 2001	7	19	0.37	[0.16; 0.62]	4.6%
Proven A 2003	11	120	0.09	[0.05; 0.16]	5.6%
Schmidt WA 2008	29	106	0.27	[0.19; 0.37]	6.1%
Khalifa M 2009	18	96	0.19	[0.12; 0.28]	5.9%
Luciana ML 2011	8	153	0.05	[0.02; 0.10]	5.3%
Dunstan E 2013	8	111	0.07	[0.03; 0.14]	5.3%
Alba MA 2014	9	106	0.08	[0.04; 0.16]	5.4%
Random effects model		1056	0.12	[0.08; 0.17]	63.3%
Heterogeneity: I–squared=77.3%, tau–squared=0.3974, p<0.0001					
Random effects model		1976	0.09	[0.07; 0.13]	100%
Heterogeneity: I–squared=78.2%, tau–squared=0.519, p<0.0001					

0   0.1   0.2   0.3   0.4   0.5   0.6

Conclusions: Physicians should screen patients treated for PMR/GCA for DM but it remains unclear what is the time-period of greatest risk and the influence of risk factors. Our meta-analysis produced plausible estimates of DM incidence in patients with PMR and GCA but there is insufficient published data to allow precise quantification of the DM risk or, crucially, which patients are at greatest risk.

References: [1]Sharma M et al. BMJ Open, 2016. 6(1): p.e010210

Disclosure of Interest: None declared