Trends in Prevention of Glucocorticoid-Induced Osteoporosis

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ABSTRACT. ***Objective.*** To determine longitudinal patterns and predictors for the utilization of bone mass measurements and anti-osteoporotic medications in the prevention of glucocorticoid-induced osteoporosis.

Methods. Within a managed care population of 7 million persons, we identified 3125 adult men and women who had initiated longterm glucocorticoid therapy (≥ 7.5 mg/day of prednisone equivalent for > 6 mo). The study population was examined by 3 biennial intervals between years 1996 and 2001 for receipt of a bone mass measurement and use of anti-osteoporotic medication (bisphosphonate, calcitonin, raloxifene, hormone replacement therapy).

Results. Receipt of a bone mass measurement increased among postmenopausal women from 10% in 1996-97 to 19% in 2000-01, but remained below 6% in all biennial intervals among women under age 50 and men. The use of anti-osteoporotic medication was most common among postmenopausal women, where it approached 50%. The largest absolute increase in anti-osteoporotic medication utilization was among women ages 65 and over, increasing from 24% in 1996-97 to 44% in 2000-01. The specialty of physician providing care was associated with receipt of both testing and treatment. Odds of receipt of a bone mass measurement and anti-osteoporotic medication were 3 to 4 times greater among patients of rheumatologists compared to those of internists or family practitioners.

Conclusion. Among patients initiating longterm glucocorticoid therapy, the proportion of individuals receiving a bone mass measurement or anti-osteoporotic medication remains relatively low, but has improved temporally among postmenopausal women. (First Release July 15 2006; J Rheumatol 2006;33:1651–7)

Key Indexing Terms:
GLUCOCORTICOIDS OSTEOPOROSIS QUALITY OF CARE PRACTICE GUIDELINES

Approximately 1% of the adult population uses glucocorticoids to treat a variety of allergic and inflammatory diseases[1]. Regular use of glucocorticoids leads to a loss of bone mass and an increased risk for fractures of the hip, vertebrae, and wrist. The magnitude of this increased risk for fractures ranges from 1.5 to 5.0-fold, dependent on dose and duration of glucocorticoid use[2,3]. This resultant increase in fractures has a significant public health influence[3].

Options to prevent and treat glucocorticoid-induced osteoporosis include lifestyle changes, exercise, calcium and vitamin D supplementation, hormone replacement therapy, bisphosphonates, and calcitonin[4-6]. In addition to the 1997 US Congressional Act allowing Medicare reimbursement for testing bone mineral density (BMD) among patients taking glucocorticoids, there have also been advances in the measurement of BMD. Bone mass measurement may influence treatment decisions for chronic users of glucocorticoids and influence therapeutic decision-making. Despite international guidelines and recommendations advocating prevention options and bone mass measurement, their utilization in actual practice has been low based on studies examining practices in the late 1990s[7-13].

It is unknown if promulgation of growing scientific evidence about glucocorticoid-induced osteoporosis coupled with gradual improvements in postmenopausal osteoporosis care in general[14] have led to an increase in utilization of prevention practices for glucocorticoid-induced osteoporosis. To assess changes in utilization and to examine factors associated with greater utilization of prevention modalities, we conducted a retrospective cohort study using an administrative claims database among longterm users of glucocorticoids and analyzed longitudinal patterns from 1996 through 2001. Despite growing evidence, we hypothesized continued low primary and secondary preventive care of glucocorticoid-induced osteoporosis along with significant practice pattern variation by physician specialty.

MATERIALS AND METHODS

Data source. All data were contained in a commercial database providing linkable information on demographics, medical claims, and pharmaceutical claims for 7 million individuals in 20 US states enrolled in either a private

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insurance program or Medicare risk plan between January 1995 and December 2002 (Protocare Sciences, Constella Group, Durham, NC, USA). From this data source, we identified a study population of patients receiving longterm glucocorticoid therapy. We also identified those patients who had received a prescription treatment for an anti-osteoporosis medicine or a BMD test.

Study population. We identified patients who were 18 years of age or older who had at least 2 oral glucocorticoid pharmacy claims within 6 months. We restricted our focus to patients who had newly initiated (no prior use in a 12 mo period) longterm therapy (≥ 7.5 mg prednisone equivalent for treatment duration of ≥ 6 mo) between 1996 and 2001, consistent with the 1996 American College of Rheumatology (ACR) guidelines for management of glucocorticoid-induced osteoporosis[4]. An individual's use of oral, systemic glucocorticoid therapy was identified within the pharmaceutical claims by searching for National Drug Codes specific to the following active ingredients: triamcinolone, prednisone, prednisolone, methylprednisolone, dexamethasone, budesonide, betamethasone, cortisone, and hydrocortisone. Eligible patients were also required to have continuous medical and drug coverage for 12 months before and after initiation of longterm glucocorticoid therapy. We excluded patients who had an organ transplant procedure or malignant neoplasm diagnoses during the 12 months before and after initiation of glucocorticoid therapy. These patients were excluded in order to focus on a more homogeneous population and to remove the possibility that confounding factors unique to these patients might have influenced the study outcome.

Outcomes. We focused on both primary and secondary prevention strategies and, similar to the osteoporosis Health Plan Employer Data and Information Set (HEDIS) measure[15], included bone mass measurement as an outcome of interest that might alter therapeutic decision-making. We identified persons who had received a bone mass measurement within 12 months before and after initiating glucocorticoid therapy based on the relevant Current Procedural Terminology (CPT) codes (Appendix). Prescription therapies potentially intended for prevention of glucocorticoid-induced osteoporosis were categorized as either hormone replacement therapy or non-estrogen therapy (bisphosphonate, calcitonin, raloxifene). Receipt of one or more prescription treatments for glucocorticoid-induced osteoporosis within 12 months after initiating glucocorticoid therapy was identified within pharmaceutical claims by searching for National Drug Codes specific to the following active ingredients: risedronate, alendronate, calcitonin, raloxifene, etidronate, pamidronate, and the hormone replacement therapies (estrogen, estradiol, estrone, and estropipate). Using our data source, we were not able to capture the use of nonprescription osteoporosis preventive strategies (i.e., over-the-counter use of calcium and vitamin D).

Potential factors associated with outcomes of interest. Demographic variables examined included sex and age category (18–49, 50–64, or 65+). Clinical history variables included the occurrence within 12 months before initiation of glucocorticoid therapy of nontraumatic fractures and illnesses related to glucocorticoid use. Nontraumatic fractures were identified by using International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM codes) (Appendix). Illnesses for which steroids would potentially be a component of treatment were identified using ICD-9-CM codes (Appendix). We were particularly interested in the association of the physician specialty of the provider who diagnosed the above illness with the receipt of prescription therapies and bone mass. Providers were characterized as family practitioner/internist, rheumatologist, gastroenterologist, pulmonologist, other specialist, or unknown.

Data analysis. Within the study population, we defined 3 mutually exclusive cohorts based upon biennial periods of dates for initiation of glucocorticoids. Rates of utilization for prevention therapy of glucocorticoid-induced osteoporosis were calculated by dividing the number of people receiving prevention therapy by the number of people on longterm glucocorticoid therapy. For the most recent biennial cohort, years 2000-01, forward stepwise logistic regression was used to define the association of provider type with receipt of therapy for glucocorticoid-induced osteoporosis after adjustment for important covariates. First, a univariate statistic (chi-square or t test, as appropriate) was used to analyze each dependent variable and independent variable pair to identify variables for inclusion in a multivariate model (i.e., statistical significance < 0.20). Second, a multivariable model with all significant independent variables and the outcome variable was used to obtain adjusted odds ratios (OR) and their 95% confidence intervals (CI). The Hosmer-Lemeshow goodness of fit was used to assess fit of the model[16]. SAS statistical software (SAS, Cary, NC, USA) was used to compute the summary statistics and to conduct the logistic modeling (Proc Logistic).

RESULTS

We identified a study population of 3125 men and women who initiated longterm glucocorticoid therapy. The 3 period cohorts (Table 1) had similar characteristics for gender (percentage of women ranged from 59% to 63%), for the proportion with pulmonary illnesses as a potential reason for glucocorticoid therapy (range 38%–42%), and for mean daily dosage of glucocorticoid (11 ± 6 mg/day). The greatest difference between the temporal cohorts was the percentage of people aged 65 and over (range 40%–52%).

Receipt of a bone mass measurement. Receipt of a bone mass measurement either one year before or after initiation of glucocorticoid use was also associated with age and sex of the patient. Receipt was most common among women aged 50 years and over and least common among younger women and men (Figure 1). Over the course of the 6-year study period the utilization of bone mass measurement among women aged 50 and over increased from 10% to 19% (chi-square p value < 0.01). In contrast, among all men and women aged 18–49, the proportion receiving a bone mass measurement was less than 6% for all biennial periods.

In addition to being seen by a rheumatologist, patient characteristics associated with an increased likelihood of receiving a bone mass measurement in the 2000–01 biennial period (Table 2) were female sex, age over 50 years, and a history of a fracture.

Table 1. Characteristics of study population. Pulmonary includes diagnoses for asthma or chronic obstructive pulmonary disease. Rheumatalogic includes diagnoses for giant cell arteritis, inflammatory myopathy, polymyalgia rheumatica, rheumatoid arthritis, sarcoidosis, systemic lupus erythematosus, and Wegener granulomatosis. Gastrointestinal includes diagnosis for inflammatory bowel disease. Results are expressed as percentages unless otherwise noted.

Characteristic	1996–97, n = 959	1998–99, n = 1254	2000–01, n = 912
Sex, women	62	63	59
Age group, yrs			
18–49	35	30	26
50–64	25	22	22
65 and over	40	48	52
Inflammatory diagnosis			
Pulmonary	38	42	38
Rheumatologic	24	25	24
Gastrointestinal	7	6	6
All other	31	27	32
Mean glucocorticoid dose (mg/day), ± SD	11 ± 6	11 ± 6	11 ± 6

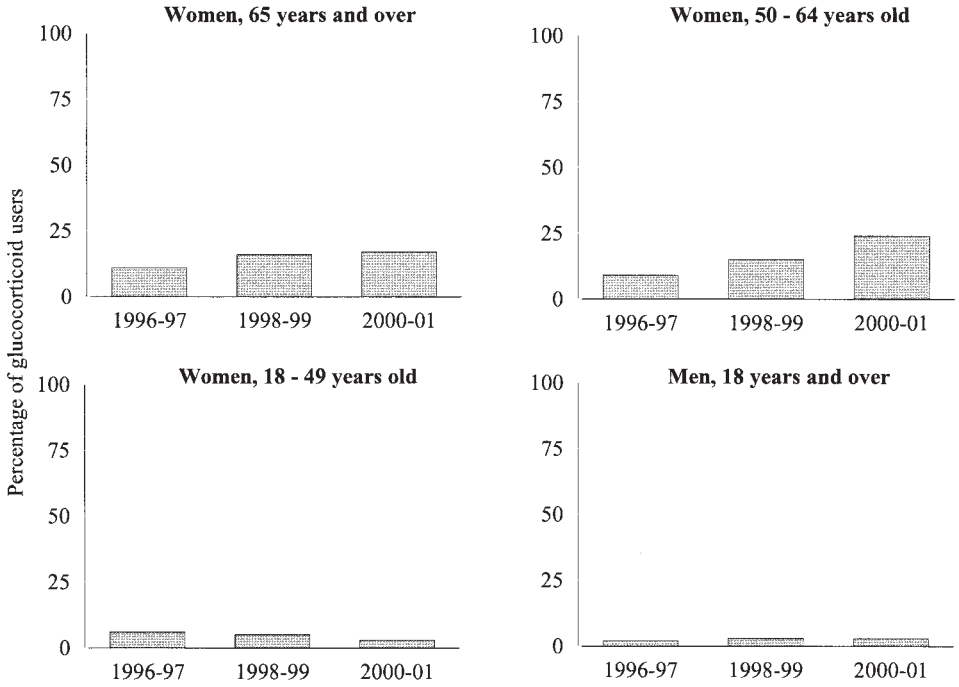

Figure 1. Trends in receipt of bone mass measurement among patients receiving glucocorticoids.

Table 2. Factors associated with the receipt of a bone mass measurement either one year before or one year after initiation of glucocorticoid use, 2000–01. The adjusted odds ratio is based on forward-stepwise logistic regression with independent variables. Hosmer-Lemeshow goodness of fit test, p = 0.39.

Characteristic	No. of Patients (n = 912)	Receiving Anti-Osteoporotic Medication, n (%)	Adjusted Odds Ratio	95% CI
Sex				
Men	373	13 (3)	Referent	—
Women	539	84 (16)	2.3	1.2–4.8
Age group, yrs				
18–49	240	8 (3)	Referent	—
50–64	198	32 (16)	3.5	1.5–8.8
65 and over	474	57 (12)	2.3	1.0–5.5
Glucocorticoid dose, 1 year cumulative, mg				
Lowest tertile, 1350–2245	304	28 (9)		
Middle tertile, 2246–3300	306	39 (13)	Not Significant	
Highest tertile, 3301–12300	302	30 (10)		
Fracture history*	62	29 (47)	8.4	4.2–16.7
Non-estrogen anti-osteoporotic therapy*	58	23 (40)	2.3	1.1–4.7
Hormone replacement therapy use*	150	46 (31)	3.9	2.3–6.7
Physician specialty				
Internist/family practitioner	362	34 (9)	Referent	—
Rheumatologist	79	24 (30)	4.0	2.0–8.0
Gastroenterologist	23	1 (4)	0.5	0.0–2.7
Pulmonologist	45	6 (13)	1.9	0.6–5.1
Other medical specialty	244	20 (8)	1.1	0.6–2.0
Not identified	159	12 (8)	1.3	0.3–1.6

* In year prior to glucocorticoid initiation.

Use of anti-osteoporotic medication. Temporal trends in the use of anti-osteoporotic medication within the year following initiation of glucocorticoid therapy were associated with the age and sex of the patient (Figure 2). Receipt of an anti-osteoporotic medication approached 50% of women aged 50 and over and was less frequent among younger women and among men. Over the course of the 6-year study period, the largest absolute increase in the proportion of glucocorticoid users receiving an anti-osteoporotic medication was among older women (65+). In contrast, among women under age 65 and all men, the absolute change in receipt within the 6-year study period was less than 5%.

Temporal trends in the type of anti-osteoporotic medication were also associated with age and sex of the patient. Among older women (65+), there was increased use over time of non-estrogen (8% to 25%; $p < 0.01$) and estrogen therapy (17% to 25%; $p < 0.01$). Among middle-aged women (50–64), there was increased use of non-estrogen therapies (5% to 18%; $p < 0.01$).

Physician characteristics as well as patient demographic and clinical characteristics were significantly associated with a higher likelihood of use of non-estrogen therapies in the 2000-01 biennial period (Table 3). In addition to seeing a rheumatologist, characteristics related to an increased likelihood of using a non-estrogen therapy included female sex, age greater than 65, higher cumulative dose of glucocorticoid, and history of a bone mass measurement.

DISCUSSION

We found that utilization of prescription therapies and bone mass measurements for prevention of glucocorticoid-induced osteoporosis remains relatively low among longterm users of glucocorticoids in managed care populations in the US. A strength of our study was the ability to analyze temporal trends in prevention practices from 1996 through 2001. We found utilization of prescription therapies and bone mass measurement for glucocorticoid-induced osteoporosis increased significantly among postmenopausal women, but not among women under age 65 or among men.

The 1996 through 2001 utilization rates of prevention practices for glucocorticoid-induced osteoporosis in our study are somewhat consistent with utilization rates of a number of past cross-sectional studies (Table 4). Estimates of the utilization of a non-estrogen anti-osteoporotic prescription have ranged from 11% to 17% among women and from 4% to 5% among men[8,9]. Estimates of the receipt of a bone mass measurement have ranged from 12% to 20% among women and 1% to 5% among men, with the exception of a study by Yood, *et al*[12]. In that study of 224 patients in a single managed care plan, utilization of bone mass measurement was 43% among women

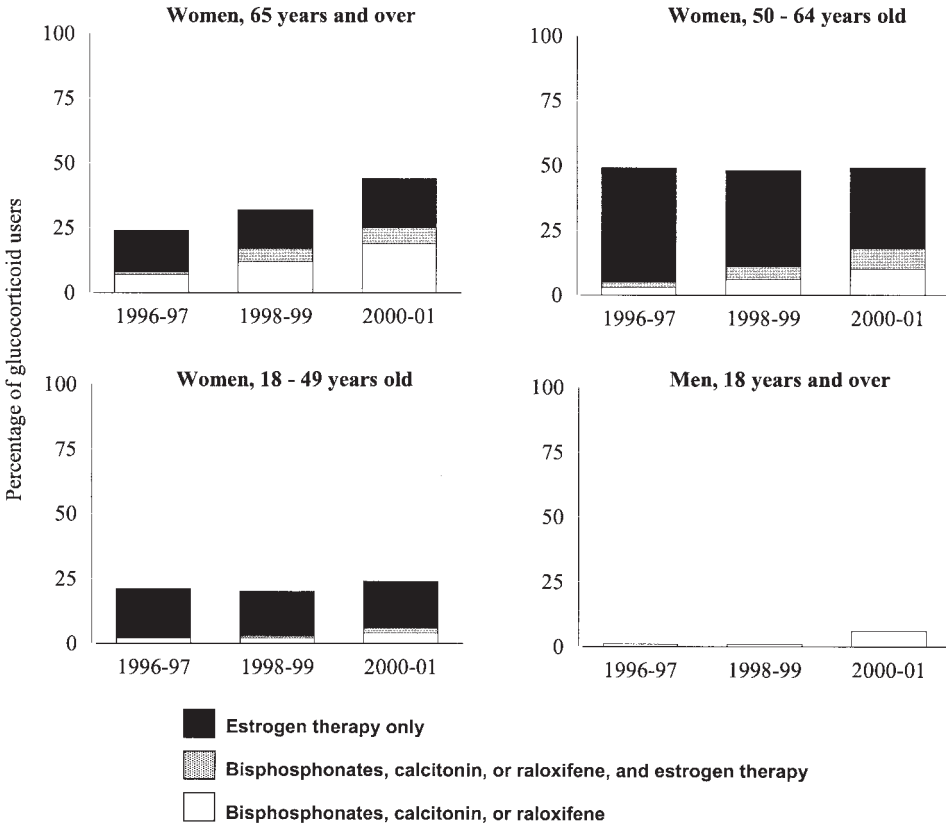

Figure 2. Trends in use of anti-osteoporotic medication among patients receiving glucocorticoids.

Table 3. Factors associated with the use of non-estrogen anti-osteoporotic therapy in the year after initiation of glucocorticoid use, 2000–01.

Characteristic	No. of Patients (n = 912)	Receiving Anti-Osteoporotic Medication, n (%)	Adjusted Odds Ratio	95% CI
Sex				
Men	373	23 (6)	Referent	—
Women	539	103 (19)	2.8	1.7–4.9
Age group, yrs				
18–49	240	15 (6)	Referent	—
50–64	198	23 (12)	1.1	0.5–2.5
65 and over	474	88 (19)	2.3	1.3–4.6
Glucocorticoid dose, 1 year cumulative, mg				
Lowest tertile, 1350–2245	304	31 (10)	Referent	—
Middle tertile, 2246–3300	306	42 (14)	1.3	0.7–2.4
Highest tertile, 3301–12300	302	53 (18)	2.4	1.3–4.3
Fracture history*	62	28 (45)	NS	
Bone mass measurement history*	32	22 (69)	3.2	1.1–9.6
Hormone replacement therapy use*	150	34 (23)	NS	
Physician specialty				
Internist/family practitioner	362	36 (10)	Referent	—
Rheumatologist	79	24 (30)	3.5	1.7–7.0
Gastroenterologist	23	2 (9)	1.6	0.2–6.0
Pulmonologist	45	6 (13)	1.0	0.3–2.9
Other medical specialty	244	33 (14)	1.5	0.8–2.8
Not identified	159	25 (16)	1.3	0.6–2.5

* In year prior to glucocorticoid initiation. † Hosmer-Lemeshow goodness of fit test p = 0.12.

Table 4. Comparison of data on prevention practices for glucocorticoid-induced osteoporosis. Results expressed as percentages.

Reference	Healthcare Setting	Population Sex, Age	Period	Estrogen Replacement or Anti-Osteoporotic	Non-Estrogen, Anti-Osteoporotic	Receiving Bone Mass Measurement
Current study	US health plans	Women, 50+	1996–01	39	15	15
		Women,		21	3	5
		men, < 50		4	4	3
Chantler[8]	UK-GPRD	Women, 50+	1997		17	20
Yood[12]	US-HMO	Women,	1997–98	35		43
		men		10		15
Ettinger[9]	US health plans	Women,	1998–99		11	12
		men			5	1
Mudano[10]	US health plans	Women, 50+	1995–97	41		16
		Women,		14		7
		men, < 50		9		5

GPRD: General Practice Research Database, HMO: health maintenance organization.

and 15% among men. We found that women above age 50 were receiving non-estrogen anti-osteoporotic therapies in the year 2001 at a rate slightly above that seen in most of these older studies, although differences in the populations studied and the methods of data collection preclude accurate direct comparisons. Our study also differed from these other studies by its broad representation of persons throughout the US, availability of information on physician specialty, and our assessment of changing utilization patterns over time.

In our study, estrogen was the principal prevention therapy for women aged 50–64. Estrogen use has declined significantly following conclusion of the Women's Health Initiative[17]. This finding, coupled with the lack of a temporal increase in bone mass measurement or of anti-osteoporotic medication among this group of women in our study, could put these women at even higher risk of bone complications of glucocorticoid therapy in the future.

In addition to use of specific anti-osteoporosis agents and bone mass measurement, we also examined the variation in prevention practices by medical practice and cumulative dose of glucocorticoids. Consistent with past studies by us[10] and others[9,12], patients prescribed glucocorticoids by rheumatologists continue to be significantly more likely to receive both bone mass measurement and prescription anti-osteoporosis

therapies. Physician practice pattern variations have also been found in the management of other musculoskeletal disorders[18,19].

We also found that patients who had received a higher cumulative dose of glucocorticoids appropriately received more preventive care. Although we have previously found an increased association in prevention practices among these higher-risk persons[20], a number of reports suggest that these patients are not always likely to get needed evaluation and treatment[7-13].

Our finding that there is a discordance between ACR[4] and other international guidelines[5,21-25] and actual practice patterns leads us to speculate that physician practices may be a modifiable factor to reduce this discrepancy. In addition to physicians being unaware of these guidelines, reasons for low guideline adherence include physicians' time constraints, lack of adequate incentives, and lack of resources and facilities for performing bone mass measurement[26]. Additionally, there may be lack of generalist-specialist communication, leading some physicians to defer responsibility to their colleagues. We contend, however, that any physician managing patients receiving chronic glucocorticoids has responsibility for monitoring and attempting to prevent the possible consequences of their longterm use.

It should be noted that while it is not possible to define an absolute standard of care in this or any other area of medicine, and that patient care should be individualized, the overall rates of clinical practices for the prevention of glucocorticoid-induced osteoporosis in our study were relatively low. To put this in a more general perspective, in the US, adherence to many of the benchmarks of clinical practice for chronic disease management routinely exceed 80%[15].

Despite our large sample size, information on temporal trends, and the good generalizability of our study, our findings have potential limitations. One limitation is that only information on prescription therapies was available from our data source. Data on vitamin D and calcium, over-the-counter medications recommended for glucocorticoid-induced osteoporosis prevention, were not available. Thus, the rates of medication utilization in this report may slightly underestimate true prevention. However, adequate calcium and vitamin D are necessary for nearly all glucocorticoid users, but for most they are not sufficient for glucocorticoid-induced osteoporosis prevention[27]. Another limitation of our report is that it is unknown if estrogen therapy was used for glucocorticoid-induced osteoporosis or another indication (e.g., postmenopausal osteoporosis). In this way, our estimate of prescription medication use represents a conservative assessment of potential agents likely to have been prescribed for a bone benefit. When working with administrative claims and linked pharmacy data, it is possible that persons could have received osteoporosis care not captured by our data. Despite these limitations, our findings are from a large population-based sample representative of privately insured patients in the US.

In conclusion, despite steadily increasing evidence of effective preventive therapies among patients initiating longterm glucocorticoid therapy, prevention of glucocorticoid-induced osteoporosis remains relatively low and varies

Appendix

Exclusion criteria for study population	CPT codes 32854, 33935, 33945, 38240, 38241, 38242,
History of organ transplant procedure	44135, 44136, 47135, 47136, 48160, 48554, 48556, 50360, 50365, 50370, 50380, 60512
History of malignant neoplasm diagnoses	ICD-9-CM codes 140–208
Receipt of a bone mass measurement	CPT codes 76070, 76075, 76076, 76078, 78350, 78351 or ICD-9-CM code 88.98
Nonvertebral or pathologic fractures	ICD-9-CM codes 733.1, 807, 808, 810, 811, 812, 813, 814, 820, 821, 822, 823, 824 — excluding 4-digit codes specific for open fractures
Illnesses potentially related to glucocorticoid use	ICD-9-CM codes
Rheumatoid arthritis	714.x
Chronic obstructive pulmonary disease	490, 491.x, 492.x, 494.x, 496.x
Asthma	493.x
Systemic lupus erythematosus	710.0
Inflammatory bowel disease	555.x, 556.x
Polymyalgia rheumatica	725
Multiple sclerosis	340
Wegener granulomatosis	446.4
Nephrotic syndrome	581.x
Myasthenia gravis	358.0
Sarcoidosis	135
Giant cell arteritis	446.5
Pemphigus	694.4
Inflammatory myopathy	710.3, 710.4

CPT: current procedural terminology; ICD-9-CM: International Classification of Diseases, ninth revision, Clinical modification.

by provider specialty, with rheumatologists prescribing more anti-osteoporotic agents and ordering more bone mass measurements than other physicians. Future research and education should address these gaps in prevention among these high-risk patients.

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