OPEN ACCESS

GAVIN PUBLISHERS

Journal of Orthopedic Research and Therapy

Mouhamed Dieng M, et al. J Orthop Ther: JORT-179.

Research Article

DOI: 10.29011/2575-8241. 000079

How Do Doctors Prevent Cortisone Osteoporosis in Dakar?

Mouhamed Dieng[1*], Djiba B[1], Ndao AC[1], Sow M[1], Kane BS[1], Ba M[1], Diouf C[3], Ndour MA[1], Fall BC[2], Niasse M[3], Diagne N[1], Faye A[1], Ndongo S[2], Pouye A[1]

[1]Department of Internal Medicine, Aristide Le Dantec Hospital, Senegal

[2]Department of Internal Medicine, Dalal Jam Hospital, Dakar Senegal

[3]Department of Rheumatology, Aristide Le Dantec Hospital, Senegal

[*]**Corresponding author:** Mouhamed Dieng M, Department of Internal Medicine, Aristide Le Dantec Hospital, BP 6003, Dakar-Étoile, Senegal. Tel : +221-772762836; Email: sigmadiez@hotmail.fr

Citation: Dieng M, Djiba B, Ndao AC, Sow M, Kane BS, et al. (2018) Swing Mechanics of the Offside Forehand in Professional Female Polo Athletes. J Orthop Ther: JORT-179. DOI: 10.29011/2575-8241. 000079

Received Date: 07 February, 2018; **Accepted Date:** 20 February, 2018; **Published Date:** 06 March, 2018

Abstract

Introduction: Cortisonic osteoporosis is the most common secondary osteoporosis and the leading cause of osteoporosis in young adults. It is a major public health problem because of the potential severity of certain fractures, which increases the risk

Materials and Methods: In order to study the prevention of this disease by our doctors in patients receiving prolonged corticosteroid treatment, we conducted a cross-sectional, multicenter, descriptive study from June 1st, 2017 to August 1st, 2017. It focused on a questionnaire given to medical specialists of all medical specialties and practicing in the University Hospitals of Dakar.

Results: 170 doctors were interviewed. Dermatologists and internists were mostly found 19.4% and 18.8% or 33 and 32 doctors. Systemic autoimmune diseases alone accounted for 48% of prescription reasons. Prednisone was the most prescribed (88% of cases). The immunosuppressive dose of 1 mg / kg was most often prescribed. Most adjuvant measures to prolonged systemic corticosteroid therapy are very heterogeneously prescribed by practitioners. Thus, while vitamin-calcium supplementation was routinely prescribed by a few rare physicians (38%), most never performed an initial assessment of fracture risk (52%), and never prescribed general preventive measures or anti-osteoporosis treatment (biphosphonate, teraparatide).

Conclusion: Due to the number of patients with systemic disease who also have risk factors for osteoporosis and are receiving extended corticosteroid therapy, homogenization of practices in line with the recommendations would significantly reduce morbidity and mortality and costs associated with osteoporosis. cortisone osteoporosis.

Introduction

The corticosteroids correspond to the natural hormones secreted by adrenal cortex as well as their synthetic derivatives mainly used for their anti-inflammatory properties [1]. Extended corticosteroid therapy is defined by the use of steroids for a period of at least three months. The latter occupy a major place in the anti-inflammatory therapy and show a remarkable effectiveness in the treatment of chronic pathologies. However, they are not without risk. It is estimated that 0.2 to 0.5% of the general population receives prolonged systemic corticosteroids [2,3]. In the United Kingdom, about 0.85% of the adult British population and up to 2.5% of the population over 70 years of age take long-term corticosteroid therapy [2]. In the United States, new oral corticosteroids are being introduced in ten million people every year [4,5]. In France, more than two million prescriptions a year prescribe glucocorticoids and several hundred thousand people are treated each year with corticosteroids [5].

Osteoporosis is a generalized skeletal condition characterized by low bone mass and deterioration of microarchitecture of bone tissue, leading to bone fragility and increased susceptibility to fractures [6]. It is considered a major public health problem by the health authorities because of the potential severity of certain fractures which it increases the risk. The morbidity and cost of these fractures are well known, but recent data highlight the impact of some of these fractures on mortality [7]. This is the case, for example, of the fracture of the upper extremity of the femur, the most serious complication of osteoporosis, because of the residual permanent physical incapacity of which it may be

the cause, of the loss of independence, institutionalization, and especially an increase in mortality risk. Cortisonic osteoporosis is the most common secondary osteoporosis and the leading cause of osteoporosis in young adults [8,9]. The frequency of use of corticosteroid therapy as well as its indisputable efficacy in many diseases, the progress made in the understanding of its pathophysiology, and the current therapeutic possibilities are so many elements justifying prevention recommendations and the treatment of the bone consequences of prolonged corticosteroids. To evaluate the prevention of this condition in our patients receiving prolonged corticosteroid therapy, we conducted a cross-sectional, descriptive, study targeting specialist prescribing physicians.

Patients and Methods

Type of Study

It was a cross-sectional, multicenter, descriptive study from June 1st, 2017 to August 1st, 2017, over a period of 2 months. It involved a questionnaire given to the target practitioners.

Study Population

Criteria for Inclusion: All internal hospital practitioners, physicians in specialization, university assistants, associate lecturers, full professors and all other non-academic specialists. The target specialties were Internal Medicine, Rheumatology, Nephrology, Dermatology, Respirology, Cardiology, Hematology, Hepatogastroenterology and Neurology were included.

Criteria for Non-Inclusion and Exclusion: All questionnaires that were not completed or completed and not completed within the specified time frame were not included. All questionnaires with incomplete answers were excluded.

Data Collection: We even made the collection of these data using a sheet that we made. It consisted of two large parts. The first part concerned the identification of practitioners, his / her hospital-grade, his level of specialization if he is an intern or a doctor in specialized training, his medical specialty. The second part concerned the prescription procedure. The last part concerned the frequency of prescription of various measures to prevent osteoporosis associated with prolonged systemic corticosteroids (vitamin-calcium supplementation, prescription of bisphosphonates, realization of osteodensitometry at then treatment). The various questions asked were in the form of single choice questions. Completed and submitted questionnaires were analyzed by sphinx software.

Results

Of the 230 questionnaires distributed, 170 were completed and re-addressed, representing a response rate of 73%. The main characteristics of responder practitioners as well as the main indications for glucocorticoids are reported in (Figures 1, 2).

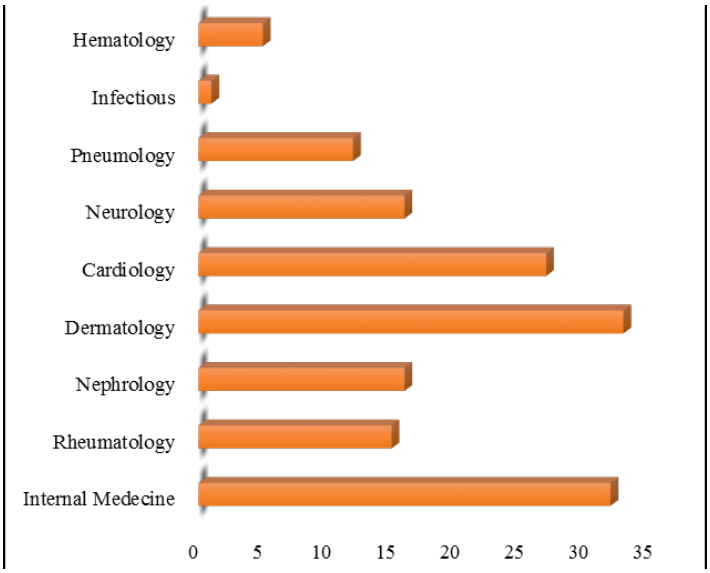

Figure 1: Specialties of the doctors.

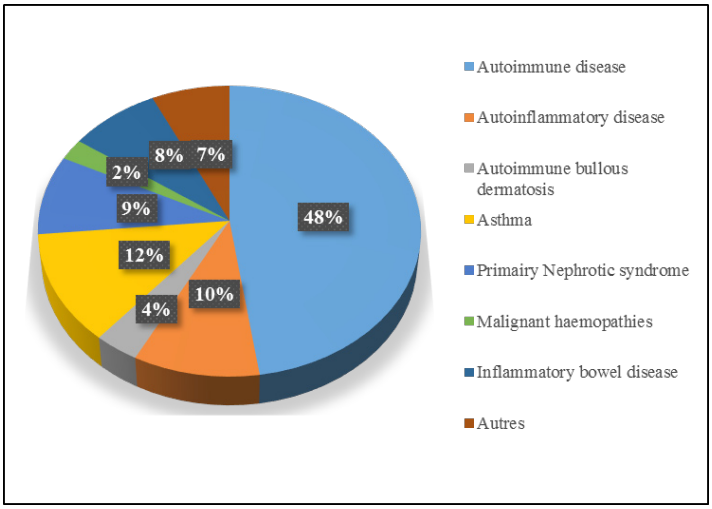

Figure 2: Main indications of prolonged oral corticosteroid therapy.

These practitioners were mainly internists and dermatologists and practiced mainly in hospitals as hospital practitioners. They used to prescribe prolonged oral corticosteroids, at an immunosuppressive dose, preferably prednisone. (Figures 3-5) describe the frequency of prescription of various preventive measures associated with corticosteroid therapy. Of these, none seemed to be unanimous among physicians. If vitamin-calcium supplementation was systematically prescribed by 38% (65 cases) of doctors, 52% of them never prescribed bisphosphonates as an adjuvant and 65% never performed bone densitometry to their patient.

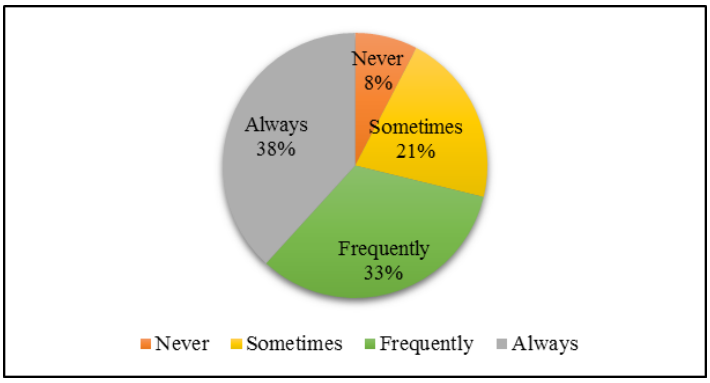

Figure 3: Frequency of prescription of vitamin-calcium supplementation.

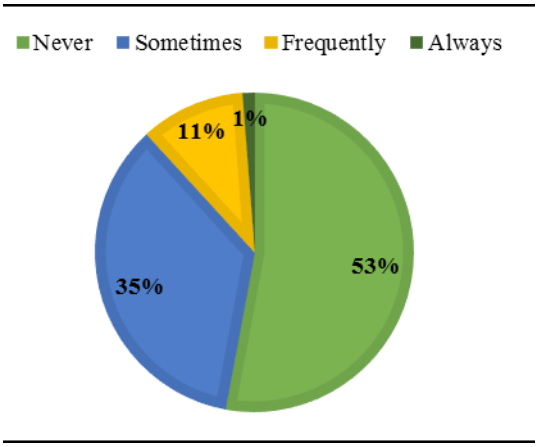

Figure 4: Frequency of prescription of bisphosphonates.

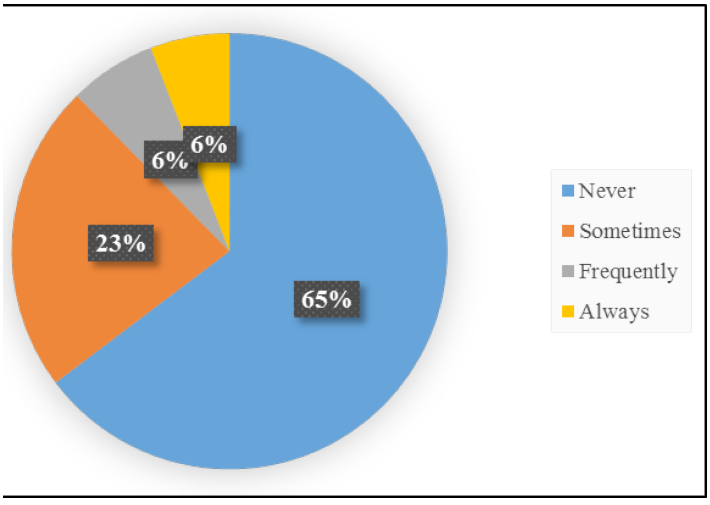

Figure 5: Frequency of performing bone densitometry in follow-up patients.

Discussion

This study, conducted with the help of 170 Senegalese medical specialists, shows that the prevention of osteoporosis by prescribers of long-term corticosteroid therapy is very poor and very heterogeneous. Thus, although vitamin-calcium supplementation was routinely prescribed by some rare physicians, most of them never performed an initial assessment of the risk of fracture, and never prescribed general preventive measures or anti-osteoporotic treatment (bisphosphonate, téraparatide). This is all the more alarming because most of the indications for long-term corticosteroid therapy are autoimmune diseases where it is prescribed at anti-inflammatory and immunosuppressive doses as shown in our study. However, Glucocorticoids (GC) have direct negative bone effects (repressing action on osteoblast and osteocyte and proliferating on osteoclast) and indirect (reduction of intestinal absorption of calcium, increased calciuria and cortisone myopathy) a factor favoring a fall). The rapidity of this effect, as illustrated by the increased risk of fracture, is partly related to the previous adverse effect of the underlying inflammation and the presumed role of autoimmunity [10-13]. In fact, the inflammation acts at two levels, in osteolysis and in osteoformation.

On the one hand, the role of the T lymphocyte is preponderant in osteoclastogenesis. The Th17 pathway is the most important one and under the influence of interleukin 6 (IL-6) and IL-23, the pro-inflammatory cytokines (TNF-a and IL-1) effect by the action of the RANKL-RANK system to promote the transformation of osteoclast precursors into osteoclasts and to promote the activity of these [13]. On the other hand, the effect of inflammation on osteoformation is related to the action of TNF-α on osteoblastic function-regulating proteins such as DKK1 and sclerostin, secreted by osteocytes [14]. As for autoimmunity, its role in bone remodeling is suggested by the demonstration of citrullinated proteins in osteoclasts, and the increase of osteoclastogenesis in the presence of anti-CCP antibodies *in vitro* and *in vivo* [15]. Thus, when corticosteroids are introduced into an inflammatory disease, bone remodeling is already abnormal, with an imbalance due to excessive bone resorption and a lack of training.

GC-induced bone loss is seen within the first 6 months of treatment. The extent of bone loss depends on the dose and duration of cortisone treatment. It is variable within a population and no densitometric, biological or clinical criterion can predict for a given individual the occurrence and extent of bone loss [16-19]. The risk of fracture increases by 30 to 50% in subjects receiving long-term corticosteroid therapy. This fracture risk is dose and duration dependent mainly occurring above 7.5 mg of prednisone equivalent per day and from 6 months of treatment and their frequency decreases from the 3rd month after stopping treatment. They are mostly of interest to the cervix and hip. The analgesic effect of corticosteroid therapy may explain the greater frequency of vertebral fractures that have gone unnoticed [20]. The main risk factors for fracture in these subjects receiving one

are age, risk factors for falls, treatments and associated pathologies and the state of the underlying disease regardless of sex, as well as occurrence of menopause and duration of hormonal deficiency in women

No fracture risk assessment was performed by the physicians interviewed. While this initial assessment is recommended in all patients starting oral corticosteroids for a duration of more than 3 months and in patients already receiving oral corticosteroids ≥ 7.5 mg / d for more than 3 months (if this assessment did not take place at the beginning of treatment) (Grade A). It is recommended regardless of the dose of GC (Grade A). It includes the calculation of FRAX, the search for personal history of fracture by X-ray two-photon absorptiometry (Grade B) and the measurement of bone mineral density (Grade C). To this, we add general measures during the follow-up, namely the evaluation at each consultation of the dose of glucocorticoids used, the means of cortisone sparing, the control of other risk factors for osteoporosis and the prevention of falls [21,22].

The treatment with anti-osteoporosis, depends or not on the existence of indications. In postmenopausal women and men after the age of 50, it is recommended that the following situations (Grade B) be considered as high risk of a fracture warranting treatment:

- Personal history of fracture due to bone fragility after 50 years;
- T low score at one of the 2 lumbar or femoral sites: ≤ -2.5;
- Age ≥ 70 years: the risk of fracture of a 70-year-old woman starting corticosteroid therapy is equivalent to that of a 70-year-old woman who has already had a fracture (according to the FRAX® score);
- Long-term high-dose corticosteroids (≥ 7.5 mg / day prednisone equivalent for more than 3 months) [18].

In other cases, it is recommended to take into account the value of FRAX® adjusted to the dose of GC and if need to seek the advice of a specialist in bone diseases. In the absence of an indication for anti-osteoporotic treatment (bisphosphonates or teriparatide), it is recommended to apply the general measures, and to carry out a new densitometric control a year later, the time being able to be adapted according to initial densitometric value and dose of corticosteroids (grade C). In case of significant bone loss (variation of bone mineral density ≥ 0.03 g / cm2), an anti-osteoporotic treatment can be started (grade C) [23-27]. The administration of calcium and vitamin D on pathophysiological bases is largely performed during the prescription of GC, but its benefit is not established. Indeed, the observation of placebo groups of major therapeutic trials, in which patients receive physiological doses of calcium and vitamin D, shows that bone loss and fractures are not prevented in these groups. The calcium intake recommended by the National Health and Nutrition Program (PNSS) is 800-1200 grams. To cover these needs, it is necessary to consume 4 dairy products per day (yogurts, fresh cheeses, fermented milks, cheeses, milk). In addition, the association of increased incidence of cardiovascular events with calcium supplementation in older women has been suggested. It has been reported mainly in subjects whose spontaneous dietary calcium intake was already sufficient, but it is not confirmed [28-30]. Offsetting dietary calcium deficiencies reduces the added risk factors for bone loss. Inputs should be evaluated by a food survey. Systematic prescription of calcium supplementation is not recommended (Grade A).

As for the prescription of vitamin D, it is not recommended systematically. In the event of a situation of potential bone fragility linked to the underlying inflammatory disease and the use of glucocorticoids, the determination of serum vitamin D (25 OH vitamin D) is indicated (Grade A). In case of vitamin D insufficiency or deficiency, it is recommended to prescribe an "attack" treatment which will make it possible to obtain a 25- (OH) -vitamin D level above the target value of 30 ng / mL (Grade A). The maintenance treatment dosage is 800 to 1200 IU / day (or equivalent doses of 100,000 IU every 2 to 3 months) [31]. Our work has two main limitations. First, concerning practitioners, a non-response bias must be mentioned. It is possible that doctors who did not answer our questionnaire have very different medical practices from those who did. Nevertheless, given the fact that the number of non-response is very low, we think that these results reflect the general practice of Senegalese doctors. Despite these limitations, our study has shown that the prevention of cortisone osteoporosis in Senegal is very heterogeneous and not very consistent with recommendations. Due to the number of patients with systemic disease who also have risk factors for osteoporosis and are receiving extended corticosteroid therapy, homogenization of practices consistent with the recommendations would significantly reduce the morbidity and mortality associated with osteoporosis. glucocorticoid-induced.

References

1. Le Jeune C (2012) Pharmacologie des glucocorticoïdes. Presse Med 41: 370-377.
2. Van Staa TP, Leufkens HG, Abenhaim L, Begaud B, Zhang B, et al. (2000) Use of oral corticosteroids in the United Kingdom. QJM 93: 105-111.
3. Walsh LJ, Wong CA, Pringle M, Tattersfield AE (1996) Use of oral corticosteroids in the community and the prevention of secondary osteoporosis: a cross sectional study. BMJ 313: 344-346.
4. Fardet L, Kassar A, Cabane J, Flahault A (2007) Corticosteroid-induced adverse events in adults: frequency, screening and prevention. Drug Safety 30: 861-881.
5. Fardet L, Petersen I, Nazareth I (2011) Description des prescriptions de corticothérapies orales en population générale. La Revue de médecine interne 32 : 594-599
6. World Health Organizing (1994) Assessment of fracture risk and its application to screening for postmenopausal osteoporosls. WHO technical report series, n˚843. Geneva: Switzerland World Health Organisation 1994.
7. DRESS (2010) Les fractures du col du fémur en France entre 1998 et 2007 : quel impact du vieillissement ? Collection Etudes et Statistiques 2010.
8. Kok C and Sambrook PN (2009) Secondary osteoporosis in patients with an osteoporotic fracture. Best Pract Res Clin Rheumatol 23: 769-779.

9. Briot K and Roux C (2013) Ostéoporose cortico-induite. La Revue de médecine interne 34 : 315-323.

10. Van Staa TP, Geusens P, Bijlsma JW, Leufkens HG, Cooper C (2006) Clinical assessment of the long-term risk of fracture in patients with rheumatoid arthritis. Arthritis Rheum 54: 3104-3112.

11. Maillefert JF, Aho LS, El Maghraoui A, Dougados M, Roux C (2001) Changes in bone density in patients with ankylosing spondylitis: a two-year follow-up study. Osteoporos Int 12: 605-609.

12. Schett G, Kiechl S, Weger S, Pederiva A, Mayr A, et al. (2006) Highsensitivity C-reactive protein and risk of non traumatic fractures in the Bruneck study. Arch Intern Med 166: 2495-501.

13. Roux C (2011) Osteoporosis in inflammatory joint diseases. Osteoporos Int 22: 421-433.

14. Uderhardt S, Diarra D, Katzenbeisser J, David JP, Zwerina J, et al. (2010) Blockade of Dickkopf (DKK)-1 induces fusion of sacroiliac joints. Ann Rheum Dis 69: 592-597.

15. Harre U, Georgess D, Bang H, Bozec A, Axmann R, et al. (2012) Induction of osteoclastogenesis and bone loss by human autoantibodies against citrullinated vimentin. J Clin Invest 122: 1791-1802.

16. Briot K, Cortet B, Roux C, Fardet L (2014) Actualisation 2014 des recommandations sur la prévention et le traitement de l'ostéoporose cortico-induite. Revue du rhumatisme 81: 385-394.

17. Walsh LJ, Wong CA, Pringle M (1996) Use of oral corticosteroids in the communityand the prevention of secondary osteoporosis: a cross sectional study. BMJ 313: 344-346.

18. Ramsey-goldmann R (2002) Missed opportunities in physician management ofglucocorticoid-induced osteoporosis? Arthritis Rheum 46: 3115-3120.

19. Kanis JA, Johansson H, Oden A, Johnell O, de Laet C, et al. (2004) A meta-analysis of prior corticosteroid useand fracture risk. J Bone Miner Res 19: 893-899.

20. Van Staa TP, Geusens P, Pols HA, de Laet C, Leufkens HG, et al. (2005) A simple score for estimating the long-term risk of fracture in patients using oral glucocorticoids. QJM 98: 191-198.

21. El-Khoury F, Cassou B, Charles MA (2013) The effect of fall prevention exer-cise programmes on fall induced injuries in community dwelling older adults:systematic review and meta-analysis of randomised controlled trials. BMJ 347: f 6234.

22. Bolland MJ, Barber PA, Doughty RN (2008) Vascular events in healthy olderwomen receiving calcium supplementation: randomised controlled trial. BMJ 336: 262-266.

23. Benhamou CL, Souberbielle JC, Cortet B, et al. (2001) Pour le Groupe de Rechercheet d'information sur les Ostéoporoses (GRIO). La vitamine D chez l'adulte : recommandations du GRIO. Presse Med 40: 673-682.

24. Sambrook PN, Roux C, Devogelaer JP, Saag K, Lau CS, et al. (2012) Bisphosphonates and glucocorti-coid osteoporosis in men: results of a randomized controlled trial comparingzoledronic acid with risedronate. Bone 50: 289-295.

25. Saag KG, Shane E, Boonen S, et al. (2007) Teriparatide or alendronate in glucocorticoid-induced osteoporosis. N Engl J Med 357: 2028-2039.

26. Saag KG, Zanchetta JR, Devogelaer JP (2009) Effects of teriparatide versus alendro-nate for treating glucocorticoid-induced osteoporosis: thirty-six-month resultsof a randomized, double-blind, controlled trial. Arthritis Rheum 60: 3346-55.

27. Kanis JA, Stevenson M, McCloskey EV, Davis S, Lloyd-Jones M (2007) Glucocorticoid-induced osteo-porosis: a systematic review and cost-utility analysis. Health Technol Assess 11: 1-231.

28. Gambertoglio JG, Romac DR, Yong CL, Birbaum J (1987) Lake of effect of sucralfate on prednisone biovailability. Am. J. Gastroenterol 82: 42-44.

29. Richard D, Senon JL, Roblot P (1997) Corticoïdes et corticothérapie. Hermann, Paris 1997.

30. Dejean C and Richard D (2013) Mécanismes d'action des glucocorticoïdes. La Revue de médecine interne 34 : 264-268

31. Brion N, Guillevin L, LE Parc J-M (1998) La corticothérapie en pratique, Masson Ed, Paris 376.