Alendronic acid 70 mg, calcium and vitamin D supplements. Patient had severe vitamin D deficiency and was commenced on treatment at primary care. She is on analgesics and physiotherapy for back pain, which was of moderate severity. She was on oral prednisolone for 5 years for polymyositis rheumatica in the past and weaned off year ago. Other past medical history includes coronary artery disease; a myocardial infarction requiring coronary artery bypass graft 3 years back. She does not have diabetes or hypertension. She has hypercholesterolaemia; probably polygenic in origin and is on lipid lowering medications since the age of 50 years.

Her alcohol intake is minimal, and is a non-smoker. There is no family history of osteoporosis or cardiovascular disease. Her milk consumption accounted for 300 mg of calcium per day. She has reached menopause at 43 years of age and has not been on hormonal replacement therapy. Her current medications are Aspirin, Atorvastatin, Bisoprolol and Omeprazole. There is no history of indigestion or dental concerns.

Except mild local tenderness at L4–5 region, rest of the clinical examination is unremarkable.

Recent blood investigations showed normal calcium, inorganic phosphate, PTH and vitamin D. Alkaline phosphatase (195 IU/L) and P1NP (55 ug/L) were raised. Renal functions showed normal creatinine with eGFR of 62 mL/min. Full blood count is unremarkable. Serum and urine electrophoresis excluded multiple myeloma.

Summary: This is a case of severe osteoporosis involving vertebral fractures requiring osteoporosis treatment. Possible causes for osteoporosis would be previous use of oral steroids for long time and severe undiagnosed vitamin D deficiency. Hypercholesterolaemia and significant history of ischaemic heart disease are other concomitant diagnoses.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.7693

SATURDAY, 16 JUNE 2018

Emerging topics in the management of the antiphospholipid syndrome

SP0164 HOW TO MANAGE ASYMPTOMATIC CARRIERS OF ANTIPHOSPHOLIPID ANTIBODIES

L. Andreoli^{1,2}. ¹Department of Clinical and Experimental Sciences, University of Brescia; ²Unit of Rheumatology and Clinical Immunology, Spedali Civili Hospital, Brescia, Italy

Individuals who do not display the classical features of the Antiphospholipid Syndrome (APS) (vascular and obstetric disease)¹ are referred to as “aPL carriers”. They can be patients affected by systemic autoimmune diseases who are screened for antiphospholipid antibodies (aPL). aPL may be found in patients with “non-criteria” manifestations or in women undergoing investigations for infertility. The presence of aPL can be serendipitously discovered before a surgical procedure because of a prolonged thromboplastin time. Are these subjects at increased risk for thrombosis and adverse pregnancy outcomes (APO)? Since aPL are pathogenic autoantibodies, the answer should be “yes”. However, the magnitude of the risk can be variable from patient to patient, accordingly to the multifactorial origin of aPL-related vascular and obstetric manifestations.

According to international consensus,² the thrombosis risk stratification should consider: 1) the aPL profile (type, titer, persistence), 2) the coexistence of other thrombotic risk factors, and 3) the presence of an underlying autoimmune disease, mainly systemic lupus erythematosus (SLE). The definition of “high-risk” aPL profile comprises positivity for Lupus Anticoagulant (LA), or ‘triple positivity’, i.e. LA+anti cardiolipin antibodies (aCL) +anti-beta2glycoprotein I antibodies (anti-B2GPI) or medium-high titers of IgG aCL or IgG anti-B2GPI. Conversely, patients with isolated, intermittently positive aCL or anti-B2GPI at low-medium titers could be considered at low risk for thrombosis.

According to the literature, aPL carriers seem to have a low annual incidence of acute thrombosis, ranging from 0% to 3.8%³. These figures are not much different from the estimated incidence of thrombosis in unselected cases (about 1% patient-years), which is also equivalent to that of major bleeding associated with the use of low dose aspirin (LDA), the most frequently used drug for primary prophylaxis.⁴ Therefore, the dilemma in clinical practice is to correctly select those aPL carriers for whom the expected benefit of therapy outweigh the risk.

Over years, the management of aPL carriers have been investigated in several studies enrolling different patients groups (SLE, pure obstetric APS, asymptomatic aPL carriers) and evaluating the efficacy of various interventions: LDA,⁵ low intensity warfarin,⁷ low molecular weight heparin (LMWH) in high risk situations such as surgery, prolonged immobilisation, and puerperium.⁹

Aside from drugs acting on platelets and on the coagulation system, there is evidence that immunomodulatory agents may be beneficial in primary prophylaxis of aPL carriers. Hydroxychloroquine (HCQ) is a well-recognised key-drug in the management of SLE patients and has an anti-thrombotic effect.¹⁰ The use of HCQ as primary prophylaxis has been proposed also for non-SLE patients.¹¹ Statins may be useful in aPL carriers not only for the correction of a proatherogenic lipid profile, but also for reducing proinflammatory and prothrombotic biomarkers.^{12,13}

Turning to the obstetric field, the detection of aPL antibodies has been increasingly performed in asymptomatic women, mainly for obstetrical reasons (e.g. before assisted reproductive techniques, after APO that are not included in APS classification criteria). Therefore, it is not infrequent to take the responsibility to recommend or not a treatment in “healthy” pregnant women carrying aPL. General obstetric risk should be assessed (age, hypertension, obesity, etc.). It is currently under discussion whether different aPL profiles confer the same degree of obstetric risk.¹⁴ LA¹⁵ and triple aPL positivity¹⁶ seem to be the major predictors of APO, although APO have been described also in patients with a “low-risk” aPL profile (e.g., IgM isotype or medium to low aPL titers).^{17,18} A key drug for primary obstetric prophylaxis is LDA and many physicians prescribe it to pregnant aPL carriers.²⁰ The immunomodulatory properties of HCQ have been advocated to be beneficial in pregnant patients with aPL²¹ and clinical retrospective studies supported its effectiveness in refractory obstetric APS.^{22,23} Puerperium is considered a high-risk period for thrombosis for all women. Women who carry aPL should be considered for LMWH for 4–6 weeks after delivery.¹⁹

REFERENCES:

- [1] Miyakis S. 2006.
- [2] Ruiz Irastorza G. 2011.
- [3] Barbhaiya M. 2011.
- [4] Ceccarelli F. 2012.
- [5] Erkan D. 2007.
- [6] Arnaud L. 2015.
- [7] Cuadrado MJ. 2014.
- [8] Giron Gonzalez JA. 2004.
- [9] Ruffatti A. 2009.
- [10] Ruiz Irastorza G. 2010.
- [11] Erkan D. 2017.
- [12] Lopez-Pedraza C. 2011.
- [13] Erkan D. 2014.
- [14] de Jesus G. 2014.
- [15] Lockshin MD. 2012.
- [16] Ruffatti A. 2011.
- [17] Mekinian A. 2012.
- [18] Gardiner C. 2013.
- [19] Andreoli L. 2017.
- [20] Erkan D. 2008.
- [21] Meroni PL. 2016.
- [22] Mekinian A. 2017.
- [23] Sciascia S. 2016.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.7811

SP0165 THE COMPLEX INTERPLAY BETWEEN SYSTEMIC LUPUS ERYTHEMATOSUS AND ANTIPHOSPHOLIPID SYNDROME

E. Svenungsson. Medicine Solna, Unit of Rheumatology, Karolinska Institutet, Stockholm, Sweden

The antiphospholipid syndrome (APS) was first described in the 1980's. It is diagnosed when antiphospholipid antibodies (aPL) i.e. anti-cardiolipin (aCL), anti-β₂Glycoprotein-I (aβ₂GPI) or positivity in the functional lupus anticoagulant test (LA) occur together with any type of thrombosis (e.g. myocardial infarction(MI), stroke, venous or microvascular thromboses) or obstetric complications.⁵ aPL recognise protein co-factors, most importantly the scavenger protein β₂GlycoproteinI (β₂GPI), that bind to membrane phospholipids. It is not fully understood how complexes of β₂GPI and anti-β₂GPI antibodies initiate a pro-thrombotic state, but activation of platelets, endothelial cells and the complement cascade are associated features.

Approximately 80% of APS patients are women, many are young and severely ill. There is a considerable overlap between APS and SLE. Approximately 30%–40% of SLE patients are aPL positive but only about half of them develop clinical symptoms fulfilling the APS classification criteria.