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VIEWPOINT

2023 ACR/EULAR classification criteria for antiphospholipid syndrome: more lights rise but shade remains

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ABSTRACT

Antiphospholipid syndrome (APS) is an autoimmune disorder for which there are no universally accepted diagnostic criteria, although classification criteria do exist, as is the case with most autoimmune diseases. Until 2023, the 2006 Sydney classification criteria were in use. Although originally intended for research purposes, these criteria have often been employed in clinical practice as a substitute for diagnostic guidelines, thereby conflating classification with diagnosis. In July 2023, ACR and European Alliance of Associations for Rheumatology convened a panel of experts to revise these criteria. The newly published classification criteria are explicitly intended for research use only. They place a strong emphasis on specificity—99%—but this comes at the expense of sensitivity—84%. The updated criteria encompass six clinical domains and two laboratory domains. Notably, the inclusion of new clinical features, such as thrombocytopenia, cardiac valve involvement and microvascular thrombosis, has broadened patient inclusion and, indirectly, aided the diagnostic process. However, a significant proportion of patients with suspected antiphospholipid antibody-related conditions may no longer meet the criteria for APS classification. In real-world settings, this could result in these individuals being denied appropriate management, thereby increasing their risk of subsequent thrombotic or obstetric events, as has already been demonstrated.

This manuscript examines the advantages and limitations of the new clinical and laboratory domains, considering their implications not only from a research but also from a clinical perspective, APS.

INTRODUCTION

Antiphospholipid syndrome (APS) is an autoimmune disease characterised by arterial, venous or microvascular thrombosis, obstetric morbidity and other non-thrombotic manifestations in the persistent presence of antiphospholipid antibodies (aPL). These autoantibodies bind to phospholipid–protein complexes such as β2-glycoprotein I, triggering cellular activation, inflammation and thrombosis.[1] As with most autoimmune diseases, APS lacks formal diagnostic criteria. Instead, only classification criteria exist, intended to standardise patient inclusion in studies and clinical trials, although they are often used in clinical practice for diagnosis and management. Until 2023, the 2006 Sydney criteria[2] were employed for APS classification, requiring a thrombotic and/or obstetric event and at least one repeatedly positive aPL. In July 2023, the American College of Rheumatology (ACR) and the European Alliance of Associations for Rheumatology (EULAR) published new criteria, explicitly for research use, prioritising specificity (99%) over sensitivity (84%).[3] Consequently, patients with aPL-related manifestations may be excluded from classification, potentially affecting their management and increasing the risk of recurrent clinical events. The new criteria include six clinical and two laboratory domains (figure 1, table 1),[3 4] assigning points to each feature; a minimum of three points in each clinical (4) and laboratory (2) domains is required to classify as APS. This manuscript critically analyses the strengths and limitations of these new criteria and their research and clinical implications.

SHOULD WE AIM TO CLASSIFY OR TO DIAGNOSE PATIENTS?

Classification criteria are intended to define homogeneous cohorts for research, not to identify all possible cases. Therefore, strictly speaking, they should not be used as diagnostic tools or for clinical decision-making. However, in practice, many experts manage patients with clinical manifestations and recurrent, positive aPL as APS, even if they do not fully meet classification criteria.[5–9] The exclusive reliance on classification criteria creates two problems: (a) some patients may be misdiagnosed or improperly treated when these criteria are used as a diagnostic guide in less experienced hands or in centres

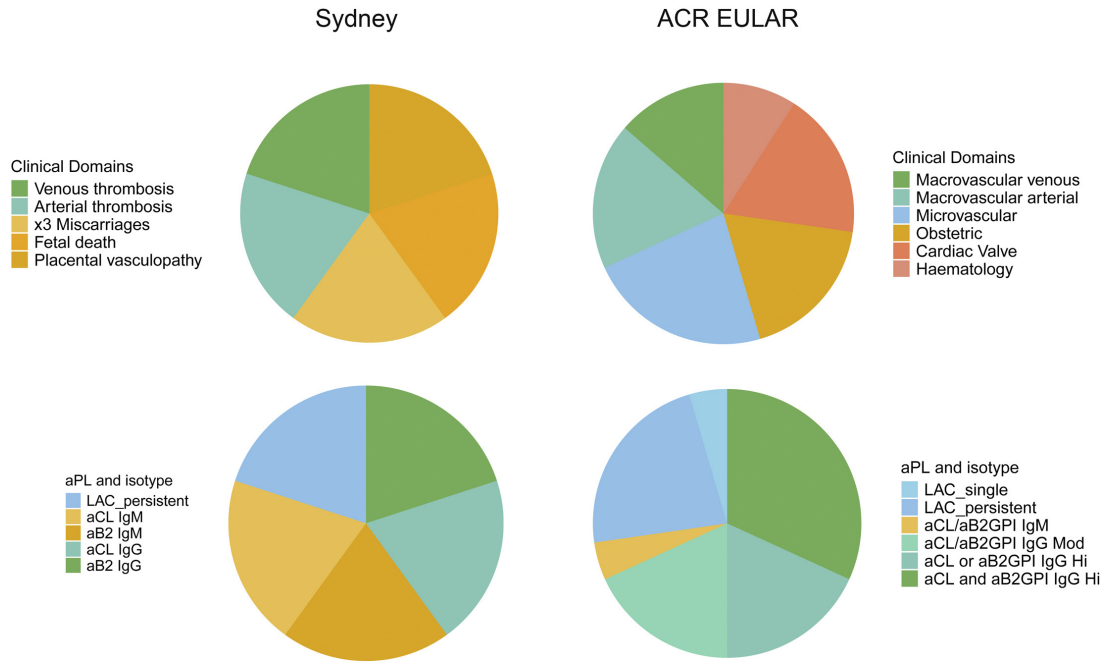

Figure 1 Clinical and laboratory criteria weights for antiphospholipid syndrome classification according to Sydney and 2023 ACR-EULAR criteria. ACR, American College of Rheumatology; aPL, antiphospholipid antibodies; EULAR, European Alliance of Associations for Rheumatology; LAC, lupus anticoagulant.

unfamiliar with the nuances of APS and (b) excluding certain patients from studies may introduce bias into future research. Thus, to avoid such errors, a diagnostic framework should ideally precede the creation of stricter classification criteria.

ACR/EULAR 2023 CRITERIA

Advantages

Regarding the thrombotic domains, first, objectivity is improved through the requirement of confirmation of venous and arterial thromboses by imaging or histopathology. Second, greater weight is given to unprovoked thrombosis, enhancing classification specificity. Third, microvascular APS is now formally recognised, aligning the criteria more closely with clinical reality. Additionally, compared with the Sydney criteria, improvements include the recognition of cardiac valve involvement, inclusion of livedo racemosa and the inclusion of thrombocytopenia that had been discussed in the previous Sapporo and Sydney criteria.[1 2] This may result in improved patient inclusion reflecting broader clinical manifestations.

Disadvantages related to clinical domains

Despite good intentions, the criteria have notable shortcomings. For example, the diagnostic weight of thrombotic events is reduced if other risk factors are present, even in patients with persistent triple aPL positivity. This may affect decisions about anticoagulation duration and intensity, with negative consequences: for instance, patients with low-risk congenital thrombophilia, thrombosis following brief hospitalisation, short bed rest or women taking oral contraceptives long-term may score low and be excluded from classification (table 1). Moreover, cases of transient ischaemic attack (TIA) without imaging confirmation are excluded, despite a high subsequent stroke risk.[10] Livedo racemosa, though well defined, can be confused with livedo reticularis, which lacks clinical relevance, potentially leading to false positives.[11 12]

Platelet count: friend or foe?

The 2023 classification criteria include thrombocytopenia as a clinical criterion, although it is insufficient alone for APS classification (table 1). While thrombocytopenia seems like it should be generally protective against thrombosis, paradoxically, in APS, it is associated with increased thrombotic risk.[13–15] Its inclusion is, therefore, beneficial. However, it is only counted if it is not severe, excluding counts <20 000×10⁹/L, which may result in false negatives in rare cases. Conversely, gestational thrombocytopenia could cause false positives, as 10% of healthy pregnancies show platelet counts ≤150 000×10⁹/L, and up to 5% fall between 70 000 and 100 000×10⁹/L.[16–19] Adjusting the threshold to 80 000×10⁹/L should be considered, as these limitations affect both classification and clinical diagnosis.

The case of obstetric morbidity: perfect is the enemy of good

Domain 4, defining obstetric morbidity, requires scrutiny regarding terminology and scoring. Terminology related to gestational losses is inconsistent and lacks consensus. New terms such as early foetal, prefoetal and foetal loss do not align with those used in specialised literature. According to the American College of Obstetrics and Gynaecology (2018, updated 2025),[20 21] the European Society of Human Reproduction and Embryology[22] and others,[23 24] early pregnancy occurs before 10 weeks

Table 1 Critical appraisal and suggestions for refinement of the 2023 ACR/EULAR classification criteria for antiphospholipid syndrome

Clinical criteria	Weight	Comments	Event
D1. Macrovascular (venous thromboembolism, VTE)			
VTE with a high-risk VTE profile	1	Low-risk genetic thrombophilia should not be considered	Factor II and factor V heterozygosis
		Acquired thrombophilia:	
		Reconsider stratification of hospital admission as major risk factor	Short-term immobilisation by hospital admission
		Use of contraceptive pills as minor VTE risk factor	Long-term contraceptive pill intake
		Consider aPL thrombophilic factor per se	Persistent positivity aCL and/or aβ2GPI and/or LAC
		Consider affected territory on minor risk factors scoring	Venous thrombosis other than leg PVT
VTE without a high-risk VTE profile	3		
D2. Macrovascular (arterial thrombosis, AT)			
AT with a high-risk CVD profile	2	Consider transient occlusions	TIA without image confirmation
AT without a high-risk CVD profile	4		
D3. Microvascular			
Suspected: livedo racemosa or livedoid vasculopathy or acute/chronic aPL nephropathy or pulmonary haemorrhage	2	Consider other small vessel thrombosis	Cognitive impairment supported by images in individuals<50 years old, without CVD risk factors
Established: livedoid vasculopathy or acute/chronic aPL-nephropathy or pulmonary haemorrhage or myocardial disease or adrenal haemorrhage	5		
D4. Obstetric			
≥ 3 consecutive pre-foetal (<10 weeks) and/or early foetal (10 weeks 0 day–15 weeks 6 days) deaths	1	Rethink obstetrical morbidity as classification criteria	Three or more consecutive early foetal deaths (<10 weeks) of suspected euploid embryos or IVT euploid embryos should be classificatory criteria
Foetal death (16 weeks 0 day–33 weeks 6 days) in the absence of PEC/PI with severe features	1		One or more foetal deaths between weeks 10 and 19+6 of normal formed fetuses should be a classificatory criteria
PEC with severe features (<34 weeks 0 day) or PI with severe features (<34 weeks 0 day) with/without foetal death	3		
PEC with severe features (<34 weeks 0 day) and PI with severe features (<34 weeks 0d) with/without foetal death	4		
D5. Cardiac valve			
Thickening	2		
Vegetation	4		
D6. Haematology			
Thrombocytopenia (lowest 20–130×10^9/L)	2	Reconsider lower threshold in platelet counts	≤ 20×10^9/L platelets in otherwise healthy people other than recurrent aPL positive

Continued

Table 1 Continued

Lab criteria	Weight	Comments	Event
D7. LAC			
Single one-time positive LAC	1		
Persistent positive LAC	5		
D8. aCL/aβ2GPI			
Moderate or high positive IgM aCL and/or aβ2GPI	1	Reconsider IgM persistent positivity as a classification criterion	Persistent positivity for isolated IgM isotypes in individuals with clinical criteria
Moderate positive IgG aCL and/or aβ2GPI	4		
High positive IgG aCL OR aβ2GPI	5		
High positive IgG aCL AND aβ2GPI	7		

aCL, anti-cardiolipin; aβ2GPI, anti-β2 glycoprotein I; CVD, cardiovascular disease; IVT, in vitro transfer; LAC, lupus anticoagulant; PEC, preeclampsia; PI, placental insufficiency; TIA, transient ischemic attack.

or within the first 12 6/7 weeks of gestation. None uses the term 'prefoetal'. Foetal loss or demise is defined as occurring from 12 6/7 weeks until 23 6/7 weeks or later (table 1). Beyond terminology, the scoring undervalues the recurrence of early and foetal losses, preventing patients from accumulating sufficient points in the obstetric domain. Additionally, there is no consideration for otherwise healthy women with persistent aPL who experience severe pre-eclampsia, HELLP syndrome or eclampsia beyond 34 weeks of gestation.[24 25] Nor is the impact of aPL on recurrent euploid implantation failure in assisted reproduction acknowledged, despite existing data.[26 27] Accordingly, these criteria warrant revision.

Disadvantages related to the laboratory: the oxymoron

Domain 8 merits detailed discussion, particularly the weight assigned to the IgM isotype of aPL (table 1). Persistently moderate to high aCL and anti-β2GPI IgM titres are assigned a score of just 1—insufficient for APS classification.[3 28] However, large cohort studies indicate that over 15% of patients present with recurrent, isolated IgM positivity, associated with both thrombotic and obstetric events[29–34] as well as thrombocytopenia—itself a significant thrombotic risk factor in APS.[13–15] Surprisingly, these criteria assign equal weight to recurrent moderate/high IgM positivity and a single episode of lupus anticoagulant positivity. It is remarkable that the source data used to justify this approach were primarily drawn from reviews/meta-analyses of patients with SLE with secondary aPL, rather than primary APS cohorts (see Barbhaiya *et al*[3]). Furthermore, IgM isotypes are included in risk-based scoring systems such as aGAPSS,[35] the aPL score,[36] and the EUREKA algorithm[37]—yet they are not deemed sufficient to classify APS patients. Concerning the recommended technologies for aPL testing, several issues emerge. While many CORE laboratories now use chemiluminescence immunoassays (CLIA), the new criteria mandate the use of ELISA. Additionally, ELISA positivity thresholds (expressed in arbitrary units) vary among laboratories and commercial kits, complicating standardisation and potentially leading to discordant results between methods (ELISA vs CLIA). In the absence of international calibrators, using the 99th percentile as a threshold is more accurate, as supported by the International Society on Thrombosis and Haemostasis.[31] Indeed, our own unpublished data show differing aPL absolute quantification among commercial ELISA kits (submitted). Thus, it is difficult to determine the viability and soundness of fixed moderate (40–79 units) and high (≥80 units) aPL levels.[3] Consequently, a single sample could test negative, moderately positive or highly positive, depending on the method—posing a major issue for both clinical and research contexts. The use of the 99th percentile would mitigate this problem.

CONCLUSION: LIGHT AROSE, BUT SHADE REMAINS

The emphasis on greater specificity in the new criteria comes at the cost of sensitivity, excluding many cases previously considered APS. Notably, the proportion of excluded patients varies by phenotype: 18%–30% of thrombotic cases, 30%–40% of mixed cases and over 70% of obstetric cases. This may result in patients being left undiagnosed and untreated, with serious clinical implications and introduces significant bias in research. Although new categories have been introduced, certain clinical situations remain excluded, such as TIAs without imaging in young patients with persistent aPL. The exclusion of severe thrombocytopenia is particularly concerning, given its high thrombotic risk in APS. The disregard of additional thrombotic risk factors, which were not excluded under the Sydney criteria, represents another setback. Moreover, the absence of cumulative scoring in the obstetric domain, and the equal weighting of recurrent early losses and single foetal deaths, does not benefit neither patients nor clinicians. The low score assigned to persistent, medium–high IgM isotypes and the lack of aPL assay standardisation also hinders accurate classification. Even in research settings, strict application of the criteria risks introducing significant

bias, the 'raison d'être' of the new criteria. Although developed with considerable effort and expertise, the new criteria require comprehensive revision to prevent unjustified exclusions and uphold clinical reason. As stated by Barbhaiya *et al*,[3] the aim was 'to develop new APS classification criteria with high specificity for use in observational studies and trials'. This aim appears not to have been fully accomplished. The term 'criteria' derives from the Greek *kriterion*, meaning 'to judge wisely'. As such, the medical and scientific communities must be equipped with tools that, improve research apart, ultimately serve to improve patient diagnostics, management and outcomes. Our final and meaningful idea, in agreement with other authors,[38 39] is that classification criteria should not unduly influence diagnosis and treatment patients suffering from APS.

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