

## **Systematic Review of the Oxford Classification of IgA Nephropathy: Reproducibility and Prognostic Value**

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Supplemental Table 1. Details of 92 papers with Oxford classification of IgA nephropathy and multivariate analysis of outcome including clinical factors and at least one individual Oxford element.

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Reference numbers are those in Supplemental Table 1. Summary of counts included.

Supplemental Table 3. Studies combining clinical factors with Oxford scores in multivariate analysis of outcome: end point: end stage renal disease

combined with 50% decline in estimated glomerular filtration rate (“combined”), or end stage renal disease with another end point. Reference numbers are those in Supplemental Table 1. Summary of counts included.

Supplemental Table 4. Studies combining clinical factors with Oxford scores in multivariate analysis of outcome: outcome: something other than end stage renal disease, alone or combined. Reference numbers are those in Supplemental Table 1. Summary of counts included.

Supplemental Table 5. References relevant to immunosuppression (n = 89), other than controlled trials (n = 3). Reference numbers are those in

Supplemental Table 1.

**Systematic Review of the Oxford Classification of IgA Nephropathy: Reproducibility, Prognostic Value, and Immunosuppressive Guidance**

**Supplemental Table I**

**Details of 92 papers with Oxford classification of IgA nephropathy and multivariate analysis of outcome including clinical factors and at least one individual Oxford element**

Reference number in Supplemental Table 1 (listed below), first author, year	Number of patients	Number of observers [minimum number of glomeruli]	Age of patients	Exclusions apart from HSP, SLE, HIV, DM, liver disease, etc.	Outcome measure: number reaching outcome	Length of follow-up [minimum* unless ESRD in that time]	Immuno-suppression before / after biopsy	Factors in multivariate analysis	Factors related to outcome	Notes

1 Cattran 2009	265	3, 4, or 5 [8]	4y-73y, median 32y	eGFR <30; p'u <0.5	1: 50% decline in eGFR or ESRD (34): 22% 58/265; 2: rate eGFR decline	69m median, minim- um 12m	14% / 29%	A: initial eGFR, p'u, MAP; MST: B: initial eGFR, fup p'u, fup MAP; MST [ <b>not I'supp; no clin stats</b> ]	1: A: M1 p=0.01, T p<0.001: 1: B: M1 p=0.03, T p<0.001; 2: A: S1 p=0.005, T p<0.001: 2: B: S1 p=0.03, T p<0.001	E, crescents, arterial lesions not associated with outcome
2 Alamartine 2011	183	2 [8]	mean 43y	none	doubled scr or ESRD	68m median,	? / 31%	eGFR, p'u, BP; MEST [ <b>not I'supp</b> ]	<b>eGFR p&lt;0.01</b>	crescents not analysed

					(30): 20%	77m				
					36/183	mean				
3 El Karoui 2011	128 adults (only 121 followed); same cases as 7 El Karoui 2012	2 [8]	18-78y, mean 38.7y	?	1: doubled scr or dialysis (36): 34% 41/121; 2: rate eGFR decline	44m mean	1 patient / none	eGFR, p'u, MAP; MEST [none l'supp]	1: <b>eGFR</b> <b>p&lt;0.0001</b> ; M1 p=0.027: 2: <b>eGFR</b> <b>p=0.012</b> , T p<0.001	crescents not analysed
4 Katafuchi 2011	702	not said [10]	8y-82y, median 30y	?	ESRD 12% 84/702	62m median, minim- um 12m*	? / 32%	eGFR, p'u, MAP, <b>steroids</b> ; MEST +/- C: also done on	<b>eGFR</b> <b>p&lt;0.01</b> , p'u <b>p&lt;0.01</b> , no <b>steroids</b>	crescents present or absent: in 416 meeting

								416 meeting Oxford criteria	<b>p&lt;0.01;</b> without C: S1 p=0.01, T1 p<0.01, T2 p<0.01; with C: T1 p<0.01, T2 p<0.01, C p=0.04	Oxford criteria, only T significant
5 Shi 2011	410 (only 294 followed)	3 [8]	mean 31y	eGFR <30; p'u <0.5	ESRD 7% 30/410	38m median, minim- um 12m	? / 41%	eGFR, p'u, MAP, l'supp; MESTC	S1 p=0.03, T p<0.001 [no clin stats]	crescents present or absent
6 Yau 2011	54	2 [not said]	>15y, mean 41y	ESRD at biopsy	50% decline in eGFR or	5.8y mean	? / 19 patients 35%	age, sex, race, scr or eGFR,	T p=0.01 [no clin stats]	crescents not analysed

					ESRD (7): 19% 10/54			p'u, BP; MEST  [not l'supp]		
7 El Karoui 2012	128 adults (only 121 followed); same cases as 3 El Karoui 2011	2 [8]	18-78y, mean 38.7y	?	1: doubled scr or dialysis (35) 40/121 33%; 2: rate decline eGFR	44m mean	1 patient / none	eGFR, p'u, BP; laboratory evidence TMA, morphologic TMA; MEST [none l'supp]	1: <b>eGFR</b> <b>p&lt;0.0001</b> , lab evid TMA p=0.03; 2: <b>eGFR</b> <b>p=0.01</b> , lab evid TMA p=0.001, S1 p=0.04	crescents not analysed

8 Gutierrez 2012	141 with slight or no p'u	not said [not said]	5-71y, mean 23.7y	eGFR <61; p'u at least 0.5	1: scr increase >50% 5/141 4%; 2: clinical remiss- ion 38% 53/141	108m median	? / none	1: time averaged p'u, S; [none I'supp] 2: initial p'u, smoking, M [none I'supp]	1: <b>not p'u</b> S1 p=0.04; 2: <b>p'u</b> <b>p&lt;0.001</b> , M0 p=0.02	crescents not analysed
9 Halling 2012	99 children (only 90 with Oxford scores)	1 [5]	children: mean 12y at first invest- igation	none	ESRD (15) or mGFR decline >50% 18/99 18%	13y mean, minim- um 5y*	2 patients / 11 patients 11%	A 74 pts: initial p'u; MESTC, global scl, +/- <b>I'supp</b> ; B 44pts: p'u at 1y; MESTC,	A: none; B: M1 p=0.012, E1 p=0.002, T p=0.007, C p=0.002,	crescents present or absent; global sclerosis present or absent; <b>no</b>

								global scl, +/- <b>I'supp</b>	global scl p=0.014 <b>[no clin stats]</b>	<b>difference if I'supp in multivar- iate</b>
<b>10</b> Kataoka 2012	43	2 [5]	15-65y, mean 40.0y	under 10y fup; eGFR <50	1.5-fold increase in scr 16/43 37%	all 10y	none / 22 patients 51%	age, sex, eGFR; MESTC, global scl score, artery score, maximum glomerular area [ <b>not I'supp</b> ]	<b>eGFR</b> <b>p=0.01,</b> M1 p=0.007, global scl p=0.04, max glom area p=0.0009	crescents present or absent
<b>11</b> Le 2012	218 children	2 [10]	2-17.9y, median 14y	eGFR <30; p'u <0.5	50% decline in eGFR or	median 56m, minim-	? / 56%	eGFR, p'u, MAP; MEST <b>[not I'supp]</b>	T p=0.04 <b>[no clin stats]</b>	crescents present or absent

					ESRD 24/218 11%	um 12m*				
<b>12</b> Lee 2012	69 adults	1 [not said]	over 18y, median 34y	eGFR decline >50% in 6m	50% decline in eGFR or ESRD (10) 16/69 23%	median 85m, minimum 3y	none / 13 patients 19%	eGFR, p'u, MAP; ET [not l'supp]	E1 p=0.004, T p=0.044 [no clin stats]	crescents not done
<b>13</b> Moriyama 2012	42 with nephrotic syndrome (only 35 with Oxford scores)	not said [8]	mean 34.2y	p'u <3.5; minimal change nephropathy	ESRD 24/42 57%	?	? / 27 patients 64%	sex, age, eGFR, p'u, BP, h't, steroids; MEST	male p=0.0241, no stds p=0.0024, eGFR p<0.0001,	crescents not done

									T p=0.0138	
<b>14</b> Shima 2012	161 children	1 [8]	3-19.4y, median 11.7y	none	eGFR <60 7/161 4%	54m median, minim- um 12m	? / 16%	A: p'u; MT; B: p'u; M, modified E, modified T, modified C = no more than 30%, >30% <b>[not l'supp]</b>	A: <b>p'u</b> <b>p=0.007</b> , M1 p=0.03, T p=0.01; B: <b>p'u</b> <b>p=0.004</b> , C >30% p=0.02	crescents present or absent, not associated with outcome
<b>15</b> Zeng 2012	1026 adults	2 [10]	18-73y, mean 34y	eGFR <30; p'u <0.5	1: 50% decline in eGFR or ESRD (90) 15%	median 53m, minim- um 12m*	1.5% / 31%	A: initial eGFR, p'u, BP; MST; B: initial eGFR, fup p'u, fup BP; MST	1: A: M1 p=0.01, T p<0.001; B: T p<0.001;	crescents present or absent, not associated

					159/ 1026; 2: rate eGFR decline			<b>[not I'supp, no clin stats]</b>	2: A: M1 p=0.005, T p<0.001; B: M1 p=0.02, T p<0.001	with outcome
<b>16</b> Moriyama 2013	38 (only 34 with Oxford scores)	not said [8]	mean about 38y	?	50% decline in p'u ?n	all at 1y	? / ?	mean BP, p'u, h't, eGFR, EPA therapy; MEST	ONLY EPA p=0.0285	crescents not done <b>[not I'supp in multivar]</b>
<b>17</b> Ochi 2013	41 = 26 tonsillect- omy + steroids, 15 steroids	not said [not said]	mean 31.1y	under 1y fup	clinical remiss- ion 21/41 51%	all at 1y	? / all	eGFR, BP, p'u, time from onset, tonsillex; MEST <b>[all I'supp]</b>	<b>eGFR</b> <b>p=0.0021</b> , tonsillex p=0.0002, T p=0.0374	crescents not done

18 Tanaka	698	not said	mean	none	ESRD	median	? / ?	eGFR, p'u;	<b>p'u 1-3.5</b>	crescents
2013	derivation	[10]	36.1y		73/698	4.7y		MST1T2 [not	<b>p=0.01,</b>	not done
	cohort				10%			<b>l'supp]</b>	<b>p'u &gt;3.5</b>	
									<b>p&lt;0.001,</b>	
									<b>eGFR 15-</b>	
									<b>29</b>	
									<b>p=0.003,</b>	
									<b>eGFR &lt;15</b>	
									<b>p&lt;0.001,</b>	
									M1	
									p=0.03; S1	
									p=0.003;	
									T1	
									p<0.001,	
									T2	
									p<0.001	

19 Coppo 2014 =VALIGA	1147	centrally reviewed [8]	mean 36y, 174 under 18y	none	1: 50% decline in eGFR or ESRD (135): 16%; 2: rate eGFR decline	median 4.7y, minim- um 12m*	10% / 46%	Initial eGFR, fup p'u, fup MAP; MST <b>[not l'supp]</b>	1: S1 p=0.02, T p<0.001; 2: M1 p=0.04, T p=0.01 [ <b>no clin stats</b> ]	E not predict any outcome, even in no l'supp: cf 1 Cattran: crescents present or absent
20 Espinosa 2014	283	locally at 11 centres / not said [not said]	mean 39.1y	none	ESRD 58/283 20%	6y median	? / 35%? Ambig- uous	age, eGFR, p'u, BP; C4d staining; ST1T2 [ <b>not l'supp</b> ]	<b>eGFR</b> <b>p&lt;0.001,</b> <b>p'u</b> <b>p=0.01,</b> C4d staining	crescents not done

									p=0.01, T2 p=0.01	
<b>21</b> Faria 2014	74 adults	not said [8]	18-78y, median 39y	?	50% decline in eGFR or ESRD (16): 20/74 27%	4y median	? / 22%	eGFR, p'u, MEST [ <b>not I'supp</b> ] [both logistic regression, Cox]	<b>eGFR [log p=0.018, Cox p&lt;0.0005], T1/2 [log p=0.011, Cox p=0.027]</b>	crescents not done
<b>22</b> Kfoury 2014	70	4 [not said]	mean 32.2y	?	50% decline in eGFR n?	3.5y median	? / ?	age, sex, eGFR, BP; MEST [ <b>not I'supp</b> ]	none of MEST significant [ <b>no clin stats</b> ]	crescents not done

23 Lee 2014	430 adults	1 [8]	19-73y, mean 34.9y	?	1: 50% decline in eGFR 59/430 14%; 2: rate eGFR decline	61m mean, minim- um 6m*	? / 10%? Ambig- uous	i: eGFR, p'u, MAP; C; ii: eGFR, p'u, MAP; MEST1T2; iii: eGFR, p'u, MAP; MEST1T2C [MESTC for rate decline] [not I'supp]	1: i-iii: <b>eGFR</b> <b>p&lt;0.001,</b> <b>p'u</b> <b>p&lt;0.001;</b> i not C; ii: T1 p=0.01, T2 p=0.001; iii: T1 p=0.003, T2 p=0.001, not C; 2: iii: S1 p=0.03, T	crescents present or absent, not associated with outcome [no clin stats for rate eGFR decline]
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									p=0.04, not C	
<b>24</b> Moriyama 2014	1012 (only 857 with Oxford scores)	not said [8]	32.9y mean	?	ESRD n?	mean 7.9y	? / 40%	age, sex, BMI, eGFR, p'u, MAP, salb, suric, chol, IgA/C3; T [not I'supp]	<b>eGFR</b> <b>p&lt;0.0001</b> , suric p=0.0176, <b>p'u</b> <b>p=0.0116</b> ; not T	crescents not in analysis
<b>25</b> Park 2014	500 adults	not said [8]	at least 18y; mean 37.1y	?	doubled scr or ESRD (35) 10% 52/500	mean 68m, minim- um 12m	? / 11%	age, eGFR, BP, MAP, p'u; MEST1T2 [not I'supp]	<b>p'u</b> <b>p&lt;0.001</b> , <b>eGFR</b> <b>p=0.002</b> ; T1 p<0.001,	crescents not done

									T2 p<0.001	
<b>26</b> Oshima 2015	31 aged at least 60y  (only 26 with Oxford scores)	not said [8]	62-69y, median 64.5y	?	ESRD 8/31 26%	mean 4.85y, minim- um 6m	? / 16 patients 52%	MAP, salb, eGFR, p'u, slgA, DM; M  <b>[not I'supp]</b>	none, including clinical	crescents not done
<b>27</b> Shen 2015	60 with 2 biopsies and i'supp after first biopsy	2 [10]	mean 28.9y at first biopsy	under 6m between 2 biopsies	30% decline in eGFR or ESRD after second biopsy 15/60 25%	median 32m (after first biopsy?)	? / all	A: initial eGFR, p'u, MAP; METC, necrosis; B: initial eGFR, fup p'u, fup MAP; METC, necrosis <b>[all</b>	on first biopsy: A: M1 p=0.023, T p=0.049; B: M1 p=0.017, T p=0.049; on second	crescents present or absent; capillary necrosis present or absent

								<b>I'supp, no clin stats]</b>	biopsy: A: T p=0.032; B: T p=0.011	
<b>28</b> Barbour 2016	901 adults from other studies	differ between studies [differ between studies]	at least 18y; mean 38.1y	ESRD at biopsy	50% decline in eGFR or ESRD 162/901 18%	5.6y median, minim- um 2y	? / 36%	initial eGFR, p'u, MAP; combined MEST score; MEST1T2 [not <b>I'supp]</b>	<b>eGFR</b> <b>p=0.02,</b> <b>p'u</b> <b>p&lt;0.0001,</b> <b>MAP</b> <b>p&lt;0.0001,</b> MEST score p<0.0001, M1 p=0.018, T1	crescents not in analysis

									P<0.0001, T2 p<0.0001	
29 Caliskan 2016	111	1 [8]	16-75y, mean 35y	none	50% decline in eGFR or ESRD (37) 37% 41/111	mean 33m, minim- um 12m*	? / 47%	age, sex, BMI, eGFR, p'u, BP, Hb, salb, suric, C3 staining intensity; MEST [ <b>not I'supp</b> ]	<b>eGFR</b> <b>p=0.004</b> , Hb p=0.037, suric p=0.046, C3 intensity p=0.049; none of MEST	crescents not in analysis

30 Chakera 2016	147 adults	2 or 3 [8]	over 18y; median 39.93y	any I'supp treatment	1: ESRD 56/147 38% 2: rapid loss eGFR >5 ml/min/ 1.73m <sup>2</sup> /y 42/147 29%	82m mean	none / none	eGFR, p'u; MESTC [none I'supp]	1: eGFR p<0.001, p'u p=0.013, E1 p<0.001; 2: p'u p=0.006, E1 p=0.025, T2 p=0.011	crescents present or absent [some p values differ between text and Abstract]
31 Iwasaki 2016	75 with heavy p'u, all I'supp (only 61	not said [8]	mean 32.3y	p'u <1g	ESRD n?	minim- um 12m	? / all	eGFR; T [all I'supp]	eGFR p=0.006	crescents not done

	with Oxford scores)									
32 Kaneko 2016	314	5 on 45 biopsies, 1 on 269 biopsies [8]	13-72y, median 36y	eGFR <30	50% decline in eGFR or ESRD 14% 43/314	106m median, minimum 12m	? / 60%	only on 186 with p'u at least 0.5g: MAP, eGFR, p'u, age; Ex; arteriolar hyalinosis	<b>eGFR p=0.041 [not l'supp in multivar]</b>	Ex = crescent in >10% of circumference of glomerulus
33 Li 2016	105	1 [not said]	median 34y	?	30% decline in eGFR 27% 28/105	13m median	none / 36%	vit D deficiency, sex, systBP, p'u, eGFR, salb; T	vit D deficiency p=0.008, <b>systBP p=0.036</b>	crescents not done [not l'supp in multivar]
34 Shin 2016	627 adults	1 [8]	at least 20y,	eGFR <30	1: 50% decline in	56.8m mean,	? / 7%	1: A: sex, age, MAP, eGFR,	1: A: <b>p'u p=0.01</b> , S1	crescents not done

			median 32y		eGFR or ESRD (47) 10% 62/627; 2: rate eGFR decline	minim- um 6m		p'u; MEST1T2; 1: B: sex, age, MAP, eGFR, p'u, MEST1T2, IgG deposit grades; 2: eGFR, MAP, p'u; MEST, IgG deposit grades [ <b>not I'supp</b> ]	p=0.001, T2 p<0.001; 1: B: <b>p'u</b> <b>p=0.04</b> , S1 p=0.002, T2 p<0.001, IgG grade1 p=0.03, grade 2/3 p=0.04; 2: S1 p<0.001, T2 p<0.001,	[ <b>no clin stats in 2: rate eGFR decline</b> ]
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									IgG grade 2/3 p<0.001	
<b>35</b> Stefan 2016	121	1 [8]	over 18y; mean 40.1y	?	doubled scr or ESRD 34/121 28%	59.7m mean	? / 49%	A: eGFR, p'u, BP; MESTC; B: age, sex, eGFR, p'u, BP, h't; IgG deposition; <b>I'supp</b> ; MESTC	A: <b>eGFR</b> <b>p&lt;0.01</b> , C p=0.03; B: <b>eGFR</b> <b>p=0.001</b> , S1 p=0.03, C p=0.004; <b>I'supp NS</b>	crescents present or absent [ <b>not</b> <b>I'supp in A</b> ]
<b>36</b> Woo 2016 see <b>80</b> Woo 2021	102 with stored blocks from 1970-1983	1 [8]	mean 24.36y	?	ESRD at 5y 14% 14/102; at 20y 39%	mean 21.4y	none / ? not said: "non- random use of	age at last fup, sex, BP, eGFR, p'u, h't, MESTC, arterial int	at 5y: <b>p'u</b> <b>p=0.007</b> , h't p=0.001, T p=0.03,	crescents not associated with outcome <b>no</b>

					40/102; at 30y 47% 48/102		immuno- supp- ression”	thick, % of gloms global scl, seg scl [how different from S1?]  [not I’supp]	seg scl? [NOT S1] p=0.02; at 20y: T p=0.04; at 30y: M1 p=0.03, global scl p=0.01	<b>clin signif at 20y or 30y</b>
<b>37 Coppo 2017</b>	261 in VALIGA	centrally reviewed [8]	all under 23y, mean 15.27y: 174 under 18y,	none	1: 50% decline in eGFR or ESRD (14) 23/261 9%; 2:	4.91y median: 4.63y median under 18y; minim-	? / 48%: 51% under 18y	1: fup p’u, fup BP, MST; [not I’supp] 2: age, sex, initial eGFR, fup p’u, fup	1: all: M1 p=0.016, S1 p=0.018, T p=0.008; under 18y: S1	crescents present or absent [no <b>clin stats in 1: RF or 2: rate eGFR decline]</b>

			mean 12.72y		rate eGFR decline	um 12m*		BP; MST [ <b>not I'supp</b> ]	p=0.016; 2: all and under 18y: none of MST	
<b>38</b> Fabiano 2017	54 children	1 [6]	2-18y, mean 9.7y	eGFR <60	50% decline in eGFR 19% 10/54 (ESRD 5)	90m mean, minim- um 9m	? / 13 patients 24%	initial p'u, E <b>[not I'supp]</b>	<b>p'u</b> <b>p=0.003</b> , E1 p=0.002	crescents present or absent
<b>39</b> Haas 2017	3096 from other studies = 234 Oxford, 1145	different between studies [8 or 10]	mean 35y	different between studies	50% decline in eGFR or ESRD (?264)	4.7y median, minim- um 12m*	? / 37%	initial eGFR, fup p'u, fup MAP; MESTC <b>[not I'supp]</b> [1950 no	All: <b>eGFR</b> <b>and p'u</b> <b>p&lt;0.001</b> , <b>MAP</b> <b>p=0.001</b> ,	crescents present or absent

	VALIGA, 700 Japanese, 1017 Chinese				2312/ 2396 13%			l'supp, 1146 l'supp]	M1 p=0.003, S1 p=0.03, T p<0.001, C p=0.01; No l'supp: <b>eGFR and p'u p&lt;0.001,</b> M1 p=0.01, T p<0.001, C p<0.01; l'supp: <b>p'u and MAP</b>	
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									<b>p&lt;0.001, T</b> p<0.001	
<b>40</b> Kaihan 2017	86 adults	7 [8]	at least 18y; median 36y	?	50% increase in scr 13/86 15%	6.8y median, minim- um 12m	1 patient / 57 patients 66%	age, sex, eGFR, p'u, BP, suric; T [ <b>not</b> <b>I'supp</b> ]	T p=0.04 <b>no clin</b> <b>signif</b>	crescents present or absent; <b>I'supp NS</b> <b>univariate</b>
<b>41</b> Knoop 2017	145 "assumed benign" (only 123 with Oxford scores)	2 [not said]	mean 30.1y	eGFR <60; p'u at least 1g	1: 50% decline in eGFR or ESRD (4) 19% 27/145; 2: remiss-	22y median	? / none	1, 2: "clinical variables" at biopsy including p'u, BP, eGFR; MESTC [ <b>none</b> <b>I'supp</b> ]	<b>none of</b> <b>"clinical</b> <b>variables",</b> none of MESTC	crescents present or absent; all biopsies T0

					ion 29% 42/145					
42 Lv 2017 see 88 Lv 2022	262 = TESTING randomised trial	not said [not said]	over 14y, mean 38.6y	eGFR <20 or >120; p'u <1	40% decline in eGFR or ESRD (14) or death kidney disease 28/262 11%	25m median	8%? / 126 placebo, 136 steroid	eGFR, p'u, maximum dose ARB/ACEi, treatment; E	treatment	crescents not done
43 Sevillano 2017	112	1 [not said]	mean 41.5y	?	ESRD 19% 21/112	mean 14y, minim- um 12m	? / 39%	time-aver- aged (TA) h't, TA p'u, age, eGFR, BP,	h't p=0.04, p'u p=0.04, eGFR	crescents not done I'supp NS univariate

								[not i'supp]; MEST	p=0.03; T p=0.001	not clear if in multivar.
44 Shu 2017	150 adults = 50 ESRD surviving <10y since biopsy, 100 surviving >10y since biopsy	not said [not said]	at least 18y, mean about 35y	usual	ESRD (50) 33%	all at 10y	? /?	MEST1T2; initial eGFR, sex, age, suric, Hb, salb, chol, p'u, BP, h't; fup TA suric, salb, Hb, chol [not I'supp]	M1 p=0.018, EST1T2 NS; initial eGFR p=0.039; TA suric p=0.026, TA Hb p=0.029	crescents not done
45 Yang 2017	919	not said [not said]	10-75y, mean 36.8y	eGFR <15	ESRD 13% 124/919	57.46m median, minim- um 12m	none / 57%	age, sex, eGFR, systBP, BUN, suric, low salb, trig,	age p=0.01, sex p=0.01,	crescents not done

								Hb, sC3, nephrotic range p'u, nonB blood group, <b>steroids; T</b>	<b>eGFR</b> <b>p&lt;0.001</b> , low salb p=0.01, Hb p=0.02, nonB blood group p=0.002, <b>steroids</b> <b>p=0.048; T</b> p<0.001	
<b>46</b> Yoon 2017	377 at "early stage"	1 [not said]	18-75y, mean 38.5y	eGFR<50; p'u at least 3.5g/gcr; persistent	1: persis- tent p'u = Upr/cr >1g/g	43.5m median	? / 18%	1: age, sex, eGFR, p'u, MAP; MEST1; <b>[not l'supp];</b>	1: M1 p<0.001; 2: M1	crescents not done

				p'u at bx = Upr/cr >1g/g; severe fibrosis = T2	48/377 13%; 2: 30% decline in eGFR 52/377 14%			2: age, sex, eGFR, p'u, MAP; RASB, <b>steroids;</b> MEST1	p=0.02 [ <b>no clin stats</b> ]	
<b>47</b> Zhang 2017 Medicine compare <b>57</b> Zhang 2018	538 with crescents	3 [10]	at least 14y; mean 32y	eGFR <15	doubled scr or ESRD (59) 69/538 13%	51m median	? / "approx- imately 40%"	eGFR, p'u, BP; MESTC [C by 5% increase of any crescents, including fibrous] [ <b>not I'supp</b> ]	<b>eGFR</b> <b>p&lt;0.001,</b> <b>p'u</b> <b>p&lt;0.001,</b> <b>BP p=0.02,</b> M1 p=0.001, S1	crescent = more than 2 cell layers involving >10% of circum- ference of Bowman's capsule

									p<0.001, C p=0.02	
48 Zhang 2017 Oncotarget	672	3 [not said]	28-45y, median 36y	eGFR <15	50% decline in eGFR or ESRD (30) 13% 90/672	36m median, minim- um 6m	none / 85%? Ambig- uous	sex, age, chol, trig, HDL, LDL, scr, p'u, salb, suric, Hb, systBP, diastBP, sfibrinogen quartiles; MT1T2C [not I'supp]	salb p=0.008, suric p=0.011, fibrin Q3 p=0.047, fibrin Q4 p=0.018, T2 p=0.004	crescents present or absent
49 Zhu 2017	742	at least 2 [7]	mean 32.7y	eGFR <30	50% decline in eGFR or ESRD ?n	at least 3y	? / 40% "treat- ment" =	MEST, initial p'u, "treatment"	T p=0.000, p'u p=0.000	crescents not done

							I'supp OR RASB			
50 Cambier 2018	82 children (only 77 followed)	2 [not said]	under 18y, median 11.3y	none?	10% decline in eGFR or stage 3CKD at 6m 9/77 12%	all at 6m	? / 51/82  62%	A: initial eGFR & p'u, age at diagnosis, S, P = tip lesion / podocyte- opathy; B: as A = fup p'u, <b>I'supp</b>	A: S1 p=0.029, P1 p<0.001; B: only P1 p=0.004 <b>[no clin stats]</b>	crescents  present or absent
51 Chen 2018	506 adults with mild proteinuria	not said [8]	at least 18y; mean 34.7y	p'u at least 1; eGFR <60	50% increase in scr 54/506 11%	50m median, minim- um 12m	none / 14%	age, sex, chol, trig, <b>steroids</b> , ACEi/ARB; p'u, BP, CKD2; MESTC	age p=0.001, <b>p'u</b> <b>p=0.003</b> ; none of MESTC	crescents absent C0 or not over 25% C1 <b>steroids NS</b> <b>multivar</b>

52 Deng 2018	988	2 [10]	at least 14y; median 32y	eGFR <15	doubled scr or ESRD (91) 107/988 11%	48.6m median, minim- um 12m	? / 32%? Ambig- uous	age, eGFR, p'u, BP; global scl, ST [not l'supp]	Male: p'u p=0.032, eGFR p<0.001, global scl p=0.028, S1 p=0.002; Female: p'u p=0.023, eGFR p<0.001, S1 p=0.012	crescents present or absent
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<b>53</b> Palamuthus- ingam 2018	45	not said [8]	mean 48y	none	50% increase in scr or ESRD ?n	2.6y mean	? / 18 patients 40%	<b>I'supp</b> , eGFR <50, >1g/24h p'u, MTC	ESRD: T; 50% rise scr: none <b>no clin sig</b>	crescents present or absent
<b>54</b> Pan 2018 HumPath	365 = 73 glomerular necrosis, 292 matched controls	2 [not said]	mean 39.1y	?	50% decline in eGFR or ESRD 10% 35/365	median about 32m, minim- um 3m	? / 56%	sex, eGFR, p'u, salb, Hb, sfibrinogen, h't, systBP, diastBP, ARB, <b>steroids</b> , glom necrosis, METC	<b>eGFR</b> <b>p&lt;0.001</b> , fibrinogen p=0.018, h't p=0.02, <b>steroids</b> <b>p&lt;0.001</b> , none of METC	crescents not associated with outcome
<b>55</b> Pan 2018 ImmRes	712	2 or 5 [not said]	mean 37.36y	eGFR <15	50% decline in eGFR or	mean 40m,	none / 86%?	Basic model: sex, C3/C4 ratio, scr, p'u,	Basic: C3/C4 p=0.02, <b>scr</b>	on univariate, C1

					ESRD (58) 84/712 12%	minim- um 3m	Ambig- uous	suric, systBP, diastBP, Hb, salb, MT1T2; Optimal model: C3/C4 ratio, scr, p'u, suric, systBP, T1T2 [ <b>not I'supp</b> ]	<b>p=0.011,</b> suric p=0.019, <b>systBP</b> <b>p=0.026,</b> T1 p=0.019, T2 p=0.003; Optimal: C3/C4 p=0.007, <b>scr</b> <b>p=0.007,</b> suric p=0.005,	favourable but C2 no effect; not consistent with Trimarchi 2017, so C excluded from multivariate
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									<b>systBP</b> <b>p&lt;0.001,</b> T1 p=0.034, T2 p=0.001	
56 Schimpf 2018	70 whose renal biopsy was available from 162 in STOP-IgA randomised trial	1 [8]	18-70y; mean 43.4y	p'u <0.7 or >3.5; BP <140/90; eGFR at least 90 or <30	1: remiss- ion 8/70 11%; 2: eGFR loss at least 15 23/70 33%; 3: ESRD 8/70 11%	median 9.4m from biopsy to random -ization, then 3y trial phase	none / 38 patients 54%	eGFR, p'u, <b>treatment</b> <b>arm; MEST</b>	1: remission, none; 2: eGFR loss none; 3: ESRD: T p=0.01 [ <b>no</b> <b>clin stats</b> ]	crescents present or absent, not in Cox

57 Zhang 2018 compare 47 Zhang 2017 Medicine	1152	2 [8]	mean 35.4y	?	ESRD 13% 144/ 1152	median 45m, minim- um 12m*	? / 53%	age, sex, eGFR, MAP, p'u, MESTC1C2, +/- I'supp	all, or with I'supp, or without I'supp: not C [no clin stats]	crescent: extracap- illary lesion of any size containing more than two cell layers
58 Barbour 2019	2781 adults in derivation cohort, from other studies = 1192 VALIGA,	different between studies [8 or 10]	at least 18y; median 35.6y	ESRD at biopsy	50% decline in eGFR (420) or ESRD (372) 492/	median 4.8y	9% / 43%	Limited model: eGFR, p'u, MAP; MEST; [not I'supp]; Full model: age, sex, BMI, eGFR, p'u,	Limited: eGFR p<0.001, p'u p<0.001, M1 p=0.002, T1	crescents not in multivariate analysis I'supp NS

	568 Japanese, 1021 Chinese				2781  18%			MAP; RASB at bx, <b>l'supp</b> at bx; MEST; +/- race; + various interaction terms	p<0.001, T2 p<0.001; Full: age p<0.001, <b>eGFR</b> <b>p&lt;0.001</b> , T1 p<0.001, T2 p<0.001; plus others	
59 Duan 2019	412	2 [8]	at least 14y,	eGFR <15	50% decline in eGFR or	mean 80.6m,	? / 79%? Ambig- uous	p'u, MAP, eGFR;	<b>p'u</b> <b>p=0.035</b> , <b>MAP</b>	crescents not associated

			mean 34.95y		ESRD (44) 17% 70/412	minim- um 36m		MEST2C [not I'supp]	p<0.001, M1 p=0.001, T2 (vs T0/1) p<0.001	with outcome
60 Kamiyama 2019	87 with T1/2, only 44 studied = matched 22 anti- platelet agents vs 22 RAS inhibitors	not said [not said]	over 16y, median 32.5y APA, 48y RASI	?	ESRD 59% 26/44?	Mean 8.5y APA, 10.5y RASI	none / none	APA vs RASI, BMI, MAP, eGFR, salb, suric, p'u; M <b>none I'supp</b>	treatment without RASI p=0.0039 <b>no other clin signif</b>	crescents not associated with outcome

61 Park 2019	3380	not said [8]	?	?	50% decline in eGFR or ESRD (417) 700/ 3380 21%	median 8.74y	? / 24%	age, sex, MAP, eGFR, p'u, global scl, int fibrosis, t atrophy, SC [not l'supp]	age p<0.001, sex p<0.001, <b>eGFR</b> <b>p&lt;0.001,</b> <b>p'u</b> <b>p&lt;0.001,</b> global scl p<0.001, int fib severe p=0.02, S1 p=0.03, C1 p=0.002,	MET not Oxford
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									C2 p<0.001	
62 Peng 2019	1328	2 [8]	at least 14y, mean 34.2y	?	50% decline in eGFR or ESRD (171) 17% 221/ 1328	mean 46.1m, minim- um 12m*	? / 61%	sex, BP, nephrotic at bx, scr, I'supp, MESTC1C2, global scl G1 26-50%, G2 >50%	BP p<0.001, NS p=0.002, scr p<0.001, I'supp p<0.001, E1 p=0.045, T p<0.001, C2 p=0.001, G1	

									p<0.001, G2 p<0.001	
<b>63</b> Champtiaux 2020	32 adults with spondylo- arthritis (only 27 with Oxford scores)	1 [8]	median 37y	?	doubled scr (13) or eGFR <30 (9) 22/32 69%	median 5.9y	? / 14 patients 44%	eGFR <60 at bx, p'u, T, global scl, rate eGFR loss [ <b>not</b> <b>I'supp</b> ]	<b>eGFR</b> <b>p=0.018</b>	crescents not associated with outcome
<b>64</b> Coppo 2020	1130 in VALIGA	centrally reviewed [8]	mean 35y	eGFR <15	1: 50% decline in eGFR or ESRD (212) 279/	median 7y	? / 48%	age, sex, BMI, eGFR, MAP, p'u; MESTC, arterio- sclerosis [ <b>not</b> <b>I'supp</b> ]	1: All: <b>eGFR</b> <b>p&lt;0.001,</b> <b>MAP</b> <b>p=0.005,</b> <b>p'u</b>	

					1130					<b>p&lt;0.001,</b>	
					25%; 2:					M1	
					rate					p=0.037,	
					eGFR					S1 p=0.01,	
					decline					T p<0.001;	
										1: 582 no	
										l'supp:	
										<b>eGFR</b>	
										<b>p=0.001,</b>	
										<b>MAP</b>	
										<b>p=0.03,</b>	
										<b>p'u</b>	
										<b>p&lt;0.001,</b>	
										M1	
										p=0.003, T	
										P<0.001;	

									2: All: <b>eGFR</b> <b>p&lt;0.001,</b> <b>MAP</b> <b>p&lt;0.001,</b> <b>p'u</b> <b>p=0.02, T</b> P<0.001; 2: 582 no l'supp: <b>p'u</b> <b>p=0.004, T</b> p=0.003, C p=0.01	
<b>65</b> Jebali 2020	50 adults	1 [8]	at least 18y,	?	ESRD 20% 10/50	median 30.5m	none / 21	BP, p'u, eGFR, <b>steroids, T</b>	T p=0.038 <b>no clin</b> <b>signif</b>	crescents not associated

			mean 35.6y				patients 42%			with outcome
66 Jullien 2020	168 with 2 biopsies	2 [8]	adult: mean 33.6y at first biopsy, 40.0 at second biopsy	?	ESRD 37/168 22%	18.4y median after first biopsy	? / 26% after first biopsy, 46% after second biopsy	Model 1: eGFR, p'u, BP at time of second biopsy; MESTC on first biopsy; Model 2: eGFR, p'u, BP at time of second biopsy; MESTC on second	all 168 bxs: Model 1: <b>eGFR</b> <b>p&lt;0.05,</b> <b>p'u</b> <b>p&lt;0.05,</b> none of MESTC; Model 2: <b>eGFR</b> <b>p&lt;0.05,</b> <b>p'u</b> <b>p&lt;0.05, T</b>	<b>use of steroids between biopsies associated with worse outcome</b> (data not shown)

								biopsy: +/- <b>steroids</b> <b>between</b> <b>biopsies</b>	P<0.05: 112 protocol bxs: Model 1: <b>p'u</b> <b>p&lt;0.05,</b> M1 P<0.05; Model 2: <b>p'u</b> <b>p&lt;0.05,</b> M1 P<0.05, T P<0.05	
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67	871	not said	median	?	ESRD	median	? / 49%	sex, age, BMI, eGFR, MAP, p'u, h't; MESTC [not I'supp] [445 no i'supp, 426 i'supp]	All: <b>MAP</b> <b>p=0.0046,</b> <b>eGFR</b> <b>p&lt;0.0001,</b> <b>p'u</b> <b>p&lt;0.0001,</b> T p=0.0085; No i'supp: male p=0.0394, <b>eGFR</b> <b>p=0.0002,</b> <b>p'u</b> <b>p&lt;0.0001;</b> I'supp:
Moriyama		[8]	31y		115/871	8y,			
2020					13%	minim- um 12m*			

									eGFR p<0.0001, p'u p<0.0001, T p=0.0287	
68 Neves 2020 KidBPre	111 (only 101 followed)	1 [8]	median 32y	?	doubled scr or ESRD (31) 30% 33/111?	median 64m	? / 66%	sex, p'u, BP, eGFR, h't, STC [not l'supp]	S1 p=0.04, T p=0.027, C p=0.031 [no clin stats]	
69 Neves 2020 PLOSOne	118	1 [8]	median 33y	solid organ tx, bmtx, pregnant, cancer, drugs	ESRD 36/118 31%	median 65m	? / 63%? Ambig- uous	sex, BP, scr, morphologic TMA; MESTC [not l'supp]	female protective p=0.03, T p=0.002,	crescents not associated with outcome

				associated TMA					TMA p=0.01	
70 Park 2020	453 adults	1 [not said]	18-74y; mean 40.3y	?	30% decline in eGFR (46) or increase p'u to at least 3.5 (17): 50/453 11%	median 33m	? / 19%	1: age, sex, BP; MSTC, mesangial C3; <b>[not l'supp]</b> 2: age, sex, eGFR, p'u, MAP; MSTC, C3 <b>[not l'supp]</b> 3: age, sex, eGFR, p'u, MAP; <b>steroids;</b> MSTC, C3	1: M1 p<0.001, S1 p=0.007, T p<0.001, C3 p<0.001; 2: M1 p=0.021, S1 p=0.002, T p<0.001, C3 p=0.003;	crescents not associated with outcome <b>[no clin stats]</b>

									3: M1 p=0.020, S1 p=0.002, T p<0.001, C3 p=0.003	
71 Wu 2020	1243 children	at least 2 [8]	not over 18y, mean 14y	eGFR <15	50% decline in eGFR or ESRD (82) 171/ 1243 14%	median 7.2y, minim- um 12m	? / 64%	eGFR, MAP, p'u; STC [ <b>not I'supp</b> ]	S1 p<0.001, T p<0.001 [ <b>no clin stats</b> ]	(C only without I'supp)

72 Aratani 2021	149 = 110 steroids + tonsillect- omy, 39 conservat- ive	not said [not said]	median 39y	previous steroids; only steroids or only tonsil- lectomy	progress- ion of CKD stages n?	3y	none / 74%	treatment = I'supp + tonsilleX, p'u, CKD1/2, MST	treatment p=0.039, p'u p=0.023	only 25/149 given C score
73 Bobart 2021	125 (only 72 followed)	1 [8]	mean 44.8y	eGFR <15	ESRD 9/72 13%	median 3.69y	none / ? not clear how many	age, sex, scr, eGFR, systBP, diastBP, p'u, h't, RASB; MESTC [not I'supp]	scr p=0.001, eGFR p=0.004, systBP p<0.001, p'u p<0.001, T p=0.018	crescents not associated with outcome: not clear if uni or multi variate

74 Canney 2021	1864 adults in “remission” of p’u, from 6 studies = 659 Nanjing, 561 VALIGA, 293 Japan, 185 Beijing, 110 Oxford, 56 North America	different in different studies	at least 18y, median 36.8y at remission	eGFR <15; p’u at least 1, and without 25% reduction in p’u from at least 1	50% decline in eGFR or ESRD (46) 15% 274/ 1864	median 3.9y from remission	? / 42%	each 3m in remission, to 4y, over 4y; eGFR, p’u, MAP, all at remission; peak p’u before remission; MESTC; age; sex; race; time to remission; RAAS at remission; <b>I’supp at or</b>	each 3m in remission to 4y, p<0.001, <b>eGFR</b> <b>p&lt;0.001;</b> T1 p=0.04, T2 p=0.002; age p=0.02; race: Chinese >24m fup p<0.001, Japanese	crests not associated with outcome <b>I’supp NS</b>
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								<b>before remission</b>	p=0.01; RAAS p=0.01	
75 Hwang 2021	545 adults	not said [not said]	at least 20y; median 39y	?	50% decline in eGFR or ESRD (37) 53/545 10%	median 3.6y	2 patients / 22%	age, eGFR, MAP, p'u, RASB; MEST1T2 C1C2 [not I'supp]	age p=0.028, <b>p'u</b> <b>p=0.045,</b> <b>eGFR</b> <b>p&lt;0.001,</b> M1 p=0.043, T1 p=0.005, T2 p<0.001	

76 Lin 2021	305 adults “without obvious chronic lesions”	2 [not said]	over 18y, median 31y	global scl at least 25%, T2, C2	decline in eGFR >15% in 1y, or >30% in 2y 34/305 11%	median 34.8m, minim- um 12m*	? / 36%	Model 1: ALL: [NOT eGFR] sex, MAP, trig, T1, seg C1, global C1; [not I’supp] Model 2 ALL = Model 1 + I’supp; Model 3, no I’supp = [NOT eGFR] sex, MAP, trig, T1, seg C1, global C1; Model 4, I’supp = same	Model 1: female p=0.003, <b>MAP</b> <b>p=0.017</b> , trig p=0.011; global C1 p=0.036; Model 2: female p=0.001, <b>MAP</b> <b>p=0.013</b> , trig p=0.025;	global crescent = extracapill- ary lesion of more than 2 cell layers in >50% of glomerular circum- ference; segmental crescent = in <50%
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								as Model 3 <b>no clin signif in Model 3</b>	<b>I'supp</b> <b>p=0.037;</b> Model 3: global C1 p=0.047; Model 4: female p=0.009	
<b>77</b> Moreno 2021	89	2 [10]	at least 15y, 16- 83y, median 45y	none	50% decline in eGFR or ESRD ?n	mean 52m, minim- um 12m*	? / 45%	p'u, mac h't, ET2C2 [ <b>not I'supp</b> ]	C2 p=0.03, T2 p=0.03 <b>no clin signif</b>	
<b>78</b> Schena 2021	948 adults = 876	not said [not said]	at least 18y:	?	ESRD 210/948 22%	median 89m	? / 27%	age, sex, BP, p'u, scr, RASB, <b>I'supp</b> ;	<b>p'u</b> <b>p&lt;0.001,</b> <b>scr</b>	crescents not associated

	VALIGA + 72 Greece		mean 40.6y					M1E1S1T1T2 C	<b>p&lt;0.001,</b> RASB p=0.03, <b>l'supp</b> <b>p=0.002,</b> S1 p=0.007, T1 p<0.001, T2 p<0.001	with outcome
<b>79</b> Tang 2021	501 with "renal vasculitic lesions" = thrombi in	not said [10]	at least 14y, median 31y	eGFR <15	doubled scr or ESRD ?n	mean 49m	? / 28%? Ambig- uous	age, sex, BP, duration, Hb, p'u, salb, suric, LDL, eGFR, global	<b>p'u</b> <b>p=0.041,</b> Hb p=0.015, <b>eGFR</b>	all with "vasculitic lesions"; C1/2 in 85%

	arteries +/- arterioles, capillary loop necrosis, crescents, fibrinoid necrosis of small blood vessels							scl; M1E1S1T [not l'supp]	<b>p=0.001,</b> gscl p=0.001, S1 p=0.004	
<b>80</b> Woo 2021 see <b>36</b> Woo 2016	A 102 from 1976-1986 SAME AS WOO 2016, B 177 from 2008-2018	1 [8]	mean A 26.4y B 42.6y	?	ESRD at 10y A 14/102 14% B 34/177 19%	mean A 51m B 61.3m	A none / how many? B ? / 54%	age, sex, BP, initial scr, final scr, initial eGFR, final eGFR, h't, p'u,	A <b>BP</b> <b>p=0.002,</b> <b>init scr</b> <b>p=0.04,</b> last scr p<0.0001,	crescents not associated with outcome; AMBIG-

								M1E1S1TC, arterial int thick, global scl, seg scl <b>[not I'supp]</b>	last eGFR p<0.001, p'u p<0.001, E1 p=0.03, S1 p=0.04, T p=0.001, global scl p<0.0001, seg scl p=0.01; B <b>BP p=0.02,</b> <b>init scr</b> <b>p=0.013,</b> last scr p<0.0001,	UOUS COX not clear if uni or multi variate
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									init eGFR p<0.0001, last eGFR p=0.03, p'u p=0.001, S1 p=0.03, T p<0.0002, global scl p<0.008, seg scl p=0.0003	
<b>81</b> Wora- wichawong 2021	101	not said [5]	mean 37.3y	eGFR <15	50% decline in eGFR or	mean 51.6m	? / ?	MEST1T2C, eGFR, BP, p'u at least 3,	<b>p'u</b> <b>p=0.03</b> , T1 p<0.001,	crescents not associated

					ESRD (20) 26% 26/101			mesangial C4d [not I'supp] [no mention eGFR stats]	T2 p=0.028, C4 p=0.034	with outcome
82 Wu 2021	98 children	2 or 3 [8]	under 18y; median 9.7y	?	50% decline in eGFR or ESRD (1) 6/98 6%	median 25m, minim- um 12m	? / 78 patients 80%	MAP, scr; ST; mesangial C3 and low sC3 [not I'supp]	C3 p=0.029 <b>no clin signif</b>	too few events
83 Yang 2021	384 with crescents	2 [8]	13y-68y, mean 32y	none?	30% decline in eGFR 12% 46/384	mean 32.3m, minim- um 6m	? / 19%? Ambig- uous	eGFR, systBP, p'u, <b>nonstd I'supp</b> , circumfer- ential crescents in	<b>eGFR p=0.001, systBP p=0.026</b> , circ cresc p=0.004,	circum- ferential crescent = at least 50% of glomerular

								>20% gloms, MESTC	<b>other</b> <b>I'supp</b> <b>p=0.018</b>	circum- ference: steroids?
<b>84</b> Caliskan 2022	47	1 [8]	mean 37y	none	ESRD 17/47 36%	median 6y, minim- um 12m*	? / 38 patients 81% [ <b>not</b> <b>I'supp in</b> <b>Cox]</b>	sex, scr, eGFR, mesangial C3, mesangial C4d; MES	<b>eGFR</b> <b>p=0.011</b> , E1 p=0.001	crescents not associated with outcome
<b>85</b> Chen 2022	144	2 [8]	at least 14y, mean C0 34.3y, C+ 36y	?	50% decline in eGFR or ESRD 12% 17/144	median C0 21m, C+ 16m, minim- um 6m	? / ?	A: MAP, eGFR, p'u, ESTC; [ <b>not</b> <b>I'supp]</b> B: MAP, eGFR, p'u, EST, C >10% [ <b>not</b> <b>I'supp]</b>	A: <b>MAP</b> <b>p=0.046</b> , <b>eGFR</b> <b>p=0.004</b> , S1 p=0.04, T p=0.036, C p=0.026; B: <b>eGFR</b>	no mention of treatment

									<p><b>p=0.009,</b> S1 p=0.043, T p=0.036, C &gt;10% p=0.019</p>	
86 Joh 2022	946	5 [10]	2.8 - 87.5y, median 37.1y	none	rate eGFR decline	median 66m	? / 64%	eGFR, MAP, p'u, <b>steroids</b> ; MESTC	<b>eGFR, p'u,</b> <b>steroids,</b> T; each p<0.05	only direct effects considered
87 Kang 2022	4151	not said [8]	at least 18y, median 37y	none	ESRD 7% 304/ 4151	median 6.1y	? / ?	1: eGFR, MAP, p'u, ST; 2: eGFR, MAP, p'u, age, sex, DM,	1: <b>eGFR</b> <b>p&lt;0.001,</b> S p=0.009, T p=0.024; 2: <b>eGFR</b> <b>p&lt;0.001,</b>	E not done; included own chronicity grading; crescents

								MSTC [not I'supp]	<b>DM</b> p=0.031, S p=0.009, T p=0.022	not associated with outcome
88 Lv 2022 see 42 Lv 2017	1: 262 = original; 2: 241 = low dose TESTING randomised trial	not said [not said]	original, over 14y; low dose, over 17y; mean 38y	1: eGFR <20 or >120; p'u <1; 2: eGFR <30 or >120; p'u <1	40% decline in eGFR or ESRD or death kidney disease 74/257 29% steroid, 106/246	median 3.5y: 6.1y original, 2.5y low dose	7%? / original = 126 placebo, 136 steroid; low dose = 120 placebo, 121 steroid	eGFR, p'u, ethnicity, time biopsy to random- ization, treatment; E	treatment	crescents not done; MEST "reviewed by each site"; signif- icantly higher eGFR, more T0 in low dose

					43% placebo					
89 Ruan 2022	458	2 [8]	over 14y, mean 33.2y	eGFR <15	50% decline in eGFR or ESRD 12% 54/458	mean 54.7m	? / 33%?  Ambig- uous	age, eGFR, MAP, ET, C1, C2 [not I'supp = NS univariate]	<b>eGFR</b> <b>p=0.002</b>	crescents not associated with outcome
90 Rui 2022	101	not said [not said]	mean 31y to 36y dep- ending on resp- onse	p'u <1; Oxford score M0E0S0T0 C0; minimal change nephro- pathy	1: 50% decline in eGFR or ESRD 22% 22/101 2: remiss-	mean 26m to 37m depend- ing on resp- onse,	? / all	1: eGFR, p'u, Hb, chol, trig, M1T1T2; 2: diastBP, eGFR, p'u; M1S1T1T2 [all I'supp]	1: <b>eGFR</b> <b>p=0.028</b> , trig p=0.01, M1 p=0.036, T2 p=0.005;	crescents not associated with outcome

					ion or not n?	minim- um 12m			2: eGFR  p=0.008,  M1  p=0.041,  S1 p=0.01	
91 Tang  2022	988	not said  [10]	at least  14y,  median  32y	eGFR <15	doubled  scr or  ESRD ?n	median  49m	? / 51%?  Ambig-  uous	age, sex,  duration, BP,  p'u, salb, Hb,  LDL, suric,  MEST; [not  I'supp; no  clin stats]	T p=0.001	clinical  statistics for  outcome  only given  for T1/T2  group; in  this, cresc-  ents not  significant

92 Xie 2022	821	2 [8]	18-75y, median 34y	ESRD	50% decline in eGFR or ESRD 18% 145/821	median 24m, minim- um 12m*	none / 53%	sex, age, Hb, scr, suric, salb, p'u, treatment, glomerular C3; MST1T2C	scr <b>p&lt;0.001,</b> suric <b>p&lt;0.001,</b> Hb <b>p=0.013;</b> C3 p=0.044; T2 p<0.001	l'supp, crescents not associated with outcome
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**Abbreviations:** Oxford scores: C, (fibro)cellular crescents; E, endocapillary hypercellularity; M, mesangial cellularity; S, segmental sclerosis; T, interstitial fibrosis/tubular atrophy. Others: ACEi, angiotensin converting enzyme inhibitor; APA, anti-platelet agents; ARB, angiotensin 2 receptor blocker; BMI, body mass index; bmtx, bone marrow transplant; BP, blood pressure; BUN, blood urea nitrogen concentration; chol, serum cholesterol concentration; CKD, chronic kidney disease; clin stats, clinical statistics; diastBP, diastolic blood pressure; DM, diabetes mellitus; eGFR, estimated glomerular filtration rate, units ml/min per 1.73m<sup>2</sup>; EPA, eicosapentanoic acid; ESRD, end stage renal disease; Ex, crescent in >10% of circumference of glomerulus; fup, follow-up; g,

glomerulus; Hb, hemoglobin concentration; HDL, serum high density lipoprotein concentration; HIV, human immunodeficiency virus; HSP, Henoch Schoenlein purpura; h't, hematuria; IgA/C3, ratio of serum concentrations of IgA and C3; int, intimal; I'supp, immunosuppression; LDL, serum low density lipoprotein concentration; m, month; mac, macroscopic; MAP, mean arterial pressure; max, maximum; no clin stats, no statistics given for clinical features in multivariate analysis; nonstd, immunosuppression other than steroids; not I'supp, immunosuppression not included in multivariate analysis; NS, not significant; p'u, proteinuria, units g/24h; RASB, renin-angiotensin system blockers; RASI, renin-angiotensin system inhibitors; salb, serum albumin concentration; sC3, serum C3 concentration; scl, sclerosis; scr, serum creatinine concentration; sfibrinogen, serum fibrinogen concentration; sIgA, serum IgA concentration; signif, significant; SLE, systemic lupus erythematosus; suric, serum uric acid concentration; systBP, systolic blood pressure; t, tubule; TA, time averaged; TMA, thrombotic microangiopathy; tonsillex, tonsillectomy; trig, serum triglyceride concentration; tx, transplant; UPr/cr, urine protein/creatinine concentration ratio; vit D, vitamin D; y, year

## References

- 1 Cattran DC, Coppo R, Cook HT, et al. The Oxford classification of IgA nephropathy: rationale, clinicopathological correlations, and classification. *Kidney Internat* 2009; **76**: 534-545.
- 2 Alamartine E, Sauron C, Laurent B, et al. The use of the Oxford classification of IgA nephropathy to predict renal survival. *Clin J Am Soc Nephrol* 2011; **6**: 2384-2388.
- 3 El Karoui K, Hill GS, Karras A, et al. Focal segmental glomerulosclerosis plays a major role in the progression of IgA nephropathy. II. Light microscopic and clinical studies. *Kidney Internat* 2011; **79**: 643-654.

- 4 Katafuchi R, Ninomiya T, Nagata M, et al. Validation study of Oxford classification of IgA nephropathy: the significance of extracapillary proliferation. *Clin J Am Soc Nephrol* 2011; **6**: 2806-2813.
- 5 Shi SF, Wang SX, Jiang L, et al. Pathologic predictors of renal outcome and therapeutic efficacy in IgA nephropathy: validation of the Oxford classification. *Clin J Am Soc Nephrol* 2011; **6**: 2175-2184.
- 6 Yau T, Korbet SM, Schwartz MM, Cimbaluk DJ. The Oxford classification of IgA nephropathy: a retrospective analysis. *Am J Nephrol* 2011; **34**: 435-444.
- 7 El Karoui K, Hill GS, Karras A, et al. A clinicopathologic study of thrombotic microangiopathy in IgA nephropathy. *J Am Soc Nephrol* 2012; **23**: 137-148.
- 8 Gutierrez E, Zamora I, Ballarin JA, et al. Long-term outcomes of IgA nephropathy presenting with minimal or no proteinuria. *J Am Soc Nephrol* 2012; **23**: 1753-1760.
- 9 Halling SE, Soderberg MP, Berg UB. Predictors of outcome in paediatric IgA nephropathy with regard to clinical and histopathological variables (Oxford classification). *Nephrol Dial Transplant* 2012; **27**: 715-722.
- 10 Kataoka H, Ohara M, Shibui K, et al. Overweight and obesity accelerate the progression of IgA nephropathy: prognostic utility of a combination of BMI and histopathological parameters. *Clin Exp Nephrol* 2012; **16**: 706-712.
- 11 Le W, Zeng CH, Liu Z, et al. Validation of the Oxford classification of IgA nephropathy for pediatric patients from China. *BMC Nephrol* 2012; **13**: 158.
- 12 Lee H, Yi SH, Seo MS, et al. Validation of the Oxford classification of IgA nephropathy: a single-center study in Korean adults. *Korean J Intern Med* 2012; **27**: 293-300.
- 13 Moriyama T, Nakayama K, Iwasaki C, et al. Severity of nephrotic IgA nephropathy according to the Oxford classification. *Internat Urol Nephrol* 2012; **44**: 1177-1184.

- 14 Shima Y, Nakanishi K, Hama T, et al. Validity of the Oxford classification of IgA nephropathy in children. *Pediatr Nephrol* 2012; **27**: 783-792.
- 15 Zeng CH, Le W, Ni Z, et al. A multicenter application and evaluation of the Oxford classification of IgA nephropathy in adult Chinese patients. *Am J Kidney Dis* 2012; **60**: 812-820.
- 16 Moriyama T, Iwasaki C, Tanaka K, et al. Effects of combination therapy with renin-angiotensin system inhibitors and eicosapentaenoic acid on IgA nephropathy. *Intern Med* 2013; **52**: 193-199.
- 17 Ochi A, Moriyama T, Takei T, et al. Comparison between steroid pulse therapy alone and in combination with tonsillectomy for IgA nephropathy. *Internat Urol Nephrol* 2013; **45**: 469-476.
- 18 Tanaka S, Ninomiya T, Katafuchi R, et al. Development and validation of a prediction rule using the Oxford classification in IgA nephropathy. *Clin J Am Soc Nephrol* 2013; **8**: 2082-2090.
- 19 Coppo R, Troyanov S, Bellur S, et al. Validation of the Oxford classification of IgA nephropathy in cohorts with different presentations and treatments. *Kidney Internat* 2014; **86**: 828-836.
- 20 Espinosa M, Ortega R, Sanchez M, et al. Association of C4d deposition with clinical outcomes in IgA nephropathy. *Clin J Am Soc Nephrol* 2014; **9**: 897-904.
- 21 Faria B, Henriques C, Matos AC, et al. Combined C4d and CD3 immunostaining predicts immunoglobulin (Ig)A nephropathy progression. *Clin Exp Immunol* 2014; **179**: 354-361.
- 22 Kfoury H, Alsuwaida A, Hussain S, et al. External validation of the Oxford classification of IgA nephropathy: a retrospective study of 70 patients from Saudi Arabia. *Hong Kong J Nephrol* 2014; **16**: 29-33.
- 23 Lee MJ, Kim SJ, Oh HJ, et al. Clinical implication of crescentic lesions in immunoglobulin A nephropathy. *Nephrol Dial Transplant* 2014; **29**: 356-364.

- 24 Moriyama T, Tanaka K, Iwasaki C, et al. Prognosis in IgA nephropathy: 30-year analysis of 1,012 patients at a single center in Japan. *PLOS one* 2014; **9**: e91756.
- 25 Park KS, San SH, Kie JH, et al. Comparison of the Haas and the Oxford classifications for prediction of renal outcome in patients with IgA nephropathy. *Hum Pathol* 2014; **45**: 236-243.
- 26 Oshima Y, Moriyama T, Itabashi M, et al. Characteristics of IgA nephropathy in advanced-age patients. *Internat Urol Nephrol* 2015; **47**:137-145.
- 27 Shen XH, Liang SS, Chen HM, et al. Reversal of active glomerular lesions after immunosuppressive therapy in patients with IgA nephropathy: a repeat-biopsy based observation. *J Nephrol* 2015; **28**: 441-449.
- 28 Barbour SJ, Espino-Hernandez G, Reich HN, et al. The MEST score provides earlier risk prediction in IgA nephropathy. *Kidney Internat* 2016; **89**: 167-175.
- 29 Caliskan Y, Ozluk Y, Celik D, et al. The clinical significance of uric acid and complement activation in the progression of IgA nephropathy. *Kidney Blood Press Res* 2016; **41**: 148-157.
- 30 Chakera A, MacEwen C, Bellur SS, et al. Prognostic value of endocapillary hypercellularity in IgA nephropathy patients with no immunosuppression. *J Nephrol* 2016; **29**: 367-375.
- 31 Iwasaki C, Moriyama T, Tanaka K, et al. Effect of hematuria on the outcome of immunoglobulin A nephropathy with proteinuria. *J Nephropathol* 2016; **5**: 72-78.
- 32 Kaneko Y, Yoshita K, Kono E, et al. Extracapillary proliferation and arteriolar hyalinosis are associated with long-term kidney survival in IgA nephropathy. *Clin Exp Nephrol* 2016; **20**: 569-577.
- 33 Li XH, Huang XP, Pan L, et al. Vitamin D deficiency may predict a poorer outcome of IgA nephropathy. *BMC Nephrol* 2016; **17**:164.

- 34 Shin DH, Lim BJ, Han IM, et al. Glomerular IgG deposition predicts renal outcome in patients with IgA nephropathy. *Modern Pathol* 2016; **29**: 743-752.
- 35 Stefan G, Ismail G, Stancu S, et al. Validation study of Oxford Classification of IgA Nephropathy: the significance of extracapillary hypercellularity and mesangial IgG immunostaining. *Pathol Internat* 2016; **66**: 453-459.
- 36 Woo KT, Lim CC, 1, Foo MWY, et al. 30-year follow-up study of IgA nephritis in a southeast Asian population: an evaluation of the Oxford histological classification. *Clin Nephrol* 2016; **86**: 270-278.
- 37 Coppo R, Lofaro D, Camilla RR, et al. Risk factors for progression in children and young adults with IgA nephropathy: an analysis of 261 cases from the VALIGA European cohort. *Pediatr Nephrol* 2017; **32**: 139-150.
- 38 Fabiano RCG, Araujo SA, Bambirra EA, et al. The Oxford Classification predictors of chronic kidney disease in pediatric patients with IgA nephropathy. *J Pediatr (Rio J)* 2017; **93**:389-397.
- 39 Haas M, Verhave JC, Liu ZH, et al. A multicenter study of the predictive value of crescents in IgA nephropathy. *J Am Soc Nephrol* 2017; **28**: 691-701.
- 40 Kaihan AB, Yasuda Y, Katsuno T, et al. The Japanese histologic classification and T-score in the Oxford classification system could predict renal outcome in Japanese IgA nephropathy patients. *Clin Exp Nephrol* 2017; **21**: 986-994.
- 41 Knoop T, Vikse BE, Mwakimonga A, et al. Long-term outcome in 145 patients with assumed benign immunoglobulin A nephropathy. *Nephrol Dial Transplant* 2017; **32**: 1841-1850.
- 42 Lv J, Zhang H, Wong MG, et al. Effect of oral methylprednisolone on clinical outcomes in patients with IgA nephropathy. *JAMA* 2017; **318**: 432-442.
- 43 Sevillano AM, Gutierrez E, Yuste C, et al. Remission of hematuria improves renal survival in IgA nephropathy. *J Am Soc Nephrol* 2017; **28**: 3089-3099.

- 44 Shu D, Xu F, Su Z, et al. Risk factors of progressive IgA nephropathy which progress to end stage renal disease within ten years: a case-control study. *BMC Nephrol* 2017; **18**: 11.
- 45 Yang M, Xie J, Ouyang Y, et al. ABO blood type is associated with renal outcomes in patients with IgA nephropathy. *Oncotarget* 2017; **8**: 73603-73612.
- 46 Yoon CY, Chang TI, Kang EW, et al. Clinical usefulness of the Oxford classification in determining immunosuppressive treatment in IgA nephropathy. *Ann Med* 2017; **49**: 217-229.
- 47 Zhang W, Zhou Q, Hong L, et al. Clinical outcomes of IgA nephropathy patients with different proportions of crescents. *Medicine* 2017; **96**: 11.
- 48 Zhang J, Chen C, Zhou Q, et al. Elevated serum fibrinogen level is an independent risk factor for IgA nephropathy. *Oncotarget* 2017; **8**: 99125-99135.
- 49 Zhu X, Li H, 1, Liu Y, et al. Tubular atrophy/interstitial fibrosis scores of Oxford classification combined with proteinuria level at biopsy provides earlier risk prediction in IgA nephropathy. *Sci Rep* 2017; **7**: 1100.
- 50 Cambier A, Rabant M, Peuchmaur M, et al. Immunosuppressive treatment in children with IgA nephropathy and the clinical value of podocytopathic features. *Kidney Internat Rep* 2018; **3**: 916-925.
- 51 Chen D, Liu J, Duan S, et al. Clinicopathological features to predict progression of IgA nephropathy with mild proteinuria. *Kidney Blood Press Res* 2018; **43**: 318-328.
- 52 Deng W, Tan X, Zhou Q, et al. Gender-related differences in clinicopathological characteristics and renal outcomes of Chinese patients with IgA nephropathy. *BMC Nephrol* 2018; **19**: 31.
- 53 Palamuthusingam D, Castledine C, Lawman S. Outcomes of immunosuppression in IgA nephropathy based on the Oxford classification. *Saudi J Kidney Dis Transpl* 2018; **29**:341-350.

- 54 Pan M, Zhang J, You X, et al. Renal outcomes in primary IgA nephropathy patients with segmental glomerular necrosis: a case-control study. *Hum Pathol* 2018; **75**: 47-54.
- 55 Pan M, Zhou XQ, Zheng SB, et al. Serum C3/C4 ratio is a novel predictor of renal prognosis in patients with IgA nephropathy: a retrospective study. *Immunolog Res* 2018; **66**: 381-391.
- 56 Schimpf JJ, Klein T, Fitzner C, et al. Renal outcomes of STOP-IgAN trial patients in relation to baseline histology (MEST-C scores). *BMC Nephrol* 2018; **19**: 328.
- 57 Zhang X, Shi S, Ouyang Y, et al. A validation study of crescents in predicting ESRD in patients with IgA nephropathy. *J Transl Med* 2018; **16**:115.
- 58 Barbour SJ, Coppo R, Zhang H, et al. Evaluating a new international risk-prediction tool in IgA nephropathy. *JAMA Intern Med* 2019; **179**: 942-952.
- 59 Duan SW, Mei Y, Liu J, et al. Predictive capabilities of three widely used pathology classification systems and a simplified classification (Beijing classification) in primary IgA nephropathy. *Kidney Blood Press Res* 2019; **44**: 928-941.
- 60 Kamiyama T, Moriyama T, Kumon S, et al. The beneficial effects of renin-angiotensin system inhibitors (RASi) on IgA nephropathy with tubulointerstitial lesions categorized by Oxford classification. *Clin Exp Nephrol* 2019; **23**:834-840.
- 61 Park S, Baek CH, Park SK, et al. Clinical significance of crescent formation in IgA nephropathy - a multicenter validation study. *Kidney Blood Press Res* 2019; **44**: 22-32.
- 62 Peng W, Tang Y, Tan L, Qin W. Crescents and global glomerulosclerosis in Chinese IgA nephropathy patients: a five-year follow-up. *Kidney Blood Press Res* 2019; **44**: 103-112.
- 63 Champtiaux N, Liote F, El Karoui K, et al. Spondyloarthritis-associated IgA nephropathy. *Kidney Internat Rep* 2020; **5**: 813-820.

- 64 Coppo R, D'Arrigo G, Tripepi G, et al. Is there long-term value of pathology scoring in immunoglobulin A nephropathy? A validation study of the Oxford classification for IgA nephropathy (VALIGA) update. *Nephrol Dial Transplant* 2020; **35**: 1002-1009.
- 65 Jebali H, Ghabi H, Mami I, et al. Prognostic value of the Oxford classification and the Oxford score in IgA nephropathy: a Tunisian study. *Saudi J Kidney Dis Transpl* 2020; **31**:1366-1375.
- 66 Jullien P, Laurent B, Berthoux F, et al. Repeat renal biopsy improves the Oxford classification-based prediction of immunoglobulin A nephropathy outcome. *Nephrol Dial Transplant* 2020; **35**: 1179-1186.
- 67 Moriyama T, Karasawa K, Miyabe Y, et al. Validation of the revised Oxford classification for IgA nephropathy considering treatment with corticosteroids/immunosuppressors. *Sci Reports* 2020; **10**: 11151.
- 68 Neves PDMM, Pinheiro RBB, Dias CB, et al. Renal outcomes in Brazilian patients with immunoglobulin A nephropathy and cellular crescentic lesions. *Kidney Blood Press Res* 2020; **45**: 431-441.
- 69 Neves PDMM, Souza RA, Torres FM, et al. Evidences of histologic thrombotic microangiopathy and the impact in renal outcomes of patients with IgA nephropathy. *PLoS ONE* 2020; **15**: e0233199.
- 70 Park S, Kim HW, Park JK, et al. Relationship between complement deposition and the Oxford classification score and their combined effects on renal outcome in immunoglobulin A nephropathy. *Nephrol Dial Transplant* 2020; **35**: 2130-2137.
- 71 Wu H, Xia Z, Gao C, et al. The correlation analysis between the Oxford classification of Chinese IgA nephropathy children and renal outcome - a retrospective cohort study. *BMC Nephrol* 2020; **21**: 247.

- 72 Aratani S, Matsunobu T, Shimizu A, et al. Tonsillectomy combined with steroid pulse therapy prevents the progression of chronic kidney disease in patients with immunoglobulin A (IgA) nephropathy in a single Japanese institution. *Cureus* 2021; **13**: e15736.
- 73 Bobart SA, Alexander MP, Shawwa K, et al. The association of microhematuria with mesangial hypercellularity, endocapillary hypercellularity, crescent score and renal outcomes in immunoglobulin A nephropathy. *Nephrol Dial Transplant* 2021; **36**: 840-847.
- 74 Canney M, Barbour SJ, Zheng Y, et al. Quantifying duration of proteinuria remission and association with clinical outcome in IgA nephropathy. *J Am Soc Nephrol* 2021; **32**: 436-447.
- 75 Hwang D, Choi K, Cho NJ, et al. Validation of an international prediction model including the Oxford classification in Korean patients with IgA nephropathy. *Nephrology* 2021; 1-9.
- 76 Lin Z, Liu L, Zhang R, et al. Volume of crescents affects prognosis of IgA nephropathy in patients without obvious chronic renal pathology. *Am J Nephrol* 2021; **52**: 507-518.
- 77 Moreno JL, Rodas LM, Draibe J, et al. Extracapillary proliferation scoring correlates with renal outcome and contributes to stratification in adult patients with immunoglobulin A nephropathy. *Clin Kidney J* 2021; **14**: 284-290.
- 78 Schena FP, Anelli VW, Trotta J, et al. Development and testing of an artificial intelligence tool for predicting end-stage kidney disease in patients with immunoglobulin A nephropathy. *Kidney Internat* 2021; **99**: 1179-1188.
- 79 Tang X, Wen Q, Zhou Q, Chen W. Clinicopathological characteristics and prognosis of patients with IgA nephropathy and renal vasculitic lesions. *BMC Nephrol* 2021; **22**: 353.

- 80 Woo KT, Chan CM, Foo M, et al. Evolution of IgA nephropathy in Singapore over four decades and a comparison of two cohorts from the first and fourth decade. *Clin Nephrol* 2021; **95**: 256-272.
- 81 Worawichawong S, Plumworasawat S, Liwlompaisan W, et al. Distribution pattern of mesangial C4d deposits as predictor of kidney failure in IgA nephropathy. *PLoS ONE* 2021; **16**: e0252638.
- 82 Wu D, Li X, Yao X, et al. Mesangial C3 deposition and serum C3 levels predict renal outcome in IgA nephropathy. *Clin Exp Nephrol* 2021; **25**: 641-651.
- 83 Yang D, Liu H, Peng Y, et al. Clinical implication of the circumferential crescents lesions in immunoglobulin A nephropathy: a single-center study of Han Chinese population. *Hum Pathol* 2021; **118**: 49-59.
- 84 Caliskan Y, Demir E, Karatay E, et al. Oxidative stress and macrophage infiltration in IgA nephropathy. *J Nephrol* 2022; **35**:1101-1111.
- 85 Chen Y, Yang Y, Liang Y, et al. Retrospective analysis of crescent score in clinical prognosis of IgA nephropathy. *Open Med* 2022; **17**: 205-215.
- 86 Joh K, Nakazato T, Hashiguchi A, et al. Structural modeling for Oxford histological classifications of immunoglobulin A nephropathy. *PLoS ONE* 2022; **17**: e0268731.
- 87 Kang D, Ban TH, Chin HJ, et al. Prognostic value of chronicity grading on renal outcomes in patients with IgA nephropathy. *Front Med* 2022; **9**:952050.
- 88 Lv J, Wong MG, Hladunewich MA, et al. Effect of oral methylprednisolone on decline in kidney function or kidney failure in patients with IgA nephropathy. *JAMA* 2022; **327**: 1888-1898.
- 89 Ruan Y, Hong F, Wu J, et al. Clinicopathological characteristics, risk factors and renal outcome in IgA nephropathy with crescents. *J Nephrol* 2022; **35**:1113-1121.

90 Rui YF, Yang ZJ, Zhai ZH, et al. The predictive value of Oxford MEST-C classification to immunosuppressive therapy of IgA nephropathy. *Internat Urol Nephrol* 2022; **54**: 959-967.

91 Tang X, Wen Q, Zhou Q, et al. Prognostic significance of the extent of tubulointerstitial lesions in patients with IgA nephropathy. *Internat Urol Nephrol* 2022; <https://doi.org/10.1007/s11255-022-03286-2>.

92 Xie M, Zhu Y, Wang X, et al. Predictive prognostic value of glomerular C3 deposition in IgA nephropathy. *J Nephrol* 2022; <https://doi.org/10.1007/s40620-022-01363-4>.

Systematic Review of the Oxford Classification of IgA Nephropathy: Reproducibility, Prognostic Value, and Immunosuppressive Guidance

Supplemental Table 2

Studies combining clinical factors with Oxford scores in multivariate analysis of outcome:

end point: end stage renal disease:

plus summary of counts

reference in Supplemental Table 1	number of patients: proportion at end point	clinical factors related to end point in multivariate study	Oxford elements studied	Oxford elements related to end point in multivariate study
<b>4</b>	702: 12%	eGFR, p'u, i'supp	MEST1T2	ST1T2
<b>4</b>	702: 12%	eGFR, p'u, i'supp	MEST1T2C	CT1T2
<b>4</b>	416: Oxford criteria: not said	eGFR, p'u, i'supp	MEST+/-C	T
<b>5</b>	294*: 7%	not said	MESTC	ST
<b>13</b>	35*: 57%	eGFR, i'supp, other	MEST	T
<b>18</b>	698: 10%	eGFR, p'u	MEST1T2	MST1T2
<b>20</b>	283: 20%	eGFR, p'u, other	MEST1T2	T2
<b>24</b>	857*: not said	eGFR, p'u, other	MEST	none
<b>26</b>	26*: 26%	none	MEST	none
<b>30</b>	147: 38%	eGFR, p'u	MESTC	E
<b>31</b>	61*: not said	eGFR	MEST	none
<b>36, 80</b>	102: 14% 5y, 39% 20y, 47% 30y	at 5y, p'u, other; at 10y, cr, BP; at 20y and 30y, none	MESTC	at 5y, T; at 10y, EST; at 20y, T; at 30y, M
<b>43</b>	112: 19%	eGFR, p'u, other	MEST	T
<b>44</b>	150: 33%	eGFR, other	MEST	M
<b>45</b>	919: 13%	eGFR, i'supp, other	MEST	T
<b>53</b>	45: not said	none	MESTC	T

56	70: 11%	not said	MESTC	T
57	1152: 13%	not said	MESTC1C2	not C; others not said
60	44: 59%	other	MESTC	none
65	50: 20%	none	MESTC	T
66	168: second biopsy: 22%	eGFR, p'u: worse after i'supp	MESTC	T
67	871: 13%	eGFR, p'u, MAP	MESTC	T
67	445 no i'supp after biopsy: not said	eGFR, p'u, other	MESTC	none
67	426 i'supp after biopsy: not said	eGFR, p'u	MESTC	T
69	118: 31%	other	MESTC	T
73	72*: 13%	eGFR, p'u, BP	MESTC	T
78	948: 22%	cr, p'u, i'supp, other	MEST1T2C	ST1T2
80	177: 19%	cr, BP, other	MESTC	ST
84	47: 36%	eGFR	MESTC	E
87	4151: 7%	eGFR, other	MSTC	ST

**Summary:** number of analyses 34, analyses with C 22; Oxford scores associated with outcome (end stage renal disease) 44 = M 3, E 3, S 7, T 24, C 1, none 6.

**Abbreviations:** BP, hypertension; C, (fibro)cellular crescents; cr, serum creatinine concentration; E, endothelial hypercellularity; eGFR, estimated glomerular filtration rate; i'supp, immunosuppression; M, mesangial cellularity; MAP, mean arterial pressure; p'u, proteinuria; S, segmental sclerosis/capsular adhesion; T, tubular atrophy/interstitial fibrosis: \* number followed

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**Supplemental Table 3**

**Studies combining clinical factors with Oxford scores in multivariate analysis of outcome:**

**end point: end stage renal disease combined with 50% decline in estimated glomerular filtration rate (“combined”), or end stage renal disease with**

**another end point:**

**plus summary of counts**

reference in Supplemental Table 1	number of patients: proportion at end point	end point with ESRD	clinical factors related to end point	Oxford elements studied	Oxford elements related to end point
<b>1</b>	265: 22%	combined	eGFR, p'u, MAP	MESTC	MT
<b>2</b>	183: 20%	doubled cr	eGFR	MEST	none
<b>3</b>	121*: 34%	doubled cr	eGFR	MEST	M
<b>6</b>	54: 19%	combined	not said	MEST	T
<b>7</b>	121*: 33%	doubled cr	eGFR, other	MEST	none
<b>9</b>	74*: 18%	combined	not said	MESTC	none
<b>11</b>	218: 11%	combined	not said	MESTC	T
<b>12</b>	69: 23%	combined	not said	MEST	ET
<b>15</b>	1026: 15%	combined	not said	MESTC	MT
<b>19</b>	1147: 16%	combined	not said	MESTC	ST
<b>21</b>	74: 27%	combined	eGFR	MEST	T
<b>25</b>	500: 10%	doubled cr	eGFR, p'u	MEST1T2	T1T2
<b>27</b>	60 on first	30% eGFR	not said	MESTC	MT

	biopsy: 25%	decline			
<b>27</b>	60 on second biopsy: 25%	30% eGFR decline	not said	MESTC	T
<b>28</b>	901: 18%	combined	eGFR, p'u, MAP	MEST1T2	MT1T2
<b>29</b>	111: 37%	combined	eGFR, other	MEST	none
<b>32</b>	186*: 14%	combined	eGFR	MESTC	not C; others not said
<b>34</b>	627: 10%	combined	p'u	MEST1T2	ST2
<b>35</b>	121: 28%	doubled cr	eGFR	MESTC	C without, SC with i'supp in analysis
<b>37</b>	261 under 23y: 9%	combined	not said	MESTC	MST
<b>37</b>	174 under 18y: not said	combined	not said	MESTC	S
<b>39</b>	3096: 13%	combined	eGFR, fup p'u, fup MAP	MESTC	MSTC
<b>39</b>	1950 no i'supp: not said	combined	eGFR, fup p'u	MESTC	MTC
<b>39</b>	1146 i'supp: not said	combined	fup p'u, fup MAP	MESTC	T
<b>41</b>	123*: 19%	combined	none	MESC: all biopsies T0	none
<b>42</b>	262: 11%	40% eGFR decline	i'supp	MEST	none
<b>47</b>	538: 13%	doubled cr	eGFR, p'u, BP	MESTC	MSC
<b>48</b>	672: 13%	combined	other	MEST1T2C	T2
<b>49</b>	742: not said	combined	p'u	MEST	T
<b>52</b>	988: 11%	doubled cr	eGFR, p'u	MESTC	S
<b>54</b>	365: 10%	combined	eGFR, i'supp, other	MESTC	none
<b>55</b>	712: 12%	combined	cr, BP, other	MEST1T2C	T1T2
<b>58</b>	2781: 18%	combined	eGFR, p'u	MEST1T2	MT1T2
<b>59</b>	412: 17%	combined	p'u, MAP	MEST1T2C	T2
<b>61</b>	3380: 21%	combined	eGFR, p'u, other	MESTC1C2	SC1C2
<b>62</b>	1328: 17%	combined	cr, p'u, BP, i'supp	MESTC1C2	ETC2

64	1130: 25%	combined	eGFR, p'u, MAP	MESTC	MST
64	582 no i'supp: not said	combined	eGFR, p'u, MAP	MESTC	MT
68	101*: 30%	doubled cr	not said	MESTC	STC
71	1243: 14%	combined	not said	MESTC	ST
74	1864: 15%	combined	eGFR, other	MEST1T2C	T1T2
75	545: 10%	combined	eGFR, p'u, other	MEST1T2C	MT1T2
77	89: not said	combined	none	MEST1T2C1C2	T2C2
79	501: not said	doubled cr	eGFR, p'u, other	MEST	S
81	101: 26%	combined	p'u, other	MEST1T2C	T1T2
82	98: 6%	combined	other	MESTC	none
85	144: 12%	combined	eGFR, MAP	MESTC	STC
88	503:36%	40% eGFR decline or death from renal failure	i'supp	MEST	none
89	458: 12%	combined	eGFR	MESTC	none
90	101: 22%	combined	eGFR	MEST1T2C	MT2
91	988: not said	doubled cr	not said	MEST	T
92	821: 18%	combined	cr, other	MST1T2C	T2

**Summary:** number of analyses 53, analyses with C 37; Oxford scores associated with outcome (end stage renal disease combined with something else) 85 = M 14, E 2, S 14, T 34, C 10, none 11.

**Abbreviations:** BP, systemic hypertension; C, (fibro)cellular crescents; cr, serum creatinine concentration; E, endothelial hypercellularity; eGFR, estimated glomerular filtration rate; ESRD, end stage renal disease; fup, time-averaged values at follow-up; i'supp, immunosuppression; M, mesangial cellularity; MAP, mean arterial pressure; p'u, proteinuria; S, segmental sclerosis/capsular adhesion; T, tubular atrophy/interstitial fibrosis; y, years of age: \* number followed

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Supplemental Table 4

Studies combining clinical factors with Oxford scores in multivariate analysis of outcome:

outcome: something other than end stage renal disease, alone or combined:

plus summary of counts

reference in Supplemental Table 1	number of patients: proportion at end point	end point or other measure	clinical factors related to end point or other measure	Oxford elements studied	Oxford elements related to end point or other measure
<b>1</b>	265	rate eGFR decline	eGFR, p'u, MAP	MESTC	ST
<b>3</b>	121*	rate eGFR decline	eGFR	MEST	T
<b>7</b>	121*	rate eGFR decline	eGFR, other	MEST	S
<b>8</b>	141: 4%	50% cr increase	none	MEST	S
<b>8</b>	141: 38%	remission	p'u	MEST	M
<b>10</b>	43: 37%	50% cr increase	eGFR, other	MESTC	M
<b>14</b>	161: 4%	eGFR under 60	p'u	MESTC	MT
<b>15</b>	1026	rate eGFR decline	not said	MESTC	MT
<b>16</b>	34*: not said	50% p'u decline	other	MEST	none

17	41: 51%	remission	eGFR, other	MEST	T
19	1147	rate eGFR decline	not said	MESTC	MT
22	70: not said	50% eGFR decline	not said	MEST	none
23	430: 14%	50% eGFR decline	eGFR, p'u	MEST1T2C	T1T2
23	430	rate eGFR decline	not said	MESTC	ST
30	147: 29%	rapid eGFR decline	p'u	MEST1T2C	ET2
33	105: 27%	30% eGFR decline	BP, other	MEST	none
34	627	rate eGFR decline	not said	MEST1T2	ST2
37	261	rate eGFR decline	not said	MESTC	none
38	54: 19%	50% eGFR decline	p'u	MESTC	E
40	86: 15%	50% cr increase	none	MESTC	T
41	123*: 29%	remission	none	MESC: all biopsies T0	none
46	377: 13%	persistent p'u	not said	MEST	M
46	377: 14%	30% eGFR decline	not said	MEST	M
50	77*: 12%	10% eGFR decline	not said	MESTC	S without, none with i'supp in analysis
51	506: 11%	50% cr increase	p'u, other	MESTC	none
53	45: not said	50% cr	none	MESTC	none

		increase			
56	70: 33%	decline eGFR >15	not said	MEST	none
56	70: 11%	remission	not said	MEST	none
63	27*: 69%	cr doubled or eGFR <30	eGFR	MESTC	none
64	1130	rate eGFR decline	eGFR, p'u, MAP	MESTC	T
64	582 no i'supp	rate eGFR decline	p'u	MESTC	TC
70	453: 11%	30% eGFR decline or p'u increase	not said	MESTC	MST
72	149: not said	progression CKD stage	p'u, i'supp [with tonsillex]	MESTC	none
76	305: 11%	decline eGFR 15% in 1y or 30% in 2y	MAP, i'supp, other	MESTC	none
83	384: 12%	30% eGFR decline	eGFR, BP, i'supp [nonsteroid]	MESTC	only circumfer- ential crescents
86	946	rate eGFR decline	eGFR, p'u, steroids	MESTC	T
90	101: not said	remission	eGFR	MESTC	MS

**Summary:** total number of analyses 38, analyses with C 25; Oxford scores associated with outcome (something other than end stage renal disease, alone or combined) 49 = M 9, E 2, S 8, T 15, C 2, none 13. **Remission or decline in proteinuria** number of analyses 6, analyses with C 2; Oxford scores associated with outcome 7 = M 2, E 0, S 1, T 2, C 0, none 2. **Rate eGFR decline (including rapid decline)** number of analyses 12, analyses with C 9; Oxford scores associated

with outcome 19 = M 2, E 1, S 4, T 10, C 1, none 1. **Various others** number of analyses 20, analyses with C 14; Oxford scores associated with outcome 23 = M 5, E 1, S 3, T 3, C 1, none 10.

**Abbreviations:** C, (fibro)cellular crescents; cr, serum creatinine concentration; E, endothelial hypercellularity; eGFR, estimated glomerular filtration rate, in ml/min per 1.73m<sup>2</sup> if a number is given; i'supp, immunosuppression; M, mesangial cellularity; MAP, mean arterial pressure; mGFR, measured glomerular filtration rate; p'u, proteinuria; RF, renal function; S, segmental sclerosis/capsular adhesion; T, tubular atrophy/interstitial fibrosis; tonsilleX, tonsillectomy:

\* number followed

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**Supplemental Table 5**

**References relevant to immunosuppression (n = 89), other than controlled trials (n = 3 [42, 56, 88])**

Reference numbers are those in Supplemental Table 1

**Immunosuppression before biopsy**

Specified treatment 9/89 [1, 3, 7, 9, 15, 19, 40, 58, 75]

Specified no treatment 14/89 [10, 12, 30, 33, 36, 45, 48, 51, 55, 60, 65, 72, 73, 92]

Not said 66/89

**Immunosuppression after biopsy**

No patients 6/89 (five studies) [3, 7, 8, 30, 41, 60]

All patients 4/89 [17, 27, 31, 90]

Between 0% and 100% of patients 79/89, although 13 only said that some were treated [16, 18, 22, 36, 44, 47, 49, 73, 80, 81, 85, 87, 89], and 10 gave ambiguous figures [20, 23, 48, 52, 55, 59, 69, 79, 83, 91]

**Significant relation between at least one Oxford feature and use of immunosuppression after biopsy n=28**

E, C [1], M, S, T [2], M, C [5], C [9], C [11], E, T [12], E, C [15], M, E, S, T [19], C [23], E [25], M [29], E, T, C [39], M, E, C [50], C [53], C2 [57], C [58], E, C [59], C2 [61], C [62], T [65], M, E, S, C [67], C [68], E [72], C [76], C2 [77], “renal vasculitic lesions” including C [79], E, S, C [86], C [89]

Totals C 21, E 11, M 6, T 5, S 4

**No significant relation between at least one Oxford feature and use of immunosuppression after biopsy n=4 [13, 35, 38, 49]**

**Immunosuppression as variate in multivariate analyses of outcome**

Immunosuppression included as a variate in outcome analysis in 25/79 papers that treated between none and all patients after biopsy. Reference numbers given with Oxford elements associated with outcome, and outcome:

Did not say whether immunosuppression had a relation to outcome 7/25 [5 ST, ESRD; 9 none, ESRD+; 46, two outcomes, M, other; 49 T, ESRD+; 50 none, other; 57 not C, ESRD; 70 MST, other]

Significantly improved outcomes with immunosuppression 10/25 [4, four analyses, STC, ESRD; 13 T, ESRD; 45 T, ESRD; 54 none, ESRD+; 62 ETC, ESRD+; 72 none, other; 76 none, other; 78 ST, ESRD; 83 C, other; 86 T, rate decline]

No improvement with immunosuppression 7/25 [35 SC, ESRD+; 51 none, other; 53, two outcomes, T or none, ESRD or other; 58 MT, ESRD+; 65 T, ESRD; 74 T, ESRD+; 92 T, ESRD+]

Worse outcome with immunosuppression 1/25 [66 T, ESRD]

The six papers that used no immunosuppression after biopsy showed relations between various outcomes and M, E, S, and T in four [3 M or T, ESRD+ or rate decline; 7 S or none, ESRD+ or rate decline; 8 M or S, remission or rate decline; 30 E or ET, ESRD or rate decline], or none of MEST or MESTC in two [41, ESRD+ or remission; 60, ESRD]. The four papers that treated all patients after biopsy showed a relation between various outcomes and M, S, and T in three [17 T, remission; 27 MT, ESRD+; 90 MT or MS, ESRD+ or remission], or none of MEST in one [31, ESRD].