changes by increasing physical activity and limiting calories (1200 kcal), and patients from Group 1 were additionally treated with orlistat 120 mg 3 times a day. WOMAC and EQ-5D scores and anthropometric data were regularly obtained during the study; biochemistry panel (lipid profile, glucose, CRP), leptin and interleukin-6 were assessed at baseline and at Mo 6.

Results: Metabolic syndrome (MS) was diagnosed at baseline in 41 patients. Following orlistat therapy pts from Group 1 managed to reduce their body weight by 10,07% ($p<0,05$; Fig 1), and waist circumference (WC) by 7,5% ($p<0,05$), improving the clinical course of OA: total WOMAC score was reduced by 55% ($p<0,05$; Fig 2), and EQ-5D quality of life was improved by 44% ($p<0,05$). Patients from Group 2 lost only 0,88% of their body weight versus the baseline, and reduced WC by 1%, without changes in EQ-5D scores. MS lab parameters – lipid profile and glucose – did not change significantly after orlistat treatment in Group 1. Patients from Group 2 showed significant increase in total cholesterol ($p<0,05$) after 6 months of dieting and exercising, revealing direct correlation between raised LDL and triglycerides levels and total WOMAC scores. CRP dropped by 23,74% ($p>0,05$) in Group 1. More pronounced decrease of CRP was observed in pts losing more than 10% of their body weight ($p=0,07$). Statistically significant decrease in leptin and IL-6 levels was documented in pts with orlistat-induced significant weight loss ($p<0,05$).

Abstract SAT0558 – Figure 1. Body mass reduction (kg, $p<0,05$).

Abstract SAT0558 – Figure 2. Dynamics of total WOMAC score ($p<0,05$).

Conclusions: Abdominal obesity is considered to be the major component of MS and key risk factor for the development and progression of knee OA. Our study demonstrates that induced by orlistat, diet and exercise weight loss by >10% improves the clinical course of KOA and patient's quality of life, reduces activity of inflammation and improves key parameters of metabolic syndrome. Therefore, KOA and MS therapeutic strategies should include pharmacotherapy of obesity in patients who fail to lose weight with the diet and physical exercise only.

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SAT0559

SYSTEMATIC LITERATURE REVIEW (SLR) FOR THE 2018 UPDATE OF THE EULAR MANAGEMENT RECOMMENDATIONS FOR HAND OSTEOARTHRITIS (OA)

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Background: As part of the update of the EULAR management recommendations an SLR of available evidence for hand OA treatment was performed.

Objectives: To inform the task force of an overview of available evidence for the efficacy and safety of all non-pharmacological, pharmacological and surgical interventions for hand OA.

Methods: We searched Medline, EMBASE, Cochrane Central, and 2016–2017 conference abstracts until June 2017 for (randomised) controlled trials or Cochrane reviews. Observational studies with a comparator were included to assess safety, or effects of surgical interventions. Main efficacy outcomes were pain, function, and hand strength. Risk of bias was assessed. Meta-analysis was performed if possible.

Results: Of 7036 records, 127 references were included, of which 50 studies concerned non-pharmacological, 64 concerned pharmacological, and 12 concerned surgical interventions. Most trials were published after 2007. Many studies were at high risk of bias, mainly due to inadequate randomisation or lack of blinding. Overall, effect sizes of effective therapies were small. Beneficial non-pharmacological treatments for pain relief include hand exercise, and prolonged splinting of the thumb base (Table). A single trial showed that assistive devices had no effect on pain, but led to functional improvements (mean difference (MD) -0.3, 95% confidence interval (CI) -0.6;0.01 on AUSCAN function, range 1–5). Topical and oral non-steroidal anti-inflammatory drugs (NSAIDs) proved equally effective for pain relief (Table), while topical NSAIDs led to less adverse events (e.g., risk ratio for withdrawals due to adverse events 0.15, 95% CI 0.03;0.63). Single trials demonstrated beneficial effects for other pharmacological interventions, including chondroitin sulphate, and intra-articular (i.a.) glucocorticoid injections in interphalangeal joints (Table). Compared to placebo, no beneficial effects were demonstrated for i.a. glucocorticoid or hyaluronic acid injections in the thumb base, hydroxychloroquine, or tumour necrosis factor inhibitors (Table). In two trials, structure modification was the primary outcome measure, though no disease-modifying properties were found in these studies. In a Cochrane review no surgical intervention for thumb base OA appeared more effective than another, although in general, more complex procedures led to more complications.

Abstract SAT0559 – Table 1. Effect on pain of main non-surgical interventions for hand OA from controlled trials.

Intervention	Control	N patients (studies)	Duration	OA type or location	Rob	Difference?	Effect estimate [95% CI]
Non-pharmacological interventions							
Hand exercise	No exercise	381 (5)	12 w	-	High	Intervention better	SMD -0.27 [-0.47; -0.07]
Joint protection	No joint protection	257 (1)	26 w	-	High	No	MD -0.79 [-1.7; 0.12] on AUSCAN pain (range 0-20)
Thumb splint	Usual care or no intervention	221 (4)	4-6 w	CMC	High	No	MD -2.9 [-12.2; 6.3] on 100 mm VAS
		137 (2)	13-52 w	CMC	High	Intervention better	MD -17.4 [-25.6; -9.2] on 100 mm VAS
Long thumb splint (MCP + CMC joint)	Short thumb splint (only CMC joint)	185 (3)	2-12 w	CMC	High	No	MD -0.85 [-5.1; 3.4] on 100 mm VAS
DIP splint	No intervention	26 (1)	12 w	DIP	High	No	Median difference -0.5 [range -7.3; 5.3, p-value 0.53] on 10 cm VAS
Assistive devices	Information provision	70 (1)	12 w	-	High	No	MD 0.4 [-9.8; 10.6] on 100 mm VAS
Pharmacological interventions							
Topical NSAID	Topical placebo	385 (1)	8 w	-	Low	Intervention better	MD -5.9 [-11.7; -0.06] on 100mm VAS
Topical NSAID	Oral NSAID	381 (2)	3 w	"activated" IP OA	Low	No	SMD -0.05 [-0.27; 0.17] (1 trial, n=321)
Oral NSAID	Placebo	695 (3)	2-4 w	-	Low	Intervention better	SMD 0.40 [0.20; 0.60] (2 trials, n=654)
Chondroitin sulphate	Placebo	162 (1)	26 w	-	Low	Intervention better	MD -8.7 [-9.0; -8.4] on 100mm VAS
I.a. glucocorticoids	I.a. placebo	206 (3)	26 w	CMC	Low/unclear	No	MD -3.6 [-13.9; 6.8] on 100mm VAS (2 trials, n=166)
I.a. glucocorticoids	I.a. placebo	60 (1)	12 w	IP	Low	Intervention better	MD -18.0 [-35.5; -2.8] on 100mm VAS
I.a. hyaluronic acid	I.a. placebo	235 (3)	26 w	CMC	Unclear	No	MD 3.3 [-5.2; 11.8] on 100mm VAS (1 trial, n=123)
Hydroxychloroquine	Placebo	503 (3)	24-52 w	-	Unclear	No	MD 2.9 [-5.4; 9.2] on 100mm VAS (2 trials, n=307)
TNF inhibitor	Placebo	235 (3)	24-52 w	erosive OA	Low	No	MD 4.9 [-12.2; 3.2] on 100mm VAS (2 trials, n=175)

AUSCAN, Australian/Canadian hand osteoarthritis index; CI, confidence interval; CMC, first carpometacarpal; DIP, distal interphalangeal joint; I.a., intra-articular; MCP, metacarpophalangeal joint; MD, mean difference; mm, millimetre; NSAID, non-steroidal anti-inflammatory drug; OA, osteoarthritis; OR, odds ratio; Rob, risk of bias; RR, risk ratio; SMD, standardized mean difference; TNF, tumor necrosis factor; VAS, visual analogue scale; w, weeks.

Conclusions: The number of trials in hand OA has steeply increased over the last decade, providing evidence for several therapeutic options for symptom relief, though no disease-modifying drugs have yet been found. High-quality trials investigating often used analgesics like paracetamol are needed.

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