OP0057

RESPONSE TO INJECTION CAN PREDICT OUTCOMES OF FEMOROACETABULAR IMPINGEMENT

E. Campos Martins[1], D. Almeida Gomes[2], H. De Brito Fontana[2], D. Araujo Fernandes[1], F. De Souza Neves[3]. [1]*Federal University of Santa Catarina, Department of Surgery, Florianópolis, Brazil*; [2]*Federal University of Santa Catarina, Department of Morphological Sciences, Florianópolis, Brazil*; [3]*Federal University of Santa Catarina, Internal Medicine Department, Florianópolis, Brazil*

Background: Femoroacetabular impingement syndrome (FAIS) is a highly prevalent painful disorder that is considered a risk factor for hip osteoarthritis. In order to relieve pain and improve cartilage preservation, surgery is often performed. However, many operated patients do not show satisfactory outcomes. Reliable diagnostic tests that can inform prognosis of surgery in patients with FAIS are needed for optimized indications and contraindications to surgery.

Objectives: This systematic review aimed to answer the following question: "Can the response to intra-articular anesthetic injections be used to predict surgical outcomes in patients with FAIS?".

Methods: This study was conducted following the Preferred Reporting Items for Systematic reviews and Meta-Analysis (PRISMA) statement. Embase, CINAHL, LILACS, PubMed, SCOPUS, SPORTDiscuss, The Cochrane Library, and Web of Science databases were screened. All studies that assessed the capability of the response to intra-articular anesthetic injections in predicting surgical outcomes for patients with FAIS were considered eligible. Study selection and data collection were performed by three independent reviewers. Risk of bias of the included studies was assessed through the QUIPS tool.

Results: Seventeen articles were selected for full-text reading, of which 6 were considered eligible and included for analysis [1-6]. A summary of the studies' descriptive characteristics can be found in the Table 1. A high risk of bias due to study attrition and the presence of confounding factors was observed for all included studies. Five out of six studies [1-4,6] presented "high risk" of bias associated to the prognostic factor measurement. A high overall risk of bias was evidenced by QUIPS for all studies (Figure 1). From 6 included studies, 5 indicated that the response to intra-articular injections can be useful in the prediction of surgical outcomes [2-6].

Conclusion: Although there seems to be some evidence supporting the use of intra-articular anesthetic injections to predict surgical outcomes in patients with FAIS, it is not conclusive. Future studies taking into account the various possible sources of bias in prognostic studies are needed. Standardizing and optimizing injection protocols as well as post-injection pain assessment and outcomes measurements are also essential to fill this gap.

REFERENCES:

[1] OR Ayeni et al., Knee Surg Sports Traumatol Arthrosc 22 (4), 801 (2014)
[2] N Chinzei et al., Eur J Orthop Surg Traumatol 30 (4), 737 (2020)
[3] G Gao et al., Arthroscopy 37 (1), 128 (2021)
[4] AJ Krych et al., Arthroscopy 32 (8), 1592 (2016)
[5] S Li et al., Orthop Surg 13 (6), 1810 (2021)
[6] T Mujahed et al., Orthop J Sports Med 9 (11), 2021

Disclosure of Interests: None declared.
DOI: 10.1136/annrheumdis-2022-eular.609

Figure 1.

Modern biological treatment in RA

OP0058

CERTOLIZUMAB-PEGOL, ABATACEPT, TOCILIZUMAB OR ACTIVE CONVENTIONAL THERAPY IN EARLY RHEUMATOID ARTHRITIS: CLINICAL AND RADIOGRAPHIC 48-WEEKS RESULTS OF THE INVESTIGATOR-INITIATED RANDOMIZED NORD-STAR TRIAL

M. Østergaard[1], R. Van Vollenhoven[2], A. Rudin[3], M. L. Hetland[4], M. Heiberg[5], D. Nordström[6], M. Nurmohamed[7], B. Gudbjornsson[8], L. Midtbøll Ørnbjerg[1],

Table 1.

Author	Mean Age	N (Female)	Injection Content	Time for pain assessment after injection (Way)	Intervention group (Control)	Follow-up time after surgery (Score used)	Results
Ayeni	? (16-62)	52 (30)	Bupivacaine + prednisolone	Daily for 2 weeks (provocative activities)	42 with any pain relief (10)	6 months (mHHS)	Likelihood Ratio of reaching >70 points (LR) for responders was 1.15; LR for nonresponders was 0.57 (p>0.05)
Chinzei	36.7 ± 14.7	49 (27)	Lidocaine	2 weeks (?)	30 with >50% of pain relief (19)	12 months (mHHS)	Good responders showed better mHHS, pre- (p=0.048) and post-operatively (p=0.026)
Gao	36.5 (16-65)	78 (41)	Lidocaine + betamethasone	10 min and 1 week (routine tasks)	33 with >50% of pain relief (3)	22.8 ± 9.7 months (mHHS)	100% of good responders surpassed the MCID, while only 1/3 of poor responders achieved it
Krych	37.6 ± 14.0	319 (?)	Variable	2 weeks, reporting the relief of the first 24h (?)	70 with >50% of pain relief (26)	11–30 months (HOS and mHHS)	For Tönnis grade 1 patients, good responders had higher HOS-SS (p=0.03)
Li	38.6 ± 14.9	60 (33)	Lidocaine + ropivacaine	30 min (physical tests)	-	12 months (mHHS and iHOT-12)	Correlation between iHOT after injection and iHOT at 12 months after surgery (r=0.784; p<0.01)
Mujahed	? (16-65)	242 (?)	Lidocaine + betamethasone	? (?)	120 with any pain relief (88)	24 months (HOS and mHHS)	Responders had greater improvement (p<0.05) in all metrics, except for MCID at mHHS (p=0.24)

P. Bøyesen[5], I. Olsen[9], K. Lend[10], K. Hørslev-Petersen[11], T. Uhlig[5], T. Sokka-Isler[12], G. Gröndal[8], S. Krabbe[13], J. Lindqvist[14], I. Gjertsson[15], D. Glinatsi[1], M. C. Kapetanovic[16], A. B. Aga[5], F. Faustini[17], P. Parmanne[18], T. Lorenzen[19], G. Cagnotto[20], J. Back[21], O. Hendricks[22], D. Vedder[2], T. Rannio[23], E. Grenholm[24], H. M. Lindegaard[25], M. K. A. Ljosa[26], E. Brodin[27], A. Soderbergh[28], M. Rizk[29], E. Hermansson[30], L. Uhrenholt[31], P. Larsson[32], S. A. Just[33], G. Bakland[34], D. Stevens[35], T. B. Laurberg[36], E. A. Haavardsholm[5], J. Lampa[37], on behalf of NORD-STAR study group. *[1]Rigshospitalet, Glostrup, Center for Rheumatology and Spine Diseases, Glostrup, Denmark*; *[2]Amsterdam University Medical Center, Amsterdam Rheumatology Center, Amsterdam, Netherlands*; *[3]Sahlgrenska University Hospital, Rheumatology Clinic, Gothenburg, Sweden*; *[4]Rigshospitalet, Glostrup, DANBIO and COPECARE, Centre for Rheumatology and Spine Diseases, Glostrup, Denmark*; *[5]Diakonhjemmet Hospital, Dept of Rheumatology, Oslo, Norway*; *[6]Helsinki University Hospital, Division of Internal Medicine and Rheumatology, Helsinki, Finland*; *[7]Amsterdam University Medical Center, Location VUmc, Amsterdam, Netherlands*; *[8]University of Iceland, Dept of Rheumatology, Reykjavik, Iceland*; *[9]Oslo University Hospital, Dept of Research Support for Clinical Trials, Oslo, Norway*; *[10]Amsterdam University Medical Center/Karolinska Institute, Dept of Rheumatology and Amsterdam Rheumatology Center/Center for Molecular Medicine, Amsterdam, Netherlands*; *[11]Kong Chr X's Hospital for Rheumatic Diseases, Dept of Rheumatology, Gråsten, Denmark*; *[12]University of Eastern Finland, Dept of Rheumatology, Jyväskylä, Finland*; *[13]Herlev University Hospital, Dept of Radiology, Herlev, Denmark*; *[14]Karolinska University Hospital, Dept of Medicine, Rheumatology Unit, Center of Molecular Medicine (CMM), Stockholm, Sweden*; *[15]Sahlgrenska University Hospital, Dept of Rheumatology, Gothenburg, Sweden*; *[16]Skåne University Hospital, Dept of Clinical Sciences, Lund, Sweden*; *[17]Karolinska University Hospital, Dept of Medicine, Rheumatology, Stockholm, Sweden*; *[18]Helsinki University Hospital, Division of Rheumatology, Helsinki, Finland*; *[19]Silkeborg University Hospital, Dept of Rheumatology, Silkeborg, Denmark*; *[20]Skåne University Hospital, Dept of Clinical Sciences, Malmö, Sweden*; *[21]Uppsala University Hospital, Dept of Medical Sciences, Uppsala, Sweden*; *[22]Danish Hospital for Rheumatic Diseases, Dept of Rheumatology, Sønderborg, Denmark*; *[23]Finland Central Hospital, Dept of Rheumatology, Jyväskylä, Finland*; *[24]Falun Hospital, Dept of Rheumatology, Falun, Sweden*; *[25]Oden se University Hospital, Rheumatology Research Unit, Odense, Denmark*; *[26]Ålesund Hospital, Dept of Rheumatology, Ålesund, Norway*; *[27]Haukeland University Hospital, Dept of Rheumatology, Haukeland, Norway*; *[28]Örebro University Hospital, Dept of Rheumatology, Örebro, Sweden*; *[29]Värmlands Hospital, Rheumatology Clinic, Västerås, Sweden*; *[30]Linköping University Hospital, Department of Rheumatology, Linköping, Sweden*; *[31]Ålborg University Hospital, Dept of Rheumatology, Ålborg, Denmark*; *[32]Academic specialist center. Stockholm, Center for Rheumatology, Stockholm, Sweden*; *[33]Odense Universitetshospital, Svendborg, Section of Rheumatology, Svendborg, Denmark*; *[34]University Hospital of North Norway, Dept of Rheumatology, North Norway, Norway*; *[35]University Hospital of Trondheim, Dept of Rheumatology, Trondheim, Norway*; *[36]Århus University Hospital, Dept of Rheumatology, Århus, Denmark*; *[37]Karolinska University Hospital, Dept of Rheumatology, Stockholm, Sweden*

Background: The optimal first-line treatment of patients with early rheumatoid arthritis (eRA) is not established.

Objectives: To compare clinical and radiographic outcomes of active conventional therapy (ACT) with each of three biological therapies with different modes of action.

Methods: In this investigator-initiated, randomized, open-label, blinded-assessor study (NCT01491815), patients with treatment-naïve eRA with DAS28>3.2 and RF+/ACPA+/CRP>10mg/L, were randomized 1:1:1:1 to methotrexate combined with: 1) oral prednisolone (tapered quickly; discontinued at w36); or: sulphasalazine, hydroxychloroquine and mandatory intra-articular (IA) glucocorticoid injections in swollen joints (ACT); 2) certolizumab-pegol (CZP); 3) abatacept (ABA) or 4) tocilizumab (TCZ). IA glucocorticoid was allowed in all arms except w20-24 and w44-48. Co-primary outcomes at w48 were CDAI remission (CDAI≤2.8) and change in total van der Heijde-modified Sharp Score from baseline ($\Delta vdHSS_{w0\text{-}w48}$). A combination of Bonferroni and Dunnet's procedure adjusted for multiple testing. The primary endpoints were estimated using logistic regression and analysis of covariance, adjusted for sex, anti-CCP status and country.

Results: 812 patients were randomized. Adjusted CDAI remission rates at w48 were: 59.3% (ABA), 52.3% (CZP), 51.9% (TCZ) and 39.2% (ACT). Compared to ACT, CDAI remission rates were superior for ABA (adjusted difference +20.1%; adjusted p<0.001) and CZP (+13.1%; p=0.021), but not TCZ (+12.7%; p=0.030) (Table 1). Key secondary clinical outcomes were consistently better in biological groups compared to ACT. Adjusted mean $\Delta vdHSS_{w0\text{-}w48}$ was low (Table 1), with no significant differences between drugs.

Table 1.

Baseline characteristics	ACT (n=200)	CZP+MTX (n=203)	ABA+MTX (n=204)	TCZ+MTX (n=188) §
Age (y)	55 (15)	55 (15)	55 (14)	52 (15)
Women, %	139 (70%)	139 (69%)	140 (69%)	129 (69%)
Time from diagnosis to baseline, days	13 (21)	12 (17)	16 (34)	16 (33)
Anti-CCP/RF positive, %	82% / 76%	82% / 73%	83% / 78%	82% / 72%
CDAI	28.7 (12.1)	27.9 (12.4)	28.6 (11.3)	26.6 (11.7)
Total vdHSS (0-448) [median; IQR)	6.3 (8.2) [4; 1 - 8.5]	5.9 (7.6) [3; 1 - 8]	5.8 (9.8) [3; 1 - 6]	4.2 (6.7) [2; 0.5 - 5]
Estimated adjusted outcome (ITT)[1], Primary				
CDAI remission, w48	39.2% (32.5 - 45.9)	52.3% (45.5 - 59.1)	59.3% (52.6 - 66)	51.9% (44.9 - 59.0)
Δ1.9% (44.9 -	0.45 (0.31 to 0.59)	0.47 (0.33 to 0.61)	0.62 (0.48 to 0.76)	0.5 (0.36 to 0.64)
Estimated adjusted treatment difference (ITT)[2], Primary				
CDAI remission, w48	Ref	**13.1% (3.5 to 22.6)***	**20.1% (10.6 to 29.5)***	12.7% (3 to 22.5)
Δ2.7% (3 to 2	Ref	0.02 (-0.17 to 0.22)	0.17 (-0.02 to 0.37)	0.05 (-0.15 to 0.25)
Key secondary				
ACR/EULAR Boolean remission, w48	Ref	14.7% (5.4 to 23.9)	19.4% (10.1 to 28.7)	13% (3.5 to 22.4)
DAS28 remission,w48	Ref	12.9% (3.5 to 22.2)	17.4% (8.2 to 26.6)	14.4% (5 to 23.9)
EULAR good response, w48	Ref	8.2% (-0.6 to 17.1)	11.3% (2.7 to 20)	2.9% (-6.3 to 12.2)
vdHSS progression ≤0.5, w0-w48	Ref	-3.3% (-11.1 to 4.6)	3.5% (-4.7 to 11.8)	-2.2% (-10.3 to 5.9)

Values are mean (SD), if not otherwise indicated. §Finnish patients randomised to TCZ+MTX, but not receiving it due to unavailability, are not included. [1]Values are estimated adjusted marginal means and estimated marginal differences against ACT with 95% CI. ITT: intention to treat population. *Superiority compared with ACT was demonstrated.

No new safety signals were reported. Total numbers of serious adverse events (% patients with ≥1 event) were for ABA 21 (8.3%), CZP 28 (12.4%), TCZ 20 (9.2%) and ACT 23 (10.7%).

Conclusion: Compared with active conventional therapy (csDMARD + glucocorticoids), superiority regarding CDAI remission rates was demonstrated for abatacept and certolizumab-pegol, and not for tocilizumab. Radiographic progression was low and similar between treatments.

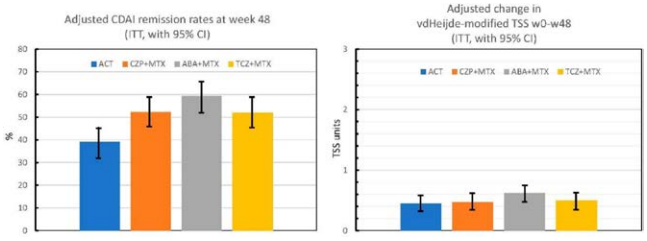

Figure 1.

Acknowledgements: We thank the patients, investigators, nurses, joint assessors and study teams who were involved in the NORD-STAR trial; Eleonore Nilsson, chief study nurse, Lise Hejl Hyldstrup, coordinating study nurse, Niels Steen Krogh, data manager, Monica Rydén Aulin study coordinator and Eva Larsson, patient research partner. We also thank members of the NORD-STAR study group: Anders Bengtsson, Anders Gülfe, Annelies Blanken, Annette Schlemmer, Åsa Reckner Olsson, Aulikki Kononoff, Carl Turesson, Christina Dackhammar, Cidem Gentline, Elisabet Lindqvist, Ellen-Margrethe Hauge, Emma Grenholm, Erik af Klint, Erik Rødevand, Eva Baecklund, Fredrik Markros, Hamed Rezaei, Hanne Merete Lindegaard, Heikki Relas, Heikki Valleala, Ilia Qirjazo, Inger Marie Jensen Hansen, Jarno Rutanen, Jens Kristian Pedersen, Jens Rathmann, Johan Wallman, Johanna Carlestam, Jon Einarsson, Jörgen Lysholm, Kajsa Öberg, Katarina Almehed, Kathrine Lederballe Grøn, Kati Mykkänen, Lena Karlberg, Malin Hemberg, Maria K. Stilling-Vinther, Marjatta Leirisalo-Repo, Mohaned Hameed, Nancy Vivar, Oili Kaipiainen-Seppänen, Peter Olsson, Petrus Linge, Pia Lindell, Pia Neuer Jensen, René Østgård, Riitta Tuompo, Sabine Dieperink, Sara Nysom Christiansen, Sofia Exarchou, Thiab Saleh, Tomas Husmark, Tor Olofsson, Torkell Ellingsen, Trude Bruun, Vappu Rantalaiho and Ylva Borgas.

Disclosure of Interests: Mikkel Østergaard Speakers bureau: AbbVie, BMS, Boehringer-Ingelheim, Eli Lilly, Janssen, Merck, Pfizer, Roche, UCB, Celgene, Sanofi, Regeneron, Novartis, Orion, Hospira, Consultant of: AbbVie, BMS, Boehringer-Ingelheim, Eli Lilly, Janssen, Merck, Pfizer, Roche, UCB, Celgene, Sanofi, Regeneron, Novartis, Orion, Hospira, Grant/research support from: AbbVie, BMS, Merck, UCB, Celgene, Novartis, Ronald van Vollenhoven Speakers bureau: AbbVie, AstraZeneca, Biogen, Celgene, Galapagos, Gilead, Janssen, Pfizer, Servier, UCB, Consultant of: AbbVie, AstraZeneca, Biogen, Celgene, Galapagos, Gilead, Janssen, Pfizer, Servier, UCB, Grant/research support from: BMS, GSK, Eli Lilly, UCB, Pfizer, Roche, Anna Rudin Grant/research support from: AstraZeneca, Merete L. Hetland Speakers bureau: Merck, Biogen, Pfizer, Eli Lilly, Orion Pharma, CellTrion, Samsun Bioepsi, Janssen Biologics BV, MSD, Consultant of: Merck, Biogen, Pfizer, Eli Lilly, Orion Pharma, CellTrion, Samsun Bioepsi, Janssen Biologics BV, MSD, Grant/research support from: BMS, AbbVie, Roche, Novartis, Merck, Biogen, Pfizer, Marte Heiberg Speakers bureau: Eli Lilly, Consultant of: Eli Lilly, Dan Nordström Grant/research support from: UCB, BMS, AbbVie, Celgene, MSD, Novartis, Pfizer, Michael Nurmohamed Speakers bureau: Celltrion, Eli Lilly, Consultant of: Celltrion, Eli Lilly, Grant/research support from: BMS, AbbVie, MSD, Pfizer, Amgen, Björn Gudbjornsson Speakers bureau: Novartis, Consultant of: Novartis, Lykke Midtbøll Ørnbjerg Grant/research support from: Novartis, Pernille Bøyesen: None declared, Inge Olsen: None declared, Kristina Lend: None declared, Kim Hørslev-Petersen: None declared, Till Uhlig Speakers bureau: Grünenthal, Eli Lilly, Novartis, Pfizer, Consultant of: Grünenthal, Eli Lilly, Novartis, Pfizer, Grant/research support from: NORDFORSK, Tuulikki Sokka-Isler Speakers bureau: AbbVie, BMS, Celgene, Medac, Merck, Novartis Orion Pharma, Pfizer, Roche, Sandoz, UCB, Boehringer Ingelheim, Consultant of: AbbVie, BMS, Celgene, Medac, Merck, Novartis Orion Pharma, Pfizer, Roche, Sandoz, UCB, Boehringer Ingelheim, Gerdur Gröndal: None declared, Simon Krabbe Grant/research support from: AbbVie, MSD, Novartis, Joakim Lindqvist: None declared, Inger Gjertsson: None declared, Daniel Glinatsi Speakers bureau: AbbVie, Eli Lilly, Consultant of: AbbVie, Eli Lilly, Meliha C Kapetanovic: None declared, Anna-Birgitte Aga Speakers bureau: AbbVie, Eli Lilly, Novartis, Pfizer, Consultant of: AbbVie, Eli Lilly, Novartis, Pfizer, Francesca Faustini: None declared, Pinja Parmanne Speakers bureau: Novartis, Consultant of: Novartis, Tove Lorenzen Speakers bureau: UCB, Consultant of: UCB, Giovanni Cagnotto: None declared, Johan Back: None declared, Oliver Hendricks Speakers bureau: AbbVie, Novartis, Consultant of: AbbVie, Novartis, Daisy Vedder: None declared, Tuomas Rannio: None declared, Emma Grenholm: None declared, Hanne Merete Lindegaard: None declared, Maud-Kristine A Ljosa: None declared, Eli Brodin: None declared, Annika Soderbergh: None declared, Milad Rizk: None declared, Elsa Hermansson: None declared, Line Uhrenholt Speakers bureau: AbbVie, Eli Lilly, Novartis, Consultant of: AbbVie, Eli Lilly, Novartis, Per Larsson: None declared, Søren Andreas Just: None declared, Gunnstein Bakland Speakers bureau: BMS, Consultant of: BMS, David Stevens Grant/research support from: KLINBEFORSK, Trine Bay Laurberg Speakers bureau: UCB, Consultant of: UCB, Espen A Haavardsholm Speakers bureau: Pfizer, AbbVie, Celgene, Novartis, Janssen, Gilead, Eli Lilly, UCB, Consultant of: Pfizer, AbbVie, Celgene, Novartis, Janssen, Gilead, Eli Lilly, UCB, Grant/research support from: NORDFORSK, Jon Lampa: None declared.
DOI: 10.1136/annrheumdis-2022-eular.868

OP0059

PROFOUND ANTICOAGULANT EFFECTS OF INITIAL ANTIRHEUMATIC TREATMENTS IN EARLY RHEUMATOID ARTHRITIS PATIENTS: A NORD-STAR SPIN-OFF STUDY

B. Dijkshoorn[1], A. Antovic[2], D. Vedder[1], A. Rudin[3], D. Nordström[4], B. Gudbjornsson[5,6], K. Lend[7,8], T. Uhlig[9,10], E. A. Haavardsholm[9], G. Gröndal[5], M. L. Hetland[11,12], M. Heiberg[9], M. Østergaard[11,12], K. Hørslev-Petersen[13,14], J. Lampa[8], R. Van Vollenhoven[7,8], M. Nurmohamed[1,7]. *[1]Amsterdam Rheumatology & immunology center location Reade, Rheumatology, Amsterdam, Netherlands*; *[2]Karolinska Institute, Department of Medicine Solna, Division of Rheumatology, Stockholm, Sweden*; *[3]Sahlgrenska Academy, Gothenburg University, Department of Rheumatology and Inflammation research, Göteborg, Sweden*; *[4]Helsinki University Hospital, Department of Rheumatology, Helsinki, Finland*; *[5]Landspitali University Hospital, Centre for Rheumatology Research, Reykjavik, Iceland*; *[6]University of Iceland, Faculty of Medicine, Reykjavik, Iceland*; *[7]Amsterdam University Medical Centers, Department of Rheumatology and Amsterdam Rheumatology Center, Amsterdam, Netherlands*; *[8]Karolinska Institute, Department of Medicine, Rheumatology Unit, Center for Molecular Medicine (CMM), Stockholm, Sweden*; *[9]Diakonhjemmet Hospital, Department of Rheumatology, Oslo, Norway*; *[10]University of Oslo, Faculty of Medicine, Oslo, Norway*; *[11]Center for Rheumatology and Spine Diseases, Rigshospitalet, Copenhagen Center for Arthritis Research (COPECARE) and DANBIO, Glostrup, Denmark*; *[12]University of Copenhagen, Department of Clinical Medicine, Faculty of Health Sciences, Copenhagen, Denmark*; *[13]University Hospital of Southern Denmark, Danish Hospital for Rheumatic Diseases, Sønderborg, Denmark*; *[14]University of Southern Denmark, Department of Regional Health Research, Odense, Denmark*

Background: Patients with rheumatoid arthritis (RA) are at an increased risk of venous thromboembolism. Thus far, there have not been any comparative studies investigating the effects of initial antirheumatic treatments in (very) early RA patients.
Objectives: To assess the effects of different initial treatments on hemostatic parameters in patients with early RA.
Methods: NORD-STAR is an international, multicentre, open-label, assessor-blinded, phase 4 study where patients with newly diagnosed RA started methotrexate (MTX) and were randomised 1:1:1:1 to a) conventional treatment (either prednisolone tapered to 5mg/day, or sulfasalazine combined with hydroxychloroquine and intra-articular corticosteroids), b) certolizumab pegol, c) abatacept, d) tocilizumab[1]. This study is a spin-off from the main NORD-STAR study extensively investigating hemostatic system in 24 per protocol consecutive Dutch participants at baseline, 12 weeks and 24 weeks after the start of the treatment. Statistical analysis was done using paired samples t-test in SPSS version 28.
Results: The mean age of investigated patients was 51.8 (± 12.7) years and 58.3% were female. At baseline patients had an average DAS28 score of 4.6 (± 0.9) and had elevated levels of investigated coagulation biomarkers: Factor 1+2, fibrinogen, D-dimer and parameters of the two global hemostatic assays, i.e. endogenous thrombin potential (ETP) and overall hemostasis potential (OHP). These biomarkers decreased significantly at 12 and 24 weeks in patients in all groups (Table 1). Overall fibrinolytic potential (OFP) was decreased and clot lysis time (CLT) was prolonged at baseline, demonstrating impaired fibrinolytic activity in early RA. The reduction of coagulation parameters was significantly higher in biological treatment arms in comparison to the standard MTX treatment arm. In addition, tocilizumab was more effective compared to certolizumab and abatacept, (Figure 1), which was expected considering the direct inhibitory effect of this drug on the IL-6 synthesis and consequently the coagulation activation as well. After 24 weeks of treatment with methotrexate and tocilizumab, the average fibrinogen of patients was reduced by 63% vs 31% and 36% in the certolizumab and abatacept groups, respectively. The changes in DAS-28 and the changes in fibrinogen had a correlation of 0.385 which did not reach statistical significance.

Table 1. ***Measurements are marked with * if p<0.05, ** if p<0.01 and *** if p<0.001***

	Baseline	W12	W24
Factor 1+2 (pmol/L)	270.25 (149.4)	190.36 (108.6)**	179.52 (85.3)***
Fibrinogen (g/L)	4.64 (1.5)	3.61 (1.6)**	2.63 (1.2)***
D-dimer (mg/L)	2.17 (3.0)	0.33 (0.23)**	0.29 (0.2)**
OHP (Abs-sum)	157.38 (64.9)	120.62 (68.7)*	100.49 (53.8)***
OCP (Abs-sum)	369.52 (58.8)	305.04 (101.7)*	275.91 (83.1)***
OFP (%)	57.97 (13.1)	63.20 (12.7)*	65.25 (11.4)***
Lag time (s)	304.5 (71.1)	306.8 (71.8)	312.7 (65.4)
Slope	0.07 (0.02)	0.066 (0.03)	0.094 (0.12)
Max Abs	1.17 (0.3)	1.00 (0.4)*	0.91 (0.3)**
CLT (s)	1405 (356)	1317 (377)	1231 (320)**
ETP (nM*min)	1480 (471)	1395 (395)*	1337 (429)*
Peak (nM)	231 (78)	223 (68)	223 (74)
Lagtime (min)	4.06 (2.1)	3.28 (1.2)**	2.87 (1.0)***
ttPeak (min)	7.40 (2.2)	6.61 (1.5)*	6.13 (1.4)**

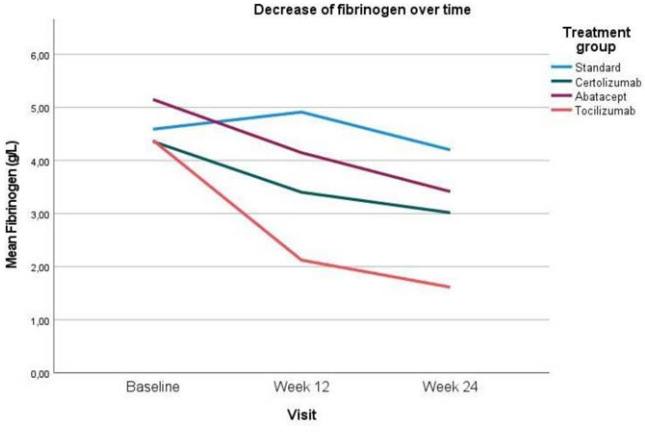

Figure 1.