Amiloidosi, anziano e frailty: chi trattare

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SOCIETÀ ITALIANA
DI GERONTOLOGIA
E GERIATRIA

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UNIVERSITÀ CATTOLICA DEL SACRO CUORE

Amiloidosi cardiaca da transtiretina
una patologia dell'anziano

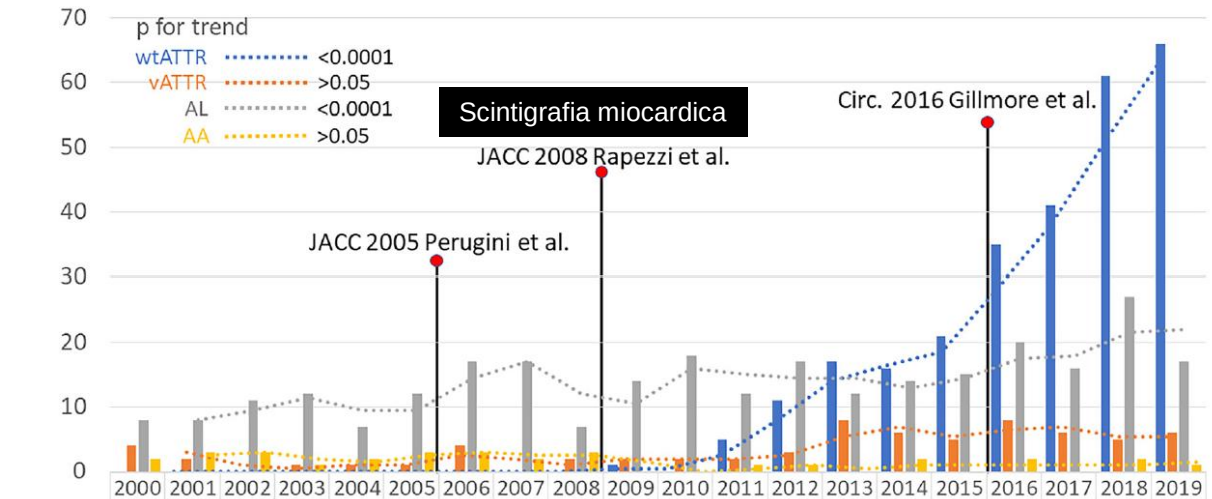
✓ Epidemiologia dell'amiloidosi cardiaca negli ultimi 20 anni

❑ Cardio-Geriatria, fragilità e amiloidosi cardiaca

❑ Amiloidosi Cardiaca: l'incontro tra cardiologia e geriatria

Epidemiologia negli ultimi 20 anni

- **Cardiomiopatia Infiltrativa più frequente >65 anni**
- **Sottodiagnosticata**
- **Ritardo Diagnostico**
- **Prognosi 'tempo dipendente'**



	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019
wtATTR	0	0	0	0	0	0	0	0	0	1	0	5	11	17	16	21	35	41	61	66
vATTR	4	2	0	1	1	1	4	0	2	2	2	2	3	8	6	5	8	6	5	6
AL	8	8	11	12	7	12	17	17	7	14	18	12	17	12	14	15	20	16	27	17
AA	2	3	3	1	2	3	3	2	3	0	0	1	1	0	2	0	2	0	2	1

Zampieri et al. International Journal of Cardiology, 2021

ORIGINAL RESEARCH ARTICLE

Impact of Earlier Diagnosis in Cardiac ATTR Amyloidosis Over the Course of 20 Years

Variable	2002–2006 (n=35)	2007–2011 (n=260)	2012–2016 (n=704)	2017–2021 (n=968)	*P* value
Baseline characteristics					
Age	67.22±6.20*†‡	73.99±7.19§‖	75.81±7.72	76.03±9.01	<0.001
Male sex, n (%)	26 (74.3%)	224 (86.2%)	613 (87.1%)	834 (86.2%)	0.200
Body surface area	…	1.89±0.20	1.89±0.19	1.91±0.20	0.180
Ischemic heart disease	4 (11.4%)	35 (13.5%)	145 (20.6%)	198 (20.5%)	0.035
Diabetes	1 (2.9%)	23 (8.8%)§‖	125 (17.8%)	167 (17.3%)	<0.001
Hypertension	5 (14.3%)	39 (15.0%)§‖	224 (31.8%)¶	396 (40.9%)	<0.001
Stroke/transient ischemic attack	0 (0.0%)	23 (8.8%)	69 (9.8%)	115 (11.9%)	0.067

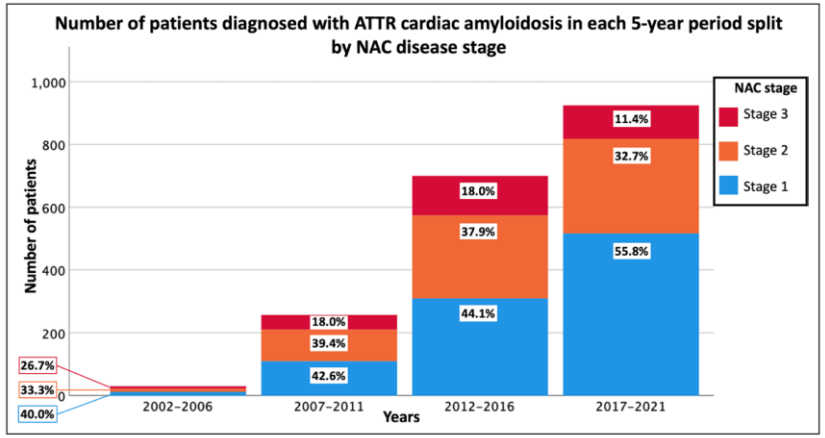


Figure 4. Number of patients diagnosed with transthyretin cardiac amyloidosis between 2002 and 2021, and the proportion of patients with each NAC disease stage for each 5-year period.
NAC indicates National Amyloidosis Centre.

1967 pazienti con ATTR-CA diagnosticati in 20 anni

Negli ultimi 20 anni:
↑ Età alla diagnosi
mediana italiana 80 anni
Comorbosita
↑ Cardiopatia Ischemica
↑ Diabete mellito
↑ Ipertensione
Clinica
↓NYHA alla diagnosi
↓Gravità (NAC score)

Ioannou, A. et al. Circulation 2022

Il ritardo diagnostico e il suo impatto clinico

National Amyloidosis Centre, UK 2002 - 2017
- 711 pazienti diagnosticati con ATTR-CA
- 305 pazienti diagnosticati con V122I-hATTR-CA,
- 118 pazienti diagnosticati con non-V122I-hATTR-CA

ORIGINAL RESEARCH ARTICLE

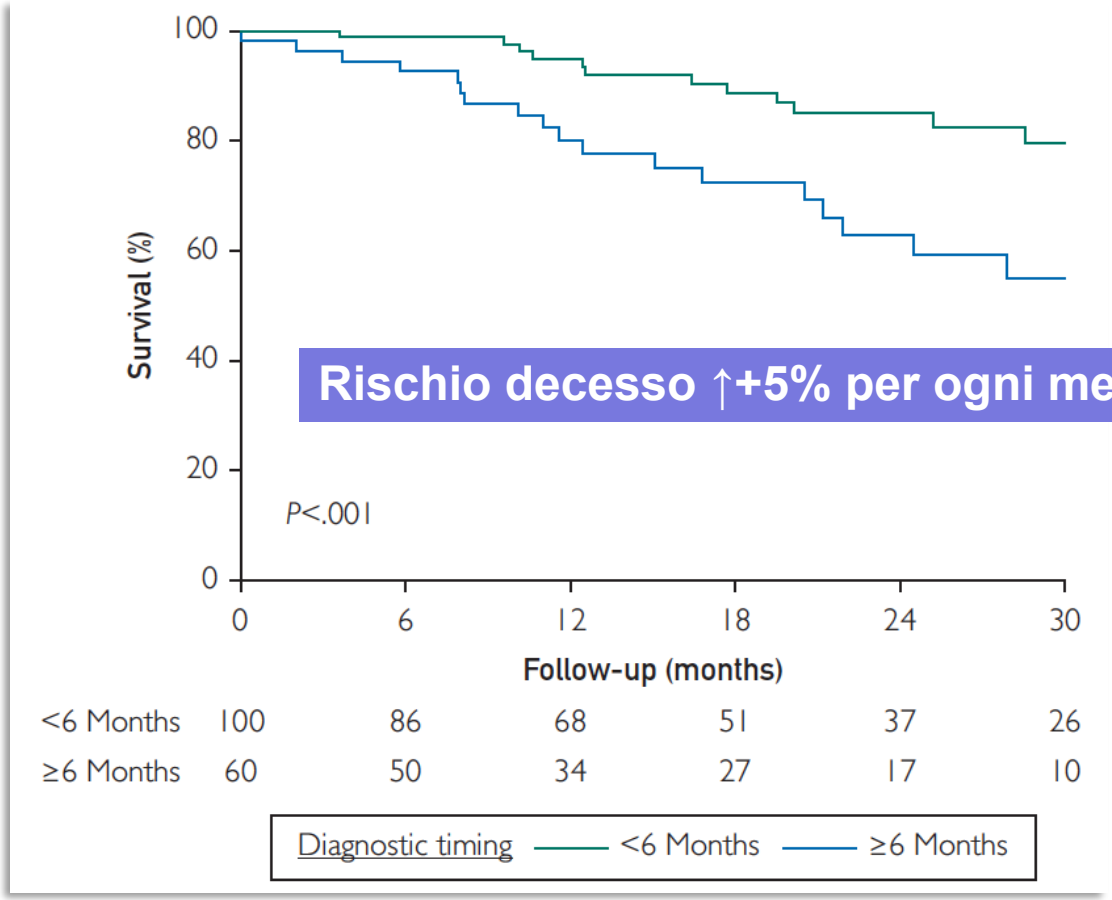
Natural History, Quality of Life, and Outcome in Cardiac Transthyretin Amyloidosis

RESULTS: There was substantial diagnostic delay, with patients using hospital services a median (interquartile range) of 17 (9–27) times during the 3 years before diagnosis, by which time quality of life was poor; diagnosis of wild-type ATTR-CM was delayed >4 years after presentation with cardiac symptoms in 42% of cases. Patients with V122I-hATTR-CM were more impaired functionally ($P<0.001$) and had worse measures of cardiac disease ($P<0.001$) at the time of diagnosis, a greater decline in quality of life, and poorer survival ($P<0.001$) in comparison with the other subgroups.

17 Visite in 3 anni prima della diagnosi

Thirusha L., et al. Circulation 2019

Time is Therapy



Rischio decesso ↑+5% per ogni mese perso

MAYO CLINIC

Early Diagnosis and Outcome in Patients With Wild-Type Transthyretin Cardiac Amyloidosis

Epoca 'Pre-Tafamidis'
N=160 pazienti

Variable	Hazard ratio (95% CI)	*P* value
Diagnostic timing (per month increase)	1.049 (1.017-1.083)	.003
Age at diagnosis (per year increase)	1.078 (1.008-1.153)	.004
Coronary artery disease	4.291 (1.678-10.979)	.002
NYHA class (III/IV vs I/II)	4.240 (1.923-9.348)	<.001
Disease stage[b]		
II vs I	2.928 (1.169-7.335)	.022
III vs I	9.951 (3.043-32.543)	<.001
Ejection fraction (per % increase)	0.971 (0.940-1.003)	.149

Fumagalli C. et al. 2021

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2022: l'anno della Cardio-Geriatria

Journal of the American College of Cardiology 2022

THE PRESENT AND FUTURE

JACC STATE-OF-THE-ART REVIEW

Interventions for Frailty Among Older Adults With Cardiovascular Disease

JACC State-of-the-Art Review

*[…]**Cardiovascular disease and frailty are interdependent and have the same physiological underpinning** that predisposes to the progression of both disease processes*

*[…] Contemporary cardiovascular practice should **actively identify patients with physical frailty** who could benefit from frailty interventions and aim to deliver these therapies in a **patient-centered model to optimize quality of life**, particularly after cardiovascular interventions.*

JACC: Advances 2022

STATE-OF-THE-ART REVIEW

GERIATRIC CARDIOLOGY

Geriatric Cardiology: Coming of Age

*[…]Multimorbidity, polypharmacy, cognitive changes, and **diminished functional capacity, along with changes in the social environment**, result in complexity that makes provision of CVD care to older adults challenging. In this review, we first describe the history of geriatric cardiology, an orientation that acknowledges the unique needs of older adults with CVD […]*

European Journal of Preventive Cardiology , 2022

ESC European Society of Cardiology

European Journal of Preventive Cardiology (2022) 29, 216–227
doi:10.1093/eurjpc/zwaa167

CONSENSUS DOCUMENT
Aging

Frailty in cardiology: definition, assessment and clinical implications for general cardiology. A consensus document of the Council for Cardiology Practice (CCP), Association for

*[…]Frailty increases cardiovascular (CV) morbidity and mortality both in patients with or without known CV disease. **Though the recognition of this additional risk factor has become increasingly clinically relevant in CV diseases, uncertainty remains about operative definitions, screening, assessment, and management of frailty.***

La fragilità nella amiloidosi cardiaca

RESEARCH LETTER

Prevalence and Prognostic Significance of Frailty Among Patients With Transthyretin Amyloidosis Cardiomyopathy

Nowell M. Fine, MD, SM; Jacqueline M. McMillan, MD

- **145** pazienti ATTR-CA dal **2014 al 2019**
- Fragilità studiata tramite la Clinical Frailty Scale (CFS)

Dopo aggiustamento per classe NYHA e NAC score, i terzili di CFS rimanevano associati alla mortalità al follow up (HR1.49 [95% CI, 1.28–1.83], P<0.001).

Parameter	Study population (N=145)	CFS 1–3 (n=46)	CFS 4–6 (n=76)	CFS 7–9 (n=23)	*P* value
Demographic and clinical					
Age, y	78 (73–84)	75 (69–83)	78 (71–85)	86 (81–89)	<0.001
Sex (male)	132 (91)	41 (89)	69 (91)	22 (96)	0.67
Body mass index, kg/m^2	22.9 (19.8–26.1)	24.3 (21.6–27.7)	22.5 (20.6–26.2)	21.1 (18.3–24.1)	0.009
NYHA class III/IV	78 (54)	22 (48)	42 (55)	15 (65)	0.38
Laboratory					
Hemoglobin, g/dl	10.8 (9.3–12.4)	11.5 (10.0–12.9)	10.7 (10.2–12.4)	9.8 (8.5–10.9)	0.011
ATTRwt	136 (94)	42 (91)	72 (95)	22 (96)	0.69
Tafamidis therapy	16 (11)	6 (13)	8 (11)	2 (9)	0.84
ATTR-CM stage*					
Stage I	45 (31)	18 (39)	26 (34)	4 (17)	0.22
Stage II	76 (52)	23 (50)	35 (46)	12 (52)	
Stage III	24 (17)	5 (11)	15 (20)	7 (31)	

Among patients with ATTR-CM, **frailty was highly prevalent and an important risk factor for mortality**. As assessed by the CFS, frailty was independently associated with mortality and **may be a valuable component of routine clinical assessment**.

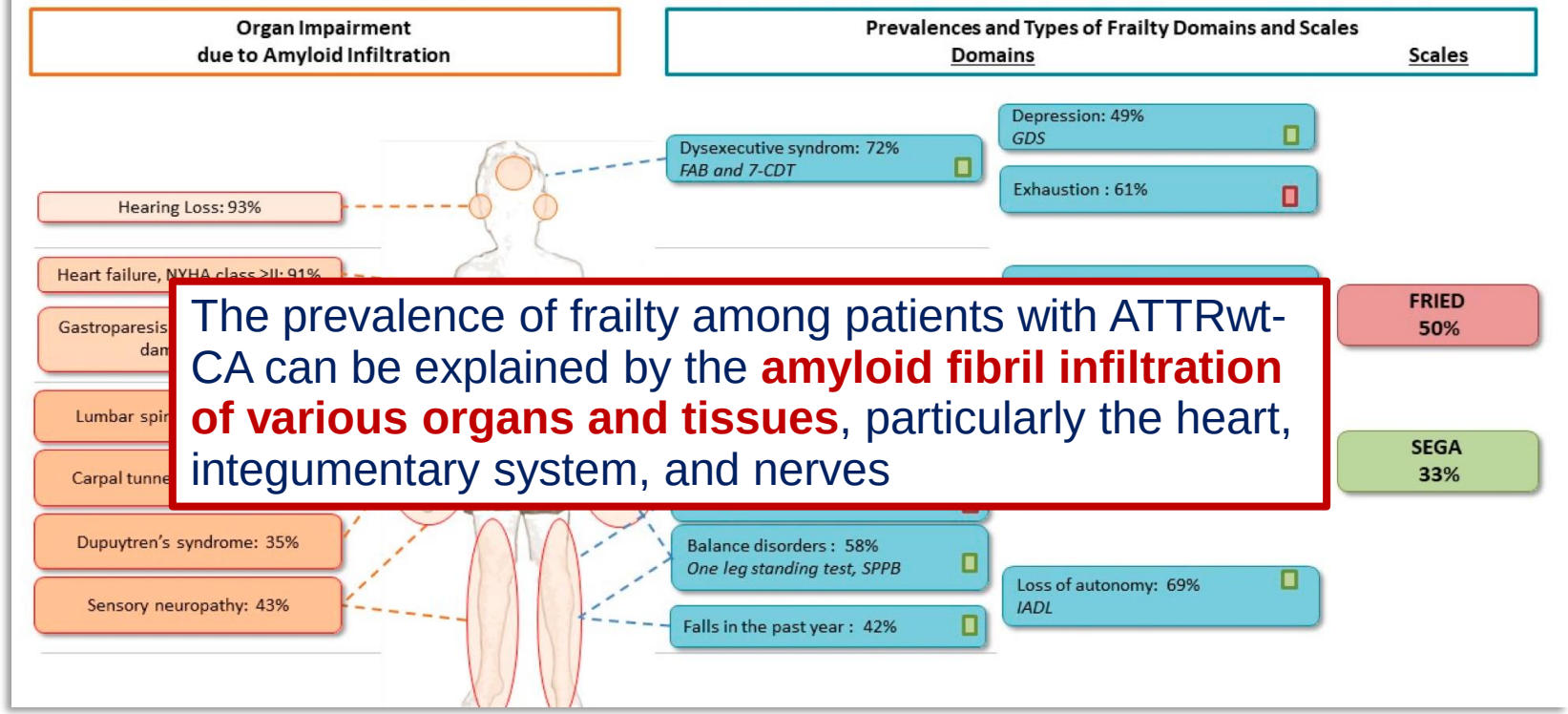
Journal of Clinical Medicine
MDPI

Article

Frailty in Wild-Type Transthyretin Cardiac Amyloidosis: The Tip of the Iceberg

Amaury Broussier [1,2,*], Jean Philippe David [1,3], Mounira Kharoubi [4], Silvia Oghina [4], Lauriane Segaux [1,5], Emmanuel Teiger [4], Marie Laurent [1,3], Isabelle Fromentin [2], Sylvie Bastuji-Garin [1,5,6,†] and Thibaud Damy [1,4,†]

- 36 pazienti ATTR-CA dal 2018 al 2019
- Fragilità studiata secondo il modello di Fried e *Short Emergency Geriatric Assessment* (SEGA)



The prevalence of frailty among patients with ATTRwt-CA can be explained by the **amyloid fibril infiltration of various organs and tissues**, particularly the heart, integumentary system, and nerves

Broussier A., et al 2022

La fragilità nella amiloidosi cardiaca
Progetto Pilota Firenze - Roma

- **Design**: studio multicentrico prospettico di prevalenza della della fragilità nei pazienti affetti da ATTR-CA
- Pazienti valutati tramite valutazione multidimensionale: **MPI, CFS, SPPB e GDS**
- **115 pazienti** ATTR-CA nel 2022

UNIVERSITÀ DEGLI STUDI FIRENZE
FLORENCE CARDIO MYOPATHIES
aouc
SAPIENZA UNIVERSITÀ DI ROMA

Fragilità nel paziente con amiloidosi cardiaca

Valutazione multidimensionale nei pazienti con amiloidosi cardiaca

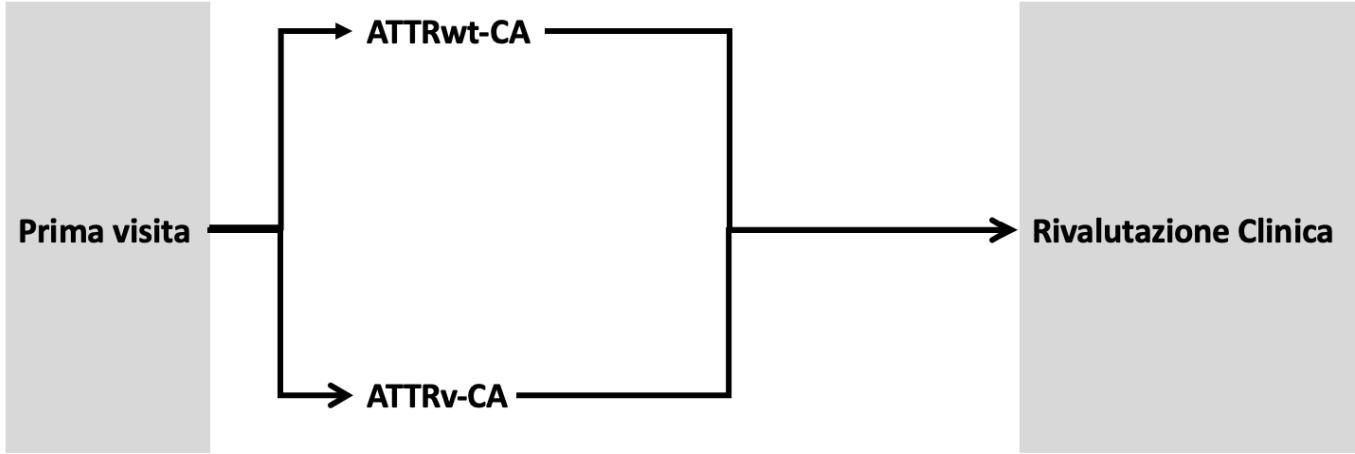
Criteri di inclusione

Diagnosi confermata di amiloidosi cardiaca da transtiretina (TTR-CA) sia wild type (wt) che variante (v)

Criteri di esclusione

-grave decadimento cognitivo

- grave disabilità



T0

Visita Cardiologica
Valutazione Genetica
Visita Multidimensionale
Registrazione Prelievo ematico
Valutazione RMN cardiaca

T1 (~6/12/24 mesi)

Visita Cardiologica
Registrazione Prelievo ematico
Anamnesi di accesso PS/DEAS
Ricovero presso reparti di Medicina/Cardiologia

Tabella. Caratteristiche generali della popolazione di Firenze (N=115)	
Età alla valutazione	81 [77-85]
Femmine	9 (8.1%)
ADL	5 [4-6]
IADL	5 [2-7]
Mini Nutritional Assessment	11 [8-13]
<=8 (malnutriti)	26 (22.7%)
Geriatric Depression Scale	4 [2-7]
Clinical Frailty Scale	4 [3-6]
>4	40 (34.7%)
Short Physical Performance Battery (SPPB)	7 [2-10]
SPPB<=6	36 (31.3%)
MPI Risk score	0.32 [0.19-0.43]
lieve <=0.33	73 (63.5%)
moderato 0.34-0.66	32 (27.8%)
grave >=0.67	10 (8.7%)

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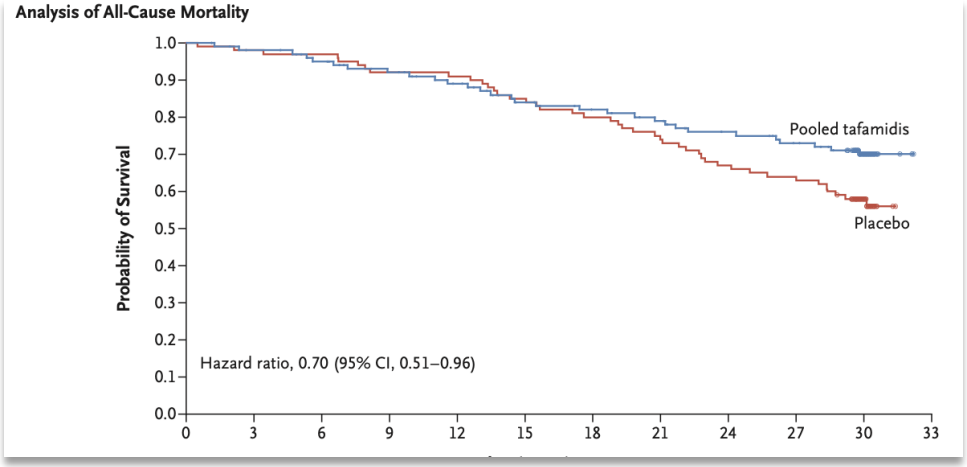
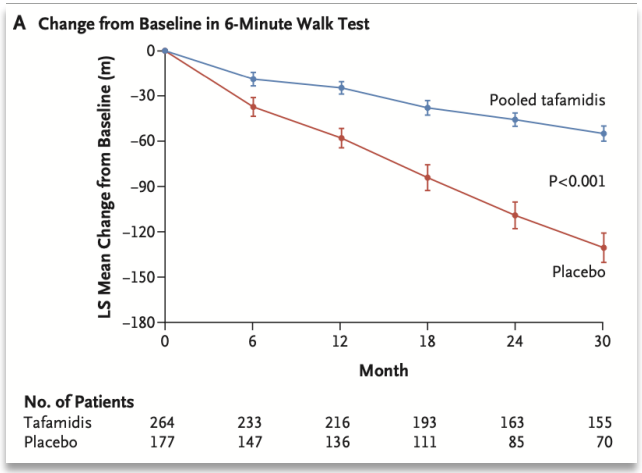
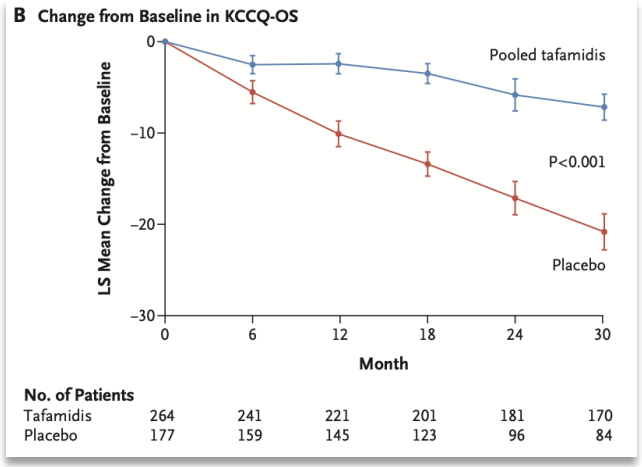
Tafamidis Treatment for Patients with Transthyretin Amyloid Cardiomyopathy







Survival

6MWT

Quality of life (KCCQ)

The NEW ENGLAND JOURNAL of MEDICINE

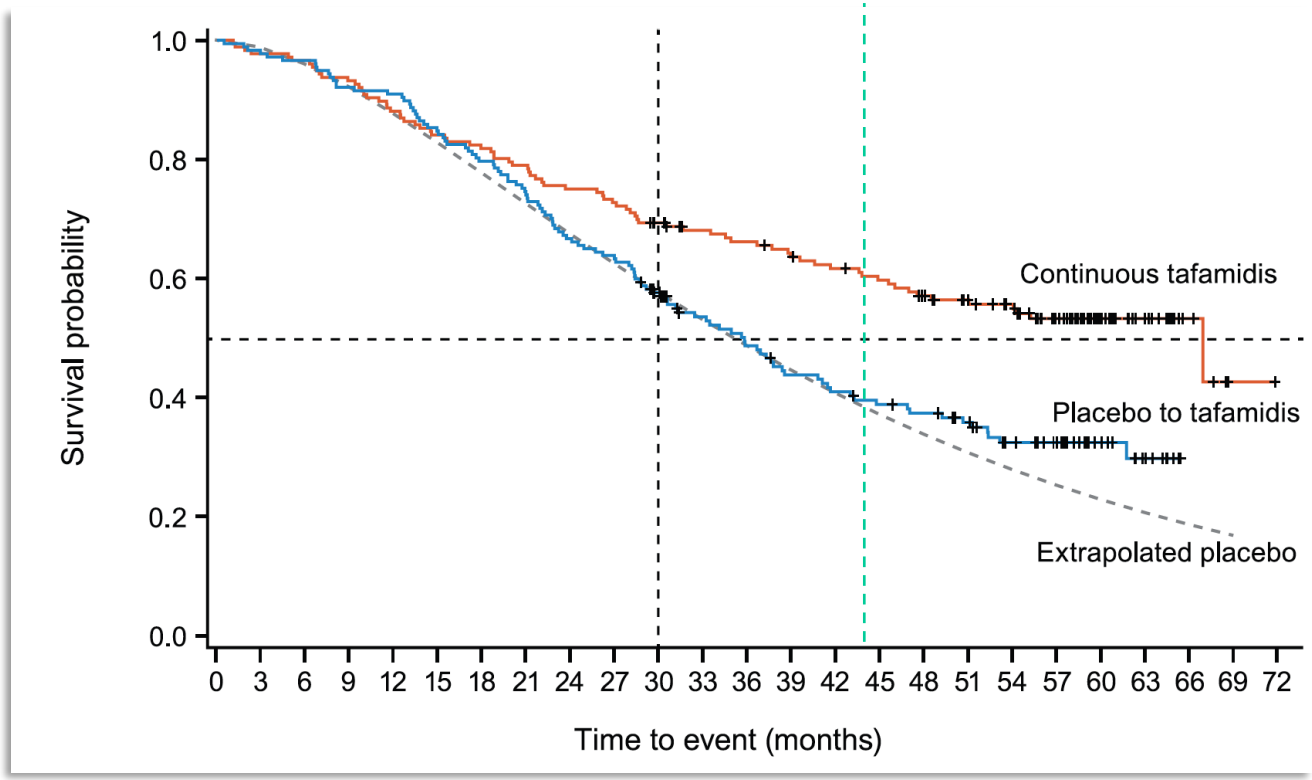
Circulation: Heart Failure

ORIGINAL ARTICLE

Long-Term Survival With Tafamidis in Patients With Transthyretin Amyloid Cardiomyopathy

Perry Elliott, MD; Brian M. Drachman, MD; Stephen S. Gottlieb, MD; James E. Hoffman, MD; Scott L. Hummel, MD; Daniel J. Lenihan, MD; Ben Ebede, MS, MBA; Balarama Gundapaneni, MS; Benjamin Li, MS; Marla B. Sultan, MD, MBA; Sanjiv J. Shah, MD

While the reduction in mortality was similar in patients with ATTRwt and patients with ATTRv (≈40% in each), there was a greater reduction in patients with NYHA class I or II (44%) than NYHA class III (35%). Together with results from ATTR-ACT,[13,20] these data support the use of tafamidis in all patients with ATTR-CM but emphasize the importance of early diagnosis and treatment.



Current Approaches to the Treatment of hATTR Amyloidosis

TTR Suppression[12,16-19]

Patisiran

Molecule
Double-stranded siRNA encapsulated in a lipid nanoparticle; targets the liver

Mechanism
Knocks down TTR mRNA

Results from the APOLLO trial

Δ 34.0	Δ 21.1
mNIS+7	Norfolk QOL-DN Score

Safety
Mild-to-moderate AEs in most patients; most common were peripheral edema or infusion-related reactions

FDA approved

Inotersen

Molecule
Antisense oligonucleotide

Mechanism
Knocks down TTR protein production

Results from the NEURO-TTR trial

Δ 19.7	Δ 11.7
mNIS+7	Norfolk QOL-DN Score

Safety
A few patients developed thrombocytopenia and glomerulonephritis; no similar events with enhanced monitoring

FDA approved

Orthotopic Liver Transplantation[12-15]

Rationale
Majority of TTR is produced by the liver

OS
75% at 5 years for patients with Val30Met amyloidosis

Limitations
- Availability of organ for transplant
- Mutation-specific outcomes
- Prior organ impairment not reversed
- Disease progression even after transplant
- Long-term post–orthotopic liver transplantation immunosuppressive therapy required

TTR Stabilization[12,20-22]

Tafamidis

Molecule
1,3-benzoxazole

Mechanism
Binds to TTR tetramer, preventing dissociation; for ATTR cardiomyopathy

Results from the ATTR-ACT trial
Lower
All-cause mortality
Rate of cardiovascular hospitalizations
Rate of decline in timed 6-m walk test
Rate of decline in HF scoring system

Safety
Well tolerated; similar AE incidences and types to patients treated with placebo

FDA approved

Diflunisal

Molecule
Generically available NSAID

Mechanism
Binds to TTR tetramer, preventing dissociation; off-label for hATTR polyneuropathy

Results from the trial

Δ 8.7	Δ 1.5	Δ 3.7	29.7%
mNIS+7	SF-36 Physical	SF-36 Mental	<2-pt mNIS+7 ↑

Safety
A few patients developed thrombocytopenia and glomerulonephritis; no similar events with enhanced monitoring

Under investigation

Study Design: Patisiran Phase 3 APOLLO-B Study

Randomized, Double-Blind, Placebo-Controlled Study in Patients with ATTR Amyloidosis with Cardiomyopathy

Patient population, N=360

- ATTR amyloidosis; wt or any TTR mutation
- Confirmed cardiomyopathy and medical history of symptomatic heart failure
- NYHA ≤III; minimum walk and NT-proBNP limits at baseline
- ≤30% on background tafamidis at baseline[a]

1:1 RANDOMIZATION

Patisiran 0.3 mg/kg IV Q3W[b]

or

Placebo IV Q3W[b]

Stratification:
Baseline tafamidis (yes or no); hATTR vs wtATTR amyloidosis; NYHA Class I/II and age <75 years vs all others

Patisiran vs Placebo

Primary endpoint

- Change in 6-MWT at Month 12

Secondary endpoints

- Cardiomyopathy symptoms and health status (KCCQ-OS) at Month 12
- Death and hospitalization outcomes over 12 months[c]

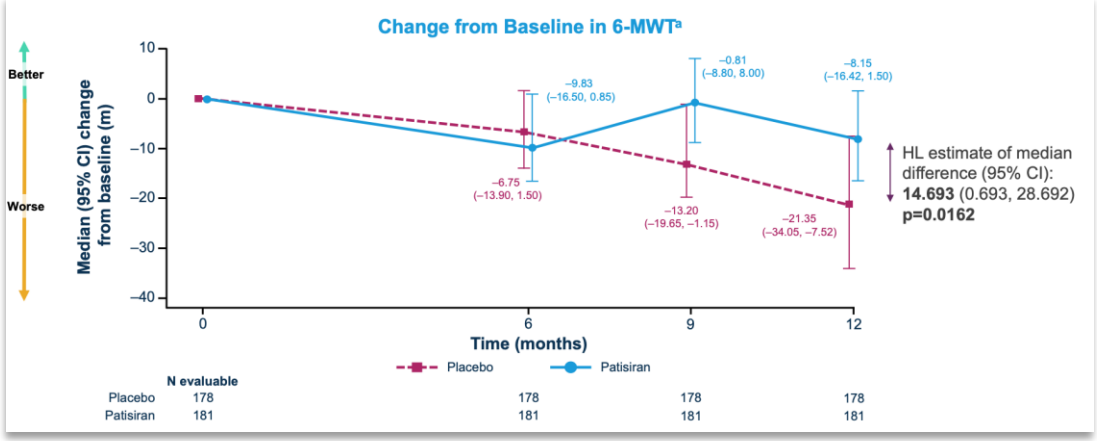
Selected exploratory endpoints

- E.g., Cardiac biomarkers (NT-proBNP, Troponin I), imaging

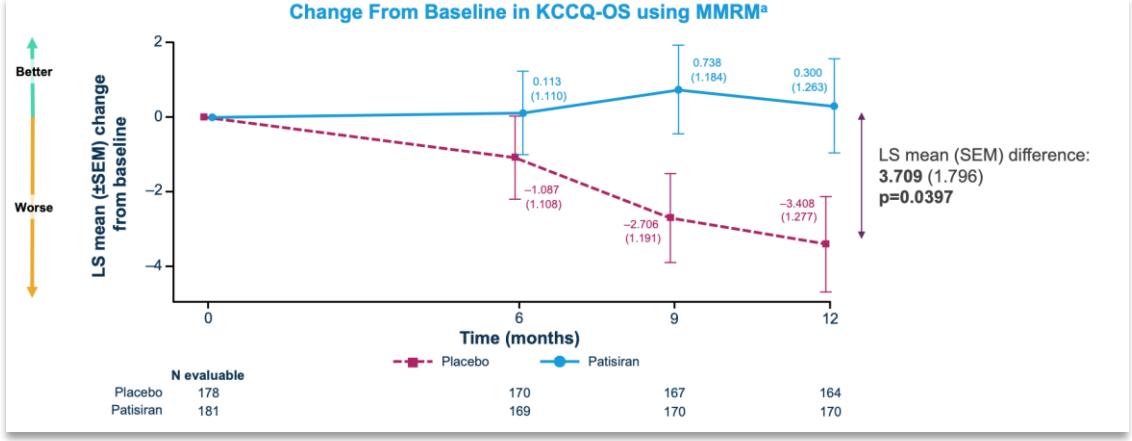
Open-label extension

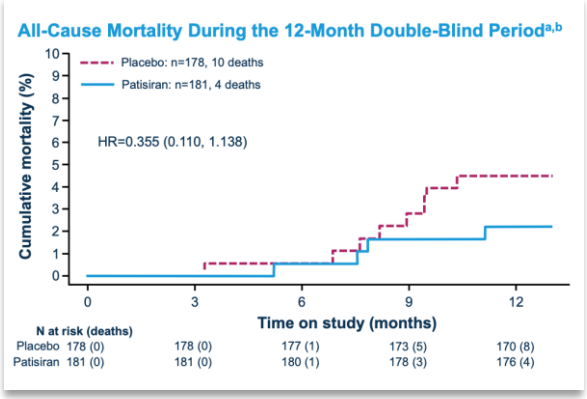
Maurer et al, Heidelberg 4-8 Sept. 2022

Primary Endpoint: 6MWT at 12 months



Secondary Endpoints: KCCQ and Death + Hosp





ISA INTERNATIONAL SOCIETY OF AMYLOIDOSIS

Maurer et al, Heidelberg 4-8 Sept. 2022

SOCIETÀ ITALIANA DI GERONTOLOGIA E GERIATRIA

67° CONGRESSO NAZIONALE SIGG

LA LONGEVITÀ DECLINATA AL FEMMINILE

Marianna FONTANA

There is a need for further treatments for ATTR-CM

- ATTR-CM, a chronic condition associated with progressive heart failure, results from extracellular deposition of misfolded TTR protein as amyloid fibrils in the myocardium (**Figure 1**).[1]
- Disease-modifying treatments are needed for patients with ATTR-CM, for whom treatment is often limited to managing symptoms and best supportive care.
  - Currently, there is only one approved disease-modifying treatment for ATTR-CM, which acts by stabilizing TTR and slowing disease progression.[2]

Figure 1. ATTR-CM pathogenesis and treatment approaches

Suppression of TTR synthesis
Gene silencing, gene editing and liver transplantation target the liver, where native TTR is produced, and reduce circulating levels of TTR

Tetramer stabilization
Tetramer stabilizers bind to tetrameric TTR and stabilize the tetrameric form

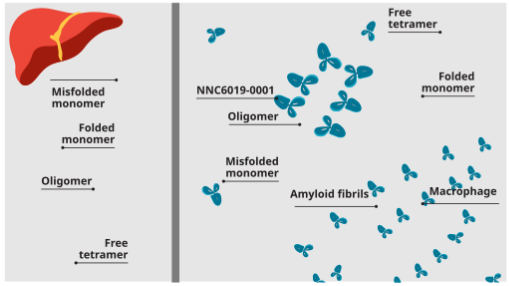
Amyloid depletion
- Amyloid-depleting agents, such as NNC6019-0001, are designed to deplete existing amyloid deposits and prevent the formation of new amyloid fibrils
- There are currently no approved amyloid-depleting therapies

Best supportive care
Management and treatments for heart failure, arrhythmias and cardiac comorbidities

Depletion of amyloid deposits is a novel and promising treatment approach

- NNC6019-0001 is a humanized monoclonal antibody designed to bind specifically to a unique epitope exposed on misfolded monomeric and aggregated forms of TTR; it does not bind to normal tetrameric TTR (**Figure 2**).[3]
  - Binding inhibits the formation of new TTR amyloid fibrils and induces the clearance of existing fibrils through antibody-mediated phagocytosis.[3]
- In a phase 1 trial in 21 patients with hereditary ATTR, NNC6019-0001 was well tolerated at all doses tested (up to and including 30 mg/kg).[4]

Figure 2. Mechanism of action of NNC6019-0001



The liver produces TTR, which circulates as free tetramers, folded monomers, misfolded monomers and oligomers (left). The antibody does not bind to native tetramers, but can bind to misfolded monomeric and aggregated forms of TTR (right).

NNC6019-0001, a humanized monoclonal antibody, in patients with transthyretin amyloid cardiomyopathy (ATTR-CM): rationale and study design of a phase 2, randomized, placebo-controlled trial

Marianna Fontana[1]; Kristine Buchholtz[2]; Mads D M Engelmann[2]; Martha Grogan[3]; Gerard Kornelis Hovingh[2]; Arnt V Kristen[4]; Pernille Poulsen[2]; Sanjiv J Shah[5]; Mathew S Maurer[6]

[1]National Amyloidosis Centre, University College London, Royal Free Campus, London, UK; [2]Novo Nordisk A/S, Søborg, Denmark; [3]Mayo Clinic, Rochester, MN, USA; [4]Department of Cardiology, Amyloidosis Center, Heidelberg University, Heidelberg, Germany; [5]Division of Cardiology, Department of Medicine, Northwestern University Feinberg School of Medicine, Chicago, IL, USA; [6]Cardiac Amyloidosis Program, Department of Medicine, Columbia University Irving Medical Center, New York Presbyterian Hospital, New York, NY, USA

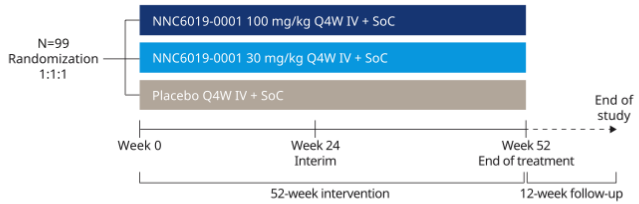
Inclusion criteria (N=99)

- Hereditary or wild-type ATTR
- NYHA class II or III
- LVWT ≥12 mm
- 6MWT ≥150 m and <450 m
- NT-proBNP ≥650 pg/mL in sinus rhythm and >1000 pg/mL in atrial fibrillation

Sentinel dosing phase

Three patients per arm will receive the study drug or placebo, in combination with 24-hour inpatient cardiac monitoring and 7 days of continuous cardiac (tele-) monitoring.

Trial design



NNC6019-0001 will be administered intravenously in 250 mL sodium chloride 0.9% over 2–3 hours.
Patients will be stratified by disease type (hereditary vs wild-type ATTR).

Trial start: mid-2022
Patient recruitment: Canada, Czech Republic, France, Germany, Italy, Japan, Netherlands, Portugal, Spain, UK, USA

Primary endpoints

Changes from baseline to week 52 in
- 6MWT
- NT-proBNP

Secondary endpoints

- ECV on cardiac MRI
- GLS
- QoL assessed via KCCQ
- Troponin I
- Hospitalization due to CV events
- Urgent visits due to heart failure
- All-cause mortality
- Pharmacokinetics
- Treatment-emergent adverse events

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Abbreviations
6MWT, 6-minute walk test; ATTR, transthyretin amyloidosis; ATTR-CM, transthyretin amyloid cardiomyopathy; CV, cardiovascular; ECV, extracellular volume; eGFR, estimated glomerular filtration rate; GLS, global longitudinal strain; IV, intravenous; KCCQ, Kansas City Cardiomyopathy Questionnaire; LVWT, left ventricle wall thickening; MRI, magnetic resonance imaging; NT-proBNP, N-terminal pro-B-type natriuretic peptide; NYHA, New York Heart Association; Q4W, every 4 weeks; QoL, quality of life; SoC, standard of care; TTR, transthyretin.

References: (**1**) Rubin J, Maurer MS. Annu Rev Med 2020;71(1):203–19; (**2**) Maurer MS et al. N Engl J Med 2018;379(11):1007–16; (**3**) Higaki JN et al. Amyloid 2016;23(2):86–97; (**4**) Suhr O. Neurology 2021;96:e2783–8. Available from https://n.neurology.org/content/96/22/e2783.abstract. Accessed 8 June 2022.

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Quali pazienti ***dobbiamo*** trattare?
Quali pazienti ***possiamo*** trattare?

Consensus SIGG per la definizione e lo studio della fragilità nei pazienti affetto da amiloidosi cardiaca da transtiretina

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Amiloidosi Cardiaca e Fragilità

SEGNI E SINTOMI CARDIOLOGICI (cardiopalmo, dispnea, sincope, edemi declivi)

SEGNI E SINTOMI NEUROLOGICI (parestesie, tunnel carpale, rottura tendine bicipite, stenosi canale lombare, ipotensione ortostatica)

SEGNI E SINTOMI SISTEMICI (astenia, malessere, perdita di peso, scarsa tolleranza allo sforzo

↓

Sospetto coinvolgimento cardiologico (ECG, ecocardiogramma, RM Cardiaca)

↓

Scintigrafia miocardica con tracciante per osso + screening componente monoclonale

↓

Terapia?

1

Età
Classe NYHA
Test del Cammino

2

3

Fragilità
Disabilità

Amiloidosi Cardiaca e Fragilità

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1 1

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Classe NYHA
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Fragilità
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2 2

Terapia?

Amiloidosi, anziano e frailty: chi trattare

Grazie per la vostra attenzione

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DI GERONTOLOGIA
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