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ORIGINAL ARTICLES

REVIEW ARTICLES

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ORIGINAL ARTICLE

Clinical profile of geriatric patients in the week before death: a cross-sectional study in a second-level hospital in Mexico

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Abstract

Background: *The final stage of life is characterized by progressive clinical deterioration, the timely recognition of which is essential to guide clinical decisions focused on the patient's quality of life. Evidence in Mexico is limited and influenced by sociodemographic, intercultural, normative, and structural factors, compared to the international literature.* **Objective:** *To describe the clinical profile of the patient, with emphasis on the frequency and progression of the main signs and symptoms in the final stage of life.* **Material and methods:** *A prospective cross-sectional observational study was conducted. Patients aged ≥ 60 years were included between December 2023 and October 2024, who met the completion criteria established in current national regulations. Patients with brain death and invasive or non-invasive mechanical ventilation were excluded. The sample was calculated using a formula for finite populations (n = 65). Signs and symptoms were recorded by daily observation. A descriptive analysis was performed with percentage estimation and 95% confidence intervals (CI).* **Results:** *65 patients were included (mean age 77.1). The most frequent signs during the week prior to death were reduced intake (100%; 95% CI: 94.5-100), loss of functionality (96.9%; 95% CI: 89.3-99.6), edema (55.4%; 95% CI: 42.3-67.9), dyspnea (46.2%; 95% CI: 33.7-59.1) and pain (40.0%; 95% CI: 28.0-53.1).* **Conclusion:** *The correct identification of the clinical profile before death allows strengthening timely palliative decision-making, promoting care focused on the patient's dignity and quality of life.*

Keywords: *Palliative care. End-of-life stage. Terminal symptoms.*

Perfil clínico del paciente geriátrico en la semana previa a la defunción: estudio transversal en un hospital de segundo nivel en México

Resumen

Antecedentes: *La etapa final de la vida se caracteriza por un deterioro clínico progresivo, cuyo reconocimiento oportuno es esencial para guiar las decisiones clínicas centradas en la calidad de vida del paciente. La evidencia en México es limitada e influenciada por factores sociodemográficos, interculturales, normativos y estructurales, en comparación con la literatura internacional.* **Objetivo:** *describir el perfil clínico del paciente, con énfasis en la frecuencia y progresión de los principales signos y síntomas en la etapa final de la vida.* **Material y métodos:** *Estudio observacional prospectivo transversal. Se incluyeron pacientes de ≥ 60 años, entre diciembre 2023 y octubre 2024, que cumplieran criterios de terminalidad establecidos en la normativa nacional vigente. Se excluyeron a pacientes con muerte cerebral y con ventilación mecánica invasiva o no invasiva. La muestra se calculó utilizando una fórmula para poblaciones finitas (n = 65). Los signos y síntomas se registraron mediante observación diaria. Se realizó un análisis descriptivo con estimación de porcentajes e intervalos de confianza (IC) del 95%.* **Resultados:** *65 pacientes fueron incluidos (edad media 77,1). Los signos más frecuentes durante la semana previa a la muerte fueron: reducción de la ingesta (100%; IC 95%: 94,5-100), pérdida de funcionalidad (96,9%; IC 95%: 89,3-99,6), edema (55,4%; IC 95%: 42,3-67,9), disnea (46,2%; IC 95%: 33,7-59,1) y dolor (40,0%; IC 95%: 28,0-53,1).* **Conclusión:** *la correcta identificación del perfil clínico antes de la muerte permite fortalecer la toma de decisiones paliativas oportunas, promoviendo una atención centrada en la dignidad y calidad de vida del paciente.*

Palabras clave: *Cuidados paliativos. Etapa final de la vida. Síntomas terminales.*

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INTRODUCTION

Population aging and the increase in advanced chronic diseases have led to a sustained increase in medical care in the final stage of life, particularly for geriatric patients. In this age group, the last days are often characterized by accelerated functional decline, accompanied by physical, respiratory, and neuropsychiatric symptoms that directly impact the quality of life of the patient and their caregivers.[1]

International literature has consistently described the presence of common clinical signs in the days before death, including decreased food intake, dyspnea, delirium, respiratory rales, and progressive loss of functional autonomy.[2-4] Multicenter studies conducted in Europe, Asia, and North America have identified that these signs intensify as death approaches and can be considered clinical markers of imminent agony, with direct implications for timely palliative care decisions and the limitation of futile interventions.[5-7]

However, most of this evidence comes from specialized palliative care units, hospices, or healthcare settings with formal end-of-life care structures. In contrast, in secondary-level general hospitals, where a significant proportion of deaths in older adults occur, patients often die outside of comprehensive palliative care programs, and the clinical identification of the end-of-life phase depends largely on empirical recognition by the treating team.[8,9]

In Mexico and other middle-income countries, evidence on the immediate clinical course before death in hospitalized geriatric patients is limited and fragmented, hindering the generalization of international findings and their application in institutional settings with restricted resources.[10] This knowledge gap limits the timely recognition of the dying stage and favors the persistence of disproportionate interventions that do not contribute to the patient's comfort or dignity.

Based on the clinical hypothesis that hospitalized geriatric patients present an identifiable pattern of signs and symptoms in the week before death, regardless of the etiology of advanced disease, this study aims to systematically describe the clinical profile of this stage in a secondary-level general hospital. Identifying these patterns seeks to provide local evidence that complements international findings and contributes to improving timely palliative care decisions in non-specialized hospital settings.

MATERIAL AND METHODS

Study design

This was a cross-sectional observational study with prospective clinical follow-up during hospitalization until death.

Study population

The study was conducted in a secondary-level public hospital in Mexico. Between December 2023 and October 2024, 133 hospitalized patients aged 60 years or older were registered. These patients were considered to be in serious condition by the attending physician or service. Of these, 65 patients (48.9%) met the inclusion criteria and were included in the study (Fig. 1).

Inclusion criteria

- Hospitalized patient aged ≥ 60 years.
- Patient meeting the terminal illness criteria established in current national regulations.[11]

Exclusion criteria

- Diagnosis of brain death
- Invasive or non-invasive mechanical ventilation.

Sample size

The sample size was calculated a priori, considering the analytical cross-sectional design of the study. For this purpose, the formula for finite populations for proportions was used, taking as a reference an average universe of 77 semi-annual deaths in people aged 60 years or older registered in the study unit.

The formula used was as follows:

$$n = \frac{N * Z2_{\acute{a}} * p * q}{e^2 * (N-1) + Z2_{\acute{a}} * p * q}$$

Signaling:

- n = desired sample size
- N = population size (77 average deaths in 6 months at the hospital in people > 60 years old)
- Zα = distance from the mean of the proposed significance value. With a 95% confidence level (α = 0.05, Zα = 1.96)
- e = maximum acceptable estimation error (5%)
- p = probability of the studied event occurring (0.5)
- q = probability of the studied event not occurring (0.5)

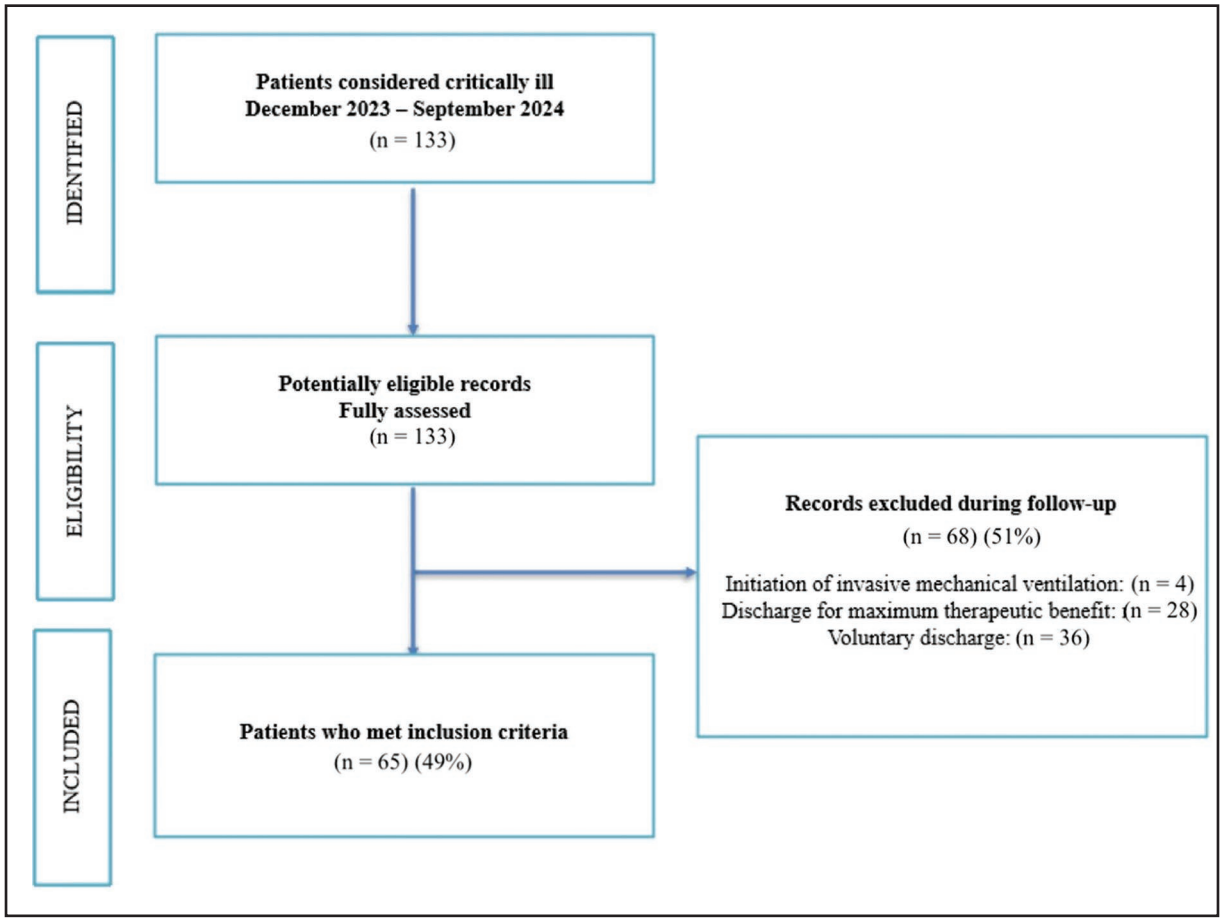

Figure 1. *Data selection diagram (preferred reporting items for systematic reviews and meta-analyses). Patients were assessed for eligibility and followed until the final in-hospital outcome. Exclusions occurred during follow-up for predefined clinical reasons. n: number of subjects or people.*

Based on these parameters, the resulting sample size was 65 patients, which provided a 95% confidence level and allowed an adequate estimate of the frequencies of the main clinical signs evaluated in the final stage of life.

Sampling

Consecutive non-probability sampling was performed until the calculated sample size was reached.

The patient selection, inclusion, and exclusion process is summarized in the preferred reporting items for systematic reviews and meta-analyses flowchart (Fig. 1).

Variables and data collection

Sociodemographic variables (age and sex) and clinical signs characteristic of the final stage of life were recorded using a well-structured data collection instrument applied daily by trained medical personnel. Clinical variables included: decreased food intake, dyspnea, edema, delirium assessed using the confusion assessment method, pain assessed with a visual analog scale or numerical analog scale, death rattle, jaw-moving breathing, periods of apnea, and whether there was a degree of autonomy or decreased functionality, assessed using the barthel index (≤ 60), as shown in the variable operationalization table (Table 1).

Potential biases

This study may be subject to methodological biases arising from its design and execution context. First, the consecutive non-probability sampling and the exclusive inclusion of patients who died in the hospital could introduce selection bias, as they do not represent all elderly patients in advanced stages of disease who are discharged for other reasons or receive care outside the hospital setting.

Furthermore, given that the identification of clinical signs was performed through daily clinical observation, there is a possibility of observer-dependent measurement bias, particularly in variables with a subjective component (such as dyspnea, delirium, pain, dreams and visions, and terminal lucidity), despite the use of validated instruments and the prior training of the medical personnel involved.

Finally, the study was conducted in a second-level public hospital within the Mexican health system, whose structural conditions, resource availability, and care practices may differ from other national and international contexts, introducing a context bias and limiting the extrapolation of the findings to environments with different care models.

Statistical analysis

A descriptive analysis was performed by calculating absolute frequencies, percentages, and 95% confidence intervals (CIs) for the clinical variables.

RESULTS

Sixty-five patients were included, with a mean age of 77.1 ± 9.7 years; 54% (n = 35) were women and 46% (n = 35) were men. All patients died during hospitalization and met clinical criteria for terminal illness.

During the week before death, a consistent clinical pattern of progressive deterioration was observed. Decreased food intake was the most frequent finding, present in 100% of patients (95% CI: 94.5-100). Loss of functional autonomy, defined as a score ≤ 60 on the Barthel Index, was documented in 96.9% of cases (95% CI: 89.3-99.6) (Table 2).

Other relevant clinical signs included edema in 55.4% of patients (95% CI: 42.3-67.9), dyspnea in 46.2% (95% CI: 33.7-59.1), and pain in 40.0% (95% CI: 28.0-53.1). Delirium was present in 30.8% of cases, while respiratory crackles were identified in 23.1% of patients (Table 2).

Respiratory signs, such as death rales and jaw-moving breathing, showed a progressive increase as death approached. Concurrently, decreased food intake and loss of functional autonomy remained virtually universal findings throughout the observation period (Table 2).

The temporal distribution of the main signs and symptoms during the days prior to death showed an intensification of respiratory and functional impairment in the last 48 h of life, while nutritional alterations were constantly present throughout the previous week (Table 2).

DISCUSSION

The timely recognition of the end-of-life stage in hospitalized geriatric patients is a central clinical challenge for making appropriate clinical decisions, particularly in general hospitals where specialized palliative care is not always available.

In this context, the present study provides relevant evidence by systematically documenting the clinical profile of progressive deterioration during the week before death in older adult patients in the Mexican context, characterized by an almost universal loss of food intake and functional autonomy, as well as the gradual appearance of terminal respiratory signs. These findings are not only consistent with the international literature but are especially important because they were identified, described, and observed in a secondary-level hospital in Mexico, reflecting everyday clinical practice outside of specialized palliative care units.

Decreased food intake and severe functional decline were the most frequent signs, which are consistent with the findings described by the World Health Organization and by Hui et al., who identify these findings as early and consistent markers of impending death in patients with advanced illness.[1,2] Similarly, systematic reviews have indicated that the loss of the ability to feed oneself and near-total functional dependence are key clinical signs for the timely recognition of the end-of-life stage.[6]

The prevalence of dyspnea and pain observed in this study falls within the range reported in research conducted in Europe and North America, where these symptoms affect approximately 40-70% of patients in the last days of life.[3,5] However, the moderate frequency of these symptoms in our cohort could be influenced by the general hospital context and by the exclusion of patients on mechanical ventilation, suggesting a possible underestimation of terminal respiratory burden, as has been noted in previous studies.[6]

The progressive increase in respiratory rales and jaw-moving breathing as death approaches is consistent with classic descriptions of the dying process, particularly in the last 48-72 h of life.[5,7] These signs have been recognized as clinical indicators of irreversibility and as critical points for transitioning to a palliative approach focused on comfort, rather than prolonging interventions without clinical benefit.[1,7]

Table 1. Definition of variables and their operationalization

Variable	Conceptual definition	Operational definition	Type of variable	Scale of measurement	Unit of measurement
Age	Time a person has lived, taking into account their birth.	Corroboration in clinical record or by means of patient identification document.	Quantitative	Discreet	0 = 60-69 years old 1 = 70-79 years old 2 = 80-89 years old 3 = 90 or more years old
Sex	Organic condition that distinguishes men from women.	Assessment by phenotype at the time of intervention, and corroboration with the clinical record.	Qualitative	Nominal, dichotomous	0 = Women 1 = Man
Service	Physically delimited spaces and areas of specialization, thus comprising the healthcare, diagnostic or therapeutic activities, and health promotion that they offer to patients.	Data collection at the time of application of the evaluation instrument.	Qualitative	Nominal polytomous	0 = Internal medicine 1 = Surgery 2 = Traumatology 3 = ICU 4 = Emergency department
Oliguria	Diuresis < 200 mL/day	Sum of urine output from the previous day reported in the discharge section of the nursing record.	Qualitative	Dichotomous nominal	0 = Yes 1 = No
Dyspnea	Subjective experience of difficulty breathing that includes qualitatively different sensations of varying intensity	A sensation of shortness of breath or suffocation verbally expressed by the patient, or evidence of increased work of breathing observed by the examining physician or diagnosed by the physicians in charge of the patient's care.	Qualitative	Dichotomous nominal	0 = Yes 1 = No
Edema	Clinically apparent increase in interstitial fluid volume.	Increased volume of lower or upper extremities or face observed by the examining physician or diagnosed by the physicians of the service in charge of the patient	Qualitative	Dichotomous nominal	0 = Yes 1 = No
Delirium	A change in attention and awareness that	Confusion assessment	Qualitative	Dichotomous nominal	0 = Yes 1 = No

(Continues)

Table 1. Definition of variables and their operationalization *(continued)*

Variable	Conceptual definition	Operational definition	Type of variable	Scale of measurement	Unit of measurement
	develops over a short period of time and tends to fluctuate.	method criteria (1 + 2) + (3 or 4)			
Pain	A sensory and emotional experience; which is not pleasant and is associated with tissue damage, whether real, potential, or described in terms of such damage.	Use of visual analog scale or analog numerical scale	Qualitative	Dichotomous nominal	0 = Yes 1 = No
Respiratory rales	Noisy breathing caused by the accumulation of mucous secretions in the airways.	Identified by the evaluating physician at the time of the examination, or a variable described in medical or nursing notes or observed by physicians in charge of the patient	Qualitative	Dichotomous nominal	0 = Yes 1 = No
Breathing with jaw movement	Abnormal breathing pattern in which breathing becomes very labored and the mouth opens as a result of periodic jaw movement during inspiration.	Identified by the evaluating physician at the time of the examination, or a variable described in medical or nursing notes or observed by physicians in charge of the patient	Qualitative	Dichotomous nominal	0 = Yes 1 = No
Terminal dreams and visions	Dreams or visual hallucinations that are associated with joy, excitement, or distress, characterized by organized thoughts without interruption of consciousness, attention, and concentration.	Experience described verbally by the patient or family members who witnessed the event.	Qualitative	Dichotomous nominal	0 = Yes 1 = No
Terminal lucidity	This period is characterized by an increase in alertness, energy, and memory function.	Experience described verbally by primary caregiver or corroborated by medical or nursing notes	Qualitative	Dichotomous nominal	0 = Yes 1 = No
Autonomy (Barthel's basic ADL)	Evaluated by the dependence in basic ADL of Barthel being positive ≤ 60%.	Use of the Barthel scale to assess independence based on ADLs	Qualitative	Dichotomous nominal	0 = Yes 1 = No

(Continues)

Table 1. Definition of variables and their operationalization *(continued)*

Variable	Conceptual definition	Operational definition	Type of variable	Scale of measurement	Unit of measurement
Periods of apnea	Absence of respiratory flow for at least 10 s.	Identified by the evaluating physician at the time of the examination, or a variable described in medical or nursing notes, or observed by physicians in charge of the patient	Qualitative	Dichotomous nominal	0 = Yes 1 = No
Gastrointestinal bleeding	Macroscopic bleeding characterized by hemoptysis, hematemesis, melena, and rectal bleeding.	Identified by the evaluating physician at the time of the examination, or a variable described in medical or nursing notes or observed by physicians in charge of the patient	Qualitative	Dichotomous nominal	0 = Yes 1 = No
Recent nutritional alteration	Unintentional weight loss of > 10% in the last 3 months, or BMI < 18.5 kg/m^2.	Corroborated with weight and height at the time of hospitalization, measured with a measuring tape and using the service scale for weighing or weight and height written on nursing sheets	Qualitative	Dichotomous nominal	0 = Yes 1 = No
Decreased intake	Refusal to eat or drink due to lack of appetite.	Identified by the evaluating physician at the time of the examination, or a variable described in medical or nursing notes or observed by physicians in charge of the patient	Qualitative	Dichotomous nominal	0 = Yes 1 = No

ADL: activities of daily living; BMI: body mass index; ICU: intensive care unit.

A relevant aspect of this study is that the findings were obtained in a secondary-level general hospital, where formal palliative care is not always systematically integrated and where there is no specialized palliative care service-a situation reflected in the vast majority of health services in the country. Unlike studies conducted in hospices or specialized palliative care units in other countries, primarily in the United States or Europe, the results presented here reflect everyday clinical practice in institutions where the identification of dying depends mainly on the clinical judgment of the treating team[8,9]. In this context, the systematization of observed clinical signs can contribute to reducing variability in care, facilitating the timely limitation of futile interventions or those without real clinical benefit and prognosis, and promoting more dignified and humane end-of-life care.[1,8]

Table 2. Frequency of clinical signs in the week before death (n = 65)

Clinical sign	n (%)	IC 95%
Decreased intake	65 (100)	94.5-100
Impaired functional autonomy (Barthel ≤ 60)	63 (96.9)	89.3-99.6
Edema	36 (55.4)	42.3-67.9
Dyspnea	30 (46.2)	33.7-59.1
Pain	26 (40.0)	28.0-53.1
Delirium	20 (30.8)	20.3-43.3
Respiratory rales	15 (23.1)	13.9-35.0
Breathing with jaw movement	14 (21.5)	12.6-33.6

CI 95%: confidence interval; n: subjects included.

From a critical perspective, the results must be interpreted considering the methodological limitations of the study. The observational, single-center design restricts the generalizability of the findings, and although the sample size was calculated a priori, some low-frequency clinical manifestations limited the possibility of performing multivariate analyses or making causal inferences. Furthermore, the lack of evaluation of specific palliative interventions or family and psychosocial outcomes prevents assessing the direct impact of these findings on clinical decision-making.[9,10]

Overall, this study complements existing international evidence by providing local data from the Mexican context, helping to close the knowledge gap regarding end-of-life care for geriatric patients in non-specialized settings. Future multicenter studies with more robust analytical designs and the integration of clinical, palliative, and psychosocial variables will be necessary to further explore these findings and assess their impact on the quality of end-of-life care in Mexico.[8-10]

The oncology versus non-oncology comparison groups were not included because comparison between groups was not feasible due to their lack of homogeneity and statistically sufficient size.

Limitations

This study has inherent limitations due to its observational, single-center, and non-probability sampling design, which restricts the generalizability of the results to other hospital settings.

In addition, data collection was based on daily clinical observation, which could introduce observer-dependent variability, despite the training of the personnel involved.

Finally, the findings must be interpreted within the context of the Mexican healthcare system, characterized by structural and regulatory limitations, variable resource availability, heterogeneity in access to palliative care stemming from a lack of training and knowledge on the subject, and the allocated budget. These factors impact decision-making processes, which are conditioned by institutional and sociocultural factors. These particularities can significantly modify local clinical practice and differ from other international contexts.

Taken together, these limitations underscore the need for future multicenter studies with more robust analytical designs that integrate clinical variables, palliative interventions, and outcomes centered on the patient, their family, and their environment, explicitly considering the impact of the health and social context on end-of-life care.

CONCLUSION

The clinical profile of geriatric patients during the week before death is characterized by progressive functional decline and the presence of identifiable clinical signs that allow for anticipation of the final phase of life. Their timely recognition is a key tool for strengthening palliative care decision-making focused on comfort, dignity, and the reduction of avoidable suffering in general hospitals.

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CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

ETHICAL CONSIDERATIONS

Protection of human subjects and animals. The authors declare that no experiments on humans or animals were performed for this research.

Confidentiality, informed consent, and ethical approval. The authors have followed their institution's confidentiality protocols, obtained informed consent from all patients, and secured approval from

the Ethics Committee. SAGER guidelines have been followed as applicable to the nature of the study.

Declaration on the use of artificial intelligence. The authors declare that no generative artificial intelligence was used in the writing or creation of the content of this manuscript.

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THE JOURNAL OF LATIN AMERICAN GERIATRIC MEDICINE ORIGINAL ARTICLE

Cognitive changes after transcatheter aortic valve implantation versus surgical aortic valve replacement in older adults

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Abstract

Background: *Cognitive impairment affects approximately 50 million people worldwide, including 5-8% of adults aged over 60 years. This condition is common in patients with severe aortic stenosis (AS). Although both transcatheter aortic valve implantation (TAVI) and surgical aortic valve replacement (SAVR) improve the quality of life, evidence on post-treatment cognitive recovery remains limited.* **Objective:** *The objective of the study is to analyze differences in cognitive status among older adults with severe AS undergoing TAVI or SAVR 3 months after intervention.* **Material and methods:** *An observational, retrospective cohort study was conducted including patients ≥ 70 years of age with severe AS treated with TAVI or SAVR (2023-2024). Comprehensive geriatric assessment and Mini-Mental State Examination (MMSE) scores were obtained from medical records and reassessed after 3 months. Statistical analyses were performed using the Statistical Package for the Social Sciences version 25.* **Results:** *A total of 103 patients were included (70 SAVR and 33 TAVI). Sex distribution was similar ($p = 0.592$). Patients who underwent TAVI were older (80.6 ± 6.2 vs. 74.8 ± 3.2 years; $p < 0.001$) and more frequently frail (48.5 vs. 25.7%; $p = 0.022$) or had suspected sarcopenia (57.6 vs. 35.7%; $p = 0.036$). No significant differences were observed in the MMSE scores between the groups.* **Conclusions:** *Despite the greater age, frailty, and sarcopenia in the TAVI group, cognitive trajectories at 3 months did not significantly differ from those in SAVR patients.*

Keywords: *Aortic valve stenosis. Cognitive dysfunction. Frail elderly.*

Cambios cognitivos tras una TAVI frente a una SAVR en adultos mayores

Resumen

Antecedentes: *La OMS estima que el deterioro cognitivo afecta a 50 millones de personas y entre el 5 y 8% de los mayores de 60 años. En pacientes con estenosis aórtica grave, este deterioro es frecuente y se ha observado mejoría cognitiva tras la intervención con implante percutáneo de válvula aórtica (TAVI). Dado que los déficits cognitivos en adultos mayores predicen deterioro funcional y mortalidad, su abordaje es crucial. Aunque la estenosis aórtica afecta calidad de vida, funcionalidad y cognición, la evidencia sobre la recuperación cognitiva tras tratamientos es limitada. Mientras TAVI muestra beneficios en este ámbito, los efectos del reemplazo valvular aórtico quirúrgico (SAVR) siguen siendo controvertidos.* **Objetivo:** *Analizar las diferencias del estado cognitivo en pacientes adultos mayores con estenosis aórtica grave, sometidos a TAVI o SAVR 3 meses posterior a su intervención terapéutica.* **Material y métodos:** *Estudio de cohorte observacional y retrospective, en el que se incluyeron pacientes ≥ 70 años con estenosis aórtica grave, tratados mediante TAVI o SAVR en el periodo de 2023 a 2024. Del expediente clínico se documentó la valoración geriátrica integral y el estado cognitivo mediante el Mini-Mental State Examination (MMSE). A los tres meses, se revaloró el estado cognitivo. Se aplicó estadística descriptiva e inferencial para analizar diferencias de puntajes en dos tiempos, utilizando SPSS v25.* **Resultados:** *Se incluyeron 103 pacientes: 70 en el grupo SAVR y 33 en el grupo TAVI. La distribución por sexo fue similar entre ambos grupos ($p = 0.592$). Los pacientes TAVI eran de mayor edad (80.6 ± 6.2 vs. 74.8 ± 3.2 años; $p < 0.001$), con*

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mayor prevalencia de fragilidad (48.5 vs. 25.7%; p = 0.022) y sospecha de sarcopenia (57.6 vs. 35.7%; p = 0.036). No hubo diferencias significativas en los puntajes del MMSE, deterioro cognitivo ni evolución de la sarcopenia. **Conclusiones:** *A pesar de que los pacientes TAVI presentaban mayor edad, fragilidad y sarcopenia, no se observaron diferencias significativas en la evolución cognitiva entre ambos grupos a los tres meses.*

Palabras clave: *Estenosis valvular aórtica. Disfunción cognitiva. Adulto mayor frágil.*

INTRODUCTION

Aging is one of the biggest challenges for contemporary healthcare systems. Among the most frequent cardiovascular diseases in older adults is aortic stenosis (AS), a progressive degenerative condition that significantly affects the quality of life and functional capacity. In this context, therapeutic interventions, such as surgical aortic valve replacement (SAVR) and transcatheter aortic valve implantation (TAVI), have transformed clinical management by extending treatment to high-risk or frail patients with AS. However, beyond hemodynamic improvement, there is growing interest in understanding the cognitive effects of these interventions, particularly in adults over 70 years of age, where cognitive decline is a primary concern.[1] Although both SAVR and TAVI prolong survival and improve cardiac function, their possible impact on the central nervous system, especially memory, attention, and processing speed, remains uncertain. Clarifying these effects has clinical and ethical implications, as it allows for person-centered care that considers cognitive capacity during decision-making and post-operative rehabilitation.[1] Globally, cognitive impairment affects approximately 50 million people, and between 5% and 8% of individuals aged ≥ 60 years present with it at any given time.[1] The Mini-Mental State Examination (MMSE) is a standard cognitive screening tool for older adults that has been widely disseminated and adapted to different languages and contexts. Originally developed by Brandt et al.[2] in 1975, it typically requires 5-10 min to administer and remains the most used standardized test in clinical settings, using the version adapted by the *Instituto Nacional de Geriatría* (INGER, Mexico). Its performance has also been validated for remote (tele-neuropsychological) administration, with no substantial differences compared to in-person assessment.[2-4] AS is the most frequent valvular disease in older adults in developed countries, with a prevalence that increases sharply with age.[5,6] The etiology is predominantly degenerative valvular calcification in those over 70 years of age, while bicuspid aortic valve predominates in younger patients.[5] As the disease progresses, patients develop angina, congestive heart failure, and syncope; the onset of symptoms carries a poor prognosis, with approximately 50% mortality at 2 years without intervention, and an annual mortality in severe AS reaching up to 25%, with an average survival of 2-3 years after diagnosis.[7] In cognitive terms, severe AS has been associated with a high burden of impairment (≈ 21-39%), and among those undergoing TAVI, the reported prevalence ranges between 28% and 45%.[8-12] In the PARTNER cohort, each one-point decrease in the pre-procedure MMSE score was associated with worse clinical outcomes at 6 months and 1 year.[13,14] Other studies have linked cognitive impairment with higher mortality rates, both after TAVI and after surgical approaches.[15] "Silent" cerebral microembolic events occur in most TAVI cases (≈ 8 of every 10), raising concerns about the potential subtle cognitive effects of the procedure. More generally, neurological injury is a common complication of cardiac surgery and may contribute to cognitive decline.[16] Despite the hemodynamic benefits of TAVI, approximately 1 in 4 treated patients died within 1 year in the PARTNER trial, and some survivors reported persistently low quality of life with symptoms of heart failure and functional limitations during the year following the procedure.[13,17,18] TAVI is less invasive than SAVR and may be associated with more favorable cognitive outcomes. However, TAVI candidates often present with a greater comorbidity burden, raising the question of how cognitive trajectories differ between patients undergoing TAVI versus SAVR. Given this background, the present study at *Unidad Médica de Alta Especialidad No. 34* aimed to determine whether there are differences in short-term cognitive outcomes (MMSE) between TAVI and SAVR.

MATERIAL AND METHODS

This was a retrospective observational cohort study. The study was approved by the Local Research Ethics Committee and the Health Research Committee of the hospital for data collection and use for research purposes while maintaining the anonymity of

Table 1. General characteristics of the 103 elderly patients underwent SAVR and TAVI

Variables	Global (n = 103) n (%)	SAVR (n = 70) n (%)	TAVI (n = 33) n (%)	p
Age (years), media ± SD	76.7 ± 5.2	74.8 ± 3.2	80.6 ± 6.2	< 0.001*
Sex				0.592**
Male	57 (55.3)	40 (57.1)	17 (51.5)	-
Female	46 (44.7)	30 (42.9)	16 (48.5)	-
Schooling				0.956**
None	5 (4.9)	4 (5.7)	1 (3)	-
Primary	56 (54.4)	38 (54.3)	18 (54.5)	-
Secondary	7 (6.8)	5 (7.1)	2 (6.1)	-
High school	16 (15.5)	10 (14.3)	6 (18.2)	-
Degree	18 (17.5)	12 (17.1)	6 (18.2)	-
Postgraduate	1 (1)	1 (1.4)	0 (0)	-
Marital status				0.131**
Married	61 (59.2)	45 (64.3)	16 (48.5)	-
Widowhood	30 (29.1)	17 (24.3)	13 (39.4)	-
Single	7 (6.8)	3 (4.3)	4 (12.1)	-
Divorced	3 (2.9)	3 (4.3)	0 (0)	-
Free union	2 (1.9)	2 (2.9)	0 (0)	-

*t-student; **Chi-square.
SAVR: aortic valve replacement; TAVI: transcatheter aortic valve implantation; SD: standard deviation.

the subjects. Registration number: R-2024-1902-001. It includes 103 adults aged 70 years with clinically confirmed severe AS undergoing TAVI or SAVR between July 2023 and July 2024, 35 of whom were lost to follow-up at 3 months or did not have a complete second cognitive evaluation. Patients diagnosed with or suspected to have major neurocognitive disorders were excluded. Cognitive status was reassessed after 3 months. Descriptive and inferential statistics (Mann–Whitney U test) were applied to analyze score differences at 2 time points using the Statistical Package for the Social Sciences v25.

RESULTS

This center routinely performs Comprehensive Geriatric Assessment (CGA), including MMSE (INGER adaptation), both at baseline and follow-up, which allows for homogeneous cognitive monitoring. Table 1 presents the general characteristics of the patients included in the study. The sample included 103 patients: 70 with SAVR and 33 with TAVI. Demographic, clinical, functional, and cognitive data were extracted from the clinical records and CGA. Statistical comparisons were performed using conventional significance tests ($p \leq 0.05$). The mean age was 76.7 ± 5.2 years; TAVI patients were older than SAVR patients (80.6 ± 6.2 vs. 74.8 ± 3.2; $p < 0.001$). The distribution by sex, education, and marital status was similar; most had a primary education. Smoking was more frequent in the SAVR group (77.1 vs. 54.5%; $p = 0.02$). Frailty and suspicion of sarcopenia were more prevalent in the TAVI group (48.5 vs. 25.7%, $p = 0.022$; 57.6 vs. 35.7%, $p = 0.036$), and atrial fibrillation was more common in the TAVI group (15.2 vs. 2.9%, $p = 0.033$). The anthropometric parameters and nutritional status of the patients are shown in Table 2. Body mass index categories and pre-intervention aortic valve area (media 0.63 ± 0.18 cm^2) did not differ significantly between groups ($p = 0.769$ and $p = 0.668$, respectively). Functionally, Barthel and Lawton-Brody scores were higher in the SAVR group at baseline and at 3 months ($p < 0.05$), while the suspicion of sarcopenia remained more frequent in the TAVI group at both time points. The anthropometric parameters and nutritional statuses of the patients are presented in Table 3. Regarding cognition, the baseline MMSE scores and their categorical interpretations did not differ between the groups ($p = 0.974$ and $p = 0.646$, respectively). At 3 months, 92.2% of all patients remained without significant cognitive impairment, with no intergroup differences ($p = 0.455$). Changes in Barthel, Lawton-Brody, and SARC-F scores from baseline to follow-up showed no significant differences

Table 2. Patient comorbidities of the 103 elderly patients underwent SAVR and TAVI

Variable	Global (n = 103) n (%)	SAVR (n = 70) n (%)	TAVI (n = 33) n (%)	p*
Smoking	72 (69.9)	54 (77.1)	18 (54.5)	0.02
Alcoholism	70 (68)	47 (67.1)	23 (69.7)	0.795
Polypharmacy	31 (30.1)	24 (34.3)	7 (21.2)	0.177
Multipathology	70 (68)	49 (70)	21 (63.6)	0.518
Frailty	34 (33)	18 (25.7)	16 (48.5)	0.022
Probable sarcopenia	44 (42.7)	25 (35.7)	19 (57.6)	0.036
Cognitive impairment or dementia	12 (11.7)	8 (11.4)	4 (12.1)	0.577
Diabetes mellitus	53 (51.5)	35 (50)	18 (54.5)	0.667
High blood pressure	77 (74.8)	51 (72.9)	26 (78.8)	0.518
Dyslipidemia	10 (9.7)	8 (11.4)	2 (6.1)	0.319
Atrial fibrillation	7 (6.8)	2 (2.9)	5 (15.2)	0.033
Heart failure	12 (11.7)	6 (8.6)	6 (18.2)	0.139
Depression	11 (10.7)	5 (7.1)	6 (18.2)	0.091
Anxiety disorder	10 (9.7)	5 (7.1)	5 (15.2)	0.176
Chronic coronary syndrome	5 (4.9)	3 (4.3)	2 (6.1)	0.516
Stroke	4 (3.9)	2 (2.9)	2 (6.1)	0.384
Hypothyroidism	7 (6.8)	3 (4.3)	4 (12.1)	0.146

*Chi-square.
SAVR: aortic valve replacement; TAVI: transcatheter aortic valve implantation.

Table 3. Anthropometric parameters and nutritional status of the 103 elderly patients underwent SAVR and TAVI

Variable	Global (n = 103) n (%)	SAVR (n = 70) n (%)	TAVI (n = 33) n (%)	p
Weight (kilograms), mean ± SD	71.7 ± 12	72.3 ± 12.3	70.6 ± 11.4	0.516*
Height (meters), mean ± SD	1.62 ± 0.9	1.62 ± 0.09	1.60 ± 0.08	0.331
BMI (kg/m^2), mean ± SD	27.4 ± 4.6	27.5 ± 4.8	27.3 ± 4.3	0.798*
Nutritional status				0.769**
Low weight	2 (1.9)	2 (2.9)	0 (0)	-
Normal	46 (44.7)	30 (42.9)	16 (48.5)	-
Overweight	39 (37.9)	11 (15.7)	5 (15.2)	-
Obesity	16 (15.5)	27 (38.6)	12 (36.4)	-

*t student; **Chi-square.
SAVR: aortic valve replacement; TAVI: transcatheter aortic valve implantation; SD: standard deviation; BMI: body mass index.

($p > 0.05$). Thus, both procedures preserved cognitive performance in the short term. Table 4 shows the diagnosis at admission and the pre-procedure valve area. Severe AS was observed in 71.8% of the patients, with no significant differences between the groups ($p = 0.891$). The mean valve area was 0.63 ± 0.18 cm^2, with no significant difference between the SAVR and TAVI groups ($p = 0.668$).

Tables 5 and 6 present the results of cognitive assessments. Regarding cognition, the initial MMSE scores and their categorical interpretations did not differ between the groups ($p = 0.974$ and $p = 0.646$, respectively). At 3 months, 92.2% of all patients remained without significant cognitive impairment, with no intergroup differences ($p = 0.455$). Functional assessments at admission and 3 months are presented

Table 4. Diagnosis and valve area of the 103 elderly patients underwent SAVR and TAVI

Variable	Global (n = 103) n (%)	SAVR (n = 70) n (%)	TAVI (n = 33) n (%)	p
Diagnosis upon admission	-	-	-	0.891*
Double aortic lesion	29 (28.2)	20 (28.6)	9 (27.3)	-
Severe aortic stenosis	74 (71.8)	50 (71.4)	24 (72.7)	-
Valve area before intervention (cm^2), mean ± SD	0.63 ± 0.18	0.63 ± 0.20	0.61 ± 0.14	0.668**

*Chi-square; **t-student.
SAVR: aortic valve replacement; TAVI: transcatheter aortic valve implantation; SD: standard deviation.

Table 5. Cognitive evaluation of the 103 elderly patients underwent SAVR and TAVI

Variable	Global (n = 103) n (%)	SAVR (n = 70) n (%)	TAVI (n = 33) n (%)	p
Baseline MMSE, median (minimum-maximum)	28 (26-29)	28 (26-29)	28 (26-29)	0.974*
Interpretations				
Baseline MMSE				0.646**
No cognitive impairment	87 (84.5)	59 (84.3)	28 (84.8)	-
Mild to moderate cognitive impairment	13 (12.6)	8 (11.4)	5 (15.2)	-
Moderate to severe cognitive impairment	2 (1.9)	2 (2.9)	0 (0)	-
Severe cognitive impairment	1 (1)	1 (1.4)	0 (0)	-
MMSE, median (minimum-maximum)	24 (23-25)	24 (23-25)	24 (23-25)	0.376*
MMSE at 3 months				0.455**
No cognitive impairment	95 (92.2)	64 (91.4)	31 (93.9)	-
Mild-to-moderate cognitive impairment	5 (4.9)	3 (4.3)	2 (6.1)	-
Moderate-to-severe cognitive impairment	0	0	0	-
Severe cognitive impairment	3 (2.9)	3 (4.3)	0	-

*U-Mann Whitney; **Chi-square.
MMSE: mini-mental state examination; SAVR: aortic valve replacement; TAVI: transcatheter aortic valve implantation.

Table 6. Changes in the initial assessments of cognitive impairment and suspected sarcopenia at follow-up of the 103 elderly patients underwent SAVR and TAVI

Variable	SAVR (n = 70) n (%)	TAVI (n = 33) n (%)	p
Changes in MMSE	-	-	0.59*
Progression of cognitive decline	2 (2.9)	0 (0)	-
No change in impairment classification	63 (90)	30 (90.9)	-
Improvement in cognitive decline status	5 (7.1)	3 (9.1)	-
Changes in the initial suspicion of sarcopenia	-	-	0.786*
Suspected sarcopenia appears	1 (1.4)	0 (0)	-
No change in initial suspicion	63 (90)	30 (90.9)	-
Suspected sarcopenia disappears	6 (8.6)	3 (9.1)	-

*Chi-square.
MMSE: mini-mental state examination; SAVR: aortic valve replacement; TAVI: transcatheter aortic valve implantation.

Table 7. Functional assessment of the 103 elderly patients underwent SAVR and TAVI

Variable median (minimum-maximum)	Global (n = 103) n (%)	SAVR (n = 70) n (%)	TAVI (n = 33) n (%)	p
Initial Barthel	95 (90-100)	97 (90-100)	95 (80-100)	0.049*
Barthel 3 months	100 (90-100)	100 (90-100)	90 (80-100)	0.013*
Initial Lawton-Brody	7 (5-8)	7 (5-8)	5 (4-8)	0.024*
Lawton-Brody 3 months	5 (5-7)	7 (5-8)	5 (3-5)	0.013*
EFT	1 (1-2)	1 (1-2)	2 (1-3)	0.178*
Suspected sarcopenia at initial stage	26 (25.2)	13 (18.6)	13 (39.4)	0.023**
Suspected sarcopenia at 3 months	18 (17.5)	8 (11.4)	10 (30.3)	0.019**

*U-Mann Whitney; **Chi-square.
SAVR: aortic valve replacement; TAVI: transcatheter aortic valve implantation; EFT: essential frailty tool.

in Table 7. The changes in Barthel, Lawton-Brody, and SARC-F scores from baseline to follow-up were not significantly different ($p > 0.05$). Thus, both procedures preserved cognitive performance in the short term.

DISCUSSION

These findings are consistent with the broader, although heterogeneous, literature: patients referred for TAVI tend to be older and frailer, but short-term cognitive trajectories are usually similar to those observed after SAVR. As we are a tertiary care unit, patients are discharged to continue their evaluation at other hospitals; therefore, some follow-ups can only be performed for short periods, as in this case. The number of patients analyzed was those who underwent TAVI and SAVR procedures that year and met the inclusion criteria. Reviews have highlighted post-operative cognitive decline and silent ischemic lesions after both TAVI and cardiac surgery; however, randomized and observational comparisons increasingly suggest that outcomes may depend more on baseline frailty/sarcopenia and comorbidity than on the surgical approach itself.[19,20] Some studies have reported a lower risk of post-operative delirium with TAVI than with SAVR and potential benefits of using cerebral embolic protection to reduce cognitive decline after TAVI.[21-24] Others showed an equivalent risk of stroke and similar changes in MMSE between approaches, with transient in-hospital MMSE drops more frequent after SAVR that tended to resolve on follow-up.[25-29] The MMSE remains a practical surveillance tool in this context, although more detailed neuropsychological batteries could detect specific domain changes that the MMSE might miss.[21,26]

CONCLUSION

In this study, no significant differences were observed in MMSE-based cognitive evolution between TAVI and SAVR at 3 months in this geriatric cohort. TAVI patients presented with greater frailty and sarcopenia – factors that might influence functional recovery more than cognitive outcomes – but their short-term cognition remained comparable to that of SAVR patients. Both strategies appear to be neurocognitively safe in the short term for older adults with severe AS. Prolonged follow-up with comprehensive neuropsychological evaluation is required to define long-term trajectories and clarify the interactions between cardiac recovery, cerebral perfusion, and microembolization.

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CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

ETHICAL CONSIDERATIONS

Protection of human subjects and animals. The authors declare that no experiments on humans or animals were performed for this research.

Confidentiality, informed consent, and ethical approval. The authors have obtained approval from the Ethics Committee for the analysis of routinely collected and anonymized clinical data; therefore, individual informed consent was not required. Relevant ethical recommendations have been followed.

Declaration on the use of artificial intelligence. The authors declare that no generative artificial intelligence was used in the writing or creation of the content of this manuscript.

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THE JOURNAL OF LATIN AMERICAN GERIATRIC MEDICINE REVIEW ARTICLE

COPD in the older adult: a comprehensive geriatric perspective, from GOLD 2026 to the bedside

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Abstract

In older adults, chronic obstructive pulmonary disease (COPD) is no longer a single disease but a multidimensional problem: beyond respiratory impairment per se, it coexists with frailty, multimorbidity, and polypharmacy. Yet most clinical guidelines are built on studies of younger patients with little comorbidity. This review highlights the particularities the clinician should recognize. The diagnosis of obstruction should rest on the lower limit of normal of the forced expiratory volume in 1 s/forced vital capacity ratio, not on the fixed ratio of 0.70, to avoid overdiagnosis. COPD also settles on a lung that has undergone its own senescence, and both dyspnea and hypoxemia are often multifactorial: they should not be attributed to COPD alone. Pharmacological treatment requires weighing risks and benefits, with particular attention to anticholinergic burden. Finally, in Latin America, biomass smoke and tuberculosis sequelae are important causes of COPD, and care is further shaped by high-altitude living and unequal access to medications and supplemental oxygen. On this basis, this review offers a practical framework to individualize decisions in older adults with COPD.

Keywords: *Chronic obstructive pulmonary disease. Aged. Frailty. Multimorbidity. Polypharmacy. Spirometry.*

EPOC en el adulto mayor: una mirada geriátrica integral, de GOLD 2026 a la cabecera del paciente

Resumen

En el adulto mayor, la enfermedad pulmonar obstructiva crónica (EPOC) deja de ser una sola enfermedad para volverse un problema multidimensional: además del compromiso respiratorio propiamente dicho, coexiste con fragilidad, multimorbilidad y polifarmacia. Sin embargo, las guías clínicas se sustentan, en su mayoría, en estudios realizados con sujetos más jóvenes y con poca comorbilidad. Esta revisión destaca las particularidades que el clínico debe reconocer. El diagnóstico de obstrucción debe basarse en el límite inferior de la normalidad del cociente FEV_1/FVC, y no en el valor fijo de 0.70, para no sobrediagnosticar. La EPOC se instala, además, sobre un pulmón con su propia senescencia, y tanto la disnea como la hipoxemia suelen ser multifactoriales y no conviene atribuirlas únicamente a la EPOC. El tratamiento farmacológico exige ponderar riesgos y beneficios, con especial atención a la carga anticolinérgica. En América Latina, el humo de biomasa y las secuelas de tuberculosis son causas importantes de EPOC, y el manejo se ve condicionado por la altitud y el acceso desigual a fármacos y a oxígeno suplementario. Con ello, esta revisión ofrece un marco práctico para individualizar las decisiones en adultos mayores con EPOC.

Palabras clave: *Enfermedad pulmonar obstructiva crónica. Adulto mayor. Fragilidad. Multimorbilidad. Polifarmacia. Espirometría.*

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INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is the fourth leading cause of death worldwide, with an estimated 3.5 million deaths in 2021, nearly 90% before age 70 in low- and middle-income countries.[1] Its global prevalence is about 10.3% of adults aged 30-79 – some 392 million people[2] – with a burden that is also economic and social.[3] Prevalence and burden rise with age, peaking in the seventh and eighth decades, and cases could reach 600 million by 2050.[4,5] COPD is shifting from the middle-aged smoker to the multimorbid older adult.

In Latin America, this reality has distinctive features. The PLATINO study showed a prevalence in adults aged 40 years or older ranging from 7.8 to 19.7% across cities, with considerable underdiagnosis.[6] Beyond smoking, biomass smoke and tuberculosis sequelae weigh more than in high-income countries[6,7] – tuberculosis more than doubles the odds of airflow obstruction[8] – reflected in GOLD's COPD etiotypes. PLATINO used the fixed ratio < 0.70, which overestimates obstruction in older groups; with scant spirometry, this feeds a documented accuracy problem.[9]

A decisive demographic shift is superimposed: in Latin America and the Caribbean the proportion of older adults is projected to rise from 9% in 2020 to 19% by 2050, at an unprecedented pace.[10] A rapidly aging population with high cumulative exposure and unequal access to spirometry will concentrate a growing burden of COPD in multimorbid older adults–making a geriatric reading of the disease a public-health necessity.

This poses challenges that guidelines, designed for younger patients, do not resolve: a diagnosis confounded by the physiological decline of the forced expiratory volume in 1 s (FEV_1)/forced vital capacity (FVC) ratio and by symptoms attributed to "old age"; an assessment inseparable from frailty, multimorbidity, and polypharmacy; and a treatment whose benefit and risk shift with age.

The GOLD 2026 report introduced relevant changes – case finding, "disease activity", biologics, vaccination (including RSV), a new classification of exacerbations and their cardiovascular link, and multimorbidity – that warrant a geriatric reading. This review integrates these developments with the principles of geriatric medicine to offer a practical framework for diagnosis, assessment, treatment, and care planning in this age group.

Search strategy. This narrative review searched PubMed for English- and Spanish-language articles on COPD in older adults, without a predetermined time frame, combining terms such as "COPD," "older adults," "frailty," "comprehensive geriatric assessment," "GOLD," "spirometry," "exacerbations" and "palliative care". References were selected for relevance, methodological quality, and geriatric contribution, prioritizing recent evidence, the GOLD 2026 report, seminal studies, and Latin American data. No formal systematic-review methodology (e.g., PRISMA) was applied – a limitation when interpreting the conclusions.

LUNG AGING AND PATHOPHYSIOLOGY OF COPD IN THE OLDER ADULT

Understanding COPD in the older adult requires distinguishing physiological lung aging from disease. Lung function peaks around age 25 and, from about age 30, declines progressively; FEV_1 falls more than FVC, so the FEV_1/FVC ratio decreases with age even in the absence of disease.[11,12] This physiological fall in the ratio is the root of the diagnostic problem addressed below: a fixed threshold of 0.70 overestimates obstruction in older people.

COPD results from the cumulative interaction between genetic and environmental factors that damage the lung or alter its development and aging.[13] Some reach a reduced peak lung function – through prematurity, low birth weight, or early infections – and cross the obstruction threshold even with a normal decline; others start from a normal peak and lose it rapidly. In the older adult, physiological decline, lifelong suboptimal trajectories, and cumulative exposure converge, explaining the high prevalence and heterogeneity.

This crystallizes in GETomics (Agustí and Faner): health and disease result from cumulative interactions between genes (G) and the environment (E) across the time (T) of life, so each interaction's effect depends on the age at which it occurs and on prior history.[13,14] The older patient with COPD is the integral of a lifetime of gene–environment interactions. This explains the heterogeneity and multimorbidity and reorients treatment: if G is unmodifiable and past exposure already consolidated as damage, the therapeutic lever falls on what remains modifiable and on treatable traits.

Closely linked but distinct is the trajectome: the lung-function trajectories in the population (normal, low, or supranormal).[15] If GETomics describes the mechanism, the trajectome maps the possible outcomes. This is of first-order geriatric value: we meet the older adult at the end of the trajectory, with a

single cross-sectional measurement, and the same reduced FEV_1 may arise from very different routes. In cohorts, about half of those who developed COPD started from a normal FEV_1 and declined rapidly, while the other half never reaching a normal peak.[16] This cautions against a single spirometric value (section 3) and underscores longitudinal spirometry.

GOLD 2026 adds the concept of "disease activity", distinguishing reversible biological pathways that influence outcomes – e.g., Type 2 inflammation reflected by eosinophils – from the mere severity of obstruction or the accumulated burden of damage.[4] For the clinician, this separates "age and consolidated damage" from what is still treatable.

DIAGNOSIS OF COPD IN THE OLDER ADULT

The diagnosis of COPD requires persistent airflow obstruction on post-bronchodilator spirometry in a patient with risk exposure and compatible symptoms. In the older adult, each element – threshold, test quality, pattern interpretation, symptom attribution – carries nuances.

The fixed ratio (0.70) versus the lower limit of normal (LLN)

The FEV_1/FVC ratio declines physiologically with age, so a fixed 0.70 threshold overdiagnoses obstruction in the older adult (in whom a ratio < 0.70 may be normal for age) and underdiagnoses it in the young.[17,18] The GLI-2012 equations give an LLN adjusted for age, sex, and height, and the ERS/ATS 2022 standard recommends defining obstruction below the LLN – a z-score below −1.645, the 5th percentile – rather than by the fixed ratio.[17,18] Race-neutral equations (GLI Global) add further reclassification.[19]

GOLD 2026 keeps the fixed post-bronchodilator ratio < 0.70 for practicality and comparability, while acknowledging its limits.[4] The geriatric recommendation is to read spirometry with the LLN/z-scores, integrate symptoms and exposure, and distrust a diagnosis based solely on a ratio just below 0.70 in an older patient without risk factors.

Feasibility and quality of spirometry in the older adult

Spirometry depends on cooperation, and acceptable, repeatable maneuvers can be hard to obtain in the frail patient with cognitive impairment, hearing loss, weakness, or postural limitation. A non-negligible proportion fail a quality test, and a suboptimal one, read literally, misleads in both directions. When the forced maneuver is not feasible, effort-independent parameters should be considered.[20]

Respiratory oscillometry has special value here: it measures respiratory-system mechanics (resistance and reactance) during quiet tidal breathing, without forced maneuvers, and is effort-independent – feasible precisely where spirometry tends to fail.[21] It is also sensitive to small-airway dysfunction that spirometry may miss. It does not replace spirometry (diagnosis still rests on the post-bronchodilator FEV_1/FVC ratio) but is a valuable alternative or complement, and a growing area of interest for functional assessment of the older adult.

Pre-COPD and PRISm

Two recent categories are pertinent. Pre-COPD designates people with respiratory symptoms and/or structural (e.g., emphysema) or functional abnormalities but without spirometric obstruction, at higher risk of developing COPD. PRISm (preserved ratio, reduced FEV_1) is frequent, associated with multimorbidity and higher mortality, and dynamic, able to transition toward obstruction or normality.[4,22] Recognizing it avoids false reassurance from a spirometry "without obstruction" in a symptomatic older adult and prompts surveillance of modifiable factors.

Differential diagnosis and the cardiopulmonary axis

In the geriatric patient, dyspnea and exercise intolerance rarely have a single cause: heart failure, ischemic heart disease, anemia, obesity, deconditioning, interstitial lung disease, and sarcopenia coexist with – or mimic – COPD. GOLD 2026 emphasizes the cardiopulmonary axis (the close COPD–cardiovascular interrelation) as cross-cutting.[4] The implication is not to attribute the picture to COPD prematurely, but to quantify the cardiovascular, hematological, and musculoskeletal contributions.

Case finding and underdiagnosis

COPD is markedly underdiagnosed – in the PLATINO cities, 88.7% of those with it by spirometry lacked a prior diagnosis[9] – partly because symptoms are

blamed on "old age" or comorbidities. GOLD 2026 reinforces active case finding in symptomatic or exposed individuals.[4] In the Undiagnosed COPD and Asthma Population, community spirometry identified undiagnosed asthma or COPD, and pulmonologist-directed care reduced health-service use and improved outcomes.[23] In the older patient, balance this with the caution of section 3.1: seek the undiagnosed patient, but read spirometry with the LLN to avoid overdiagnosis.

COMPREHENSIVE GERIATRIC ASSESSMENT IN COPD

In the older adult, assessing COPD cannot be reduced to grading obstruction and exacerbation risk (the ABE scheme); burden, prognosis, and tolerance depend as much on what lies outside the lung: multimorbidity, frailty, sarcopenia, and geriatric syndromes. Assessment must therefore be coupled with a comprehensive geriatric assessment - complementing the respiratory evaluation with its core domains: functional status, mobility and falls, cognition, nutrition and sarcopenia, polypharmacy, and the patient's context and goals. Each reshapes management: impaired function or cognition conditions device choice; malnutrition and sarcopenia alter pharmacotherapy's benefit-risk; falls risk and polypharmacy weigh against drugs of uncertain benefit; and the patient's goals anchor every decision.

Multimorbidity and comorbidity clusters

In the older adult, "pure" COPD is almost a fiction; the rule is multimorbidity, with virtually all patients having at least one comorbidity and more than half four or more.[24] Several influences survival independently of lung function: the "comorbidome" and COTE index add prognostic information beyond BODE.[25] Comorbidities cluster into recognizable groups.[24] GOLD 2026 devotes a redesigned chapter to multimorbidity,[4] and this accumulation worsens prognosis and multiplies cost.[3] Practically: seek high-impact comorbidities and recognize the dominant cluster for prioritization.

Frailty and sarcopenia

Frailty – diminished physiological reserve and heightened vulnerability to stressors – is especially prevalent in COPD. Operationalized with the Fried phenotype, it predicts falls, disability, hospitalization, and death.[26] In COPD, prefrailty is around 56% and frailty 19%, roughly twice the risk versus non-COPD, probably bidirectional.[27] Sarcopenia affects about 15% of stable patients.[28] Crucially, frailty and sarcopenia are not inevitable with aging: they should be sought systematically because they modify prognosis and tolerance and, above all, are potentially reversible.

Geriatric syndromes and polypharmacy

Added to this are the classic geriatric syndromes – falls, cognitive impairment, depression and anxiety, malnutrition, incontinence – more frequent in COPD and intertwined with its treatment: cognitive impairment and loss of dexterity compromise inhaler technique; depression and anxiety worsen dyspnea and adherence; malnutrition feeds sarcopenia; polypharmacy multiplies interactions and adverse events, including anticholinergic burden. Identifying them is indispensable for a safe plan.

Instruments and integrated assessment

Symptom scales such as the mMRC (modified Medical Research Council dyspnea scale) and the CAT (COPD Assessment Test) remain useful, but in the older adult, they should be read within the comprehensive geriatric assessment above – complemented by simple functional measures such as gait speed and grip strength – so decisions rest on the whole patient, not a single organ.

Outcomes that matter in the older adult

In older adults, success extends beyond FEV_1 and exacerbation counts: functional independence, exercise capacity and mobility, quality of life, symptom control, and the burden of hospitalization and its sequelae (deconditioning, delirium, loss of autonomy) often matter more than spirometric change. The impact on caregivers – who sustain adherence, rehabilitation, and home care – and alignment with the patient's goals of care are equally central. Framing management around these person-centered outcomes is the essence of the geriatric approach and should guide how the interventions below are prioritized.

NON-PHARMACOLOGICAL TREATMENT CENTERED ON THE OLDER ADULT

As anticipated in section 2, the greatest non-pharmacological leverage in the older adult falls on what remains modifiable – ongoing exposure, physical activity, nutrition, and immunization. Bringing GETomics to aging and exercise explains why acting on lifestyle and treatable traits keeps yielding benefit even at advanced ages, rather than lapsing into therapeutic nihilism.[14] It is not an accessory but the intervention acting directly on the modifiable part of the trajectory.

Smoking cessation

Smoking cessation remains the only intervention that substantially modifies the course of COPD, and its benefit persists in the older patient, slowing functional decline and reducing exacerbations and cardiovascular risk. It combines behavioral intervention and pharmacotherapy (nicotine replacement, varenicline, bupropion), chosen with attention to comorbidities, interactions, and availability.[4]

Pulmonary rehabilitation adapted to the frail patient

Pulmonary rehabilitation is among the most cost-effective COPD interventions, improving dyspnea, exercise capacity, and quality of life. In the frail or sarcopenic older adult, it is not contraindicated but especially valuable: training can reverse sarcopenia in some patients[28] and brings GETomics into practice via the modifiable exercise–aging interaction.[14] Intensity and modality should be adapted (strength, intervals, home-based programs) and the nutritional component integrated (protein, deficits such as vitamin D). Frail patients complete it less often, but those who do respond well many no longer meeting frailty criteria afterward.[29]

Vaccination (GOLD 2026 update)

Immunization is high-yield in the geriatric COPD patient. GOLD 2026 updates the schedule and adds the respiratory syncytial virus (RSV) vaccine from age 50, alongside annual influenza, pneumococcal, COVID-19, and pertussis (Tdap) vaccination per local recommendations.[4] In older adults, in whom infections precipitate exacerbations and cardiovascular events, this deserves explicit attention.

Long-term oxygen therapy and home non-invasive ventilation

Long-term oxygen therapy improves survival in severe resting hypoxemia – the usual criterion is a $PaO_2 \leq 55$ mmHg, or ≤ 59 mmHg with cor pulmonale or polycythemia ($SpO_2 \leq 88\%$) – but not in mild-to-moderate hypoxemia or isolated exertional desaturation, so the indication must be precise.[4]

Crucially for Latin America, these thresholds derive from low-altitude studies (NOTT, MRC) and do not transfer to populations living at altitude, where lower barometric pressure reduces PaO_2 – more so in COPD – with compensatory hyperventilation.[30] The region concentrates enormous high-altitude populations – Mexico City (2,240 m), Bogotá (2,640 m), Quito (2,850 m), La Paz (3,640 m) – and applying sea-level cut-offs may misclassify patients; adapting the criteria to the consequences of chronic hypoxemia (pulmonary hypertension, polycythemia) rather than a single value has been proposed,[31] and desaturation and home-oxygen use differ between sea level and altitude.[32] Prescribing oxygen without considering the altitude of residence is, quite simply, incomplete.

Home non-invasive ventilation may benefit selected patients with chronic hypercapnia, especially after an exacerbation. In the older patient, the decision weighs device burden, cognition, home support, and goals of care, so the technical indication is not divorced from the geriatric assessment.

PHARMACOLOGICAL TREATMENT IN THE OLDER ADULT

COPD pharmacotherapy follows the GOLD ABE scheme, but each decision in the older adult weighs multimorbidity, polypharmacy, renal and hepatic function, adherence, and device usability. The treatable-traits framework (consistent with GETomics) is especially useful: rather than one algorithm, identify what is modifiable (dyspnea, exacerbations, eosinophilic type 2 inflammation) and target the drug to that trait, weighing the benefit–risk of advanced age (Table 1).

From the ABE scheme to individualized decisions

GOLD 2026 proposes initial treatment by group (A, B, or E) and follow-up by the dominant trait: bronchodilation if dyspnea predominates; the anti-inflammatory

Table 1. Drugs in COPD and precautions in the older adult

Class or drug	Role	Precautions in the older adult
LABA	Bronchodilation	Tachycardia, arrhythmia, tremor, hypokalemia; cardiovascular caution
LAMA	Bronchodilation	Anticholinergic burden: cognitive impairment, urinary retention, glaucoma, dry mouth, constipation
LABA/LAMA	Dual bronchodilation (base)	Combines the above precautions; usually the preferred base
ICS (in combination)	Anti-inflammatory according to eosinophils	Pneumonia (IMPACT,[34] ETHOS[35]), fractures/ osteoporosis, cataracts, hyperglycemia, oral candidiasis
Triple (LABA/LAMA/ICS)	Eosinophilic exacerbator	Reduces exacerbations and hospitalization (IMPACT,[34] ETHOS[35]) against pneumonia risk; single inhaler improves adherence
Dupilumab (anti–IL-4Rα)	Biologic, Type 2 inflammation	Subcutaneous, well tolerated; add-on in the eosinophilic patient (BOREAS,[37] NOTUS[38])
Mepolizumab (anti–IL-5)	Biologic, eosinophilic	Fewer exacerbations with eos ≥ 300 on triple therapy (MATINEE[39])
Ensifentrine (PDE3/4)	Nebulized bronchodilator and anti-inflammatory	Good tolerability (ENHANCE[40]); consider nebulized route and cost
Roflumilast (oral PDE4)	Chronic bronchitis with exacerbations	Nausea, diarrhea, and weight loss: problematic in the frail or sarcopenic patient
Azithromycin	Reduces exacerbations	QT prolongation, ototoxicity, bacterial resistance

BOREAS and NOTUS: dupilumab trials in COPD; COPD: chronic obstructive pulmonary disease; ENHANCE: ensifentrine trials; eos: blood eosinophil count; ETHOS and IMPACT: triple-therapy trials; ICS: inhaled corticosteroid; IL: interleukin; LABA: long-acting β_2-agonist; LAMA: long-acting muscarinic antagonist; MATINEE: mepolizumab trial; PDE: phosphodiesterase; QT: QT interval on the electrocardiogram.

component per eosinophils if exacerbations predominate. The aim is not only to escalate – de-escalating and simplifying matter equally – and a single exacerbation may prompt reconsideration, individualized.

Read through a geriatric lens, much of GOLD remains applicable: spirometric confirmation, the emphasis on symptoms and exacerbation risk, smoking cessation, vaccination, rehabilitation, and long-acting bronchodilators as the backbone. What needs adaptation is its execution: goals shift toward function, symptom control, and quality of life; escalation is tempered by frailty, comorbidity, and polypharmacy; inhaled corticosteroids demand a conservative, eosinophil- and risk-weighted judgment; device and dose must match cognitive and manual ability; and de-escalation matters as much as escalation.

Bronchodilators: efficacy and safety in the older adult

Long-acting bronchodilators (LABA and LAMA, ideally combined) are the mainstay, with efficacy maintained in the geriatric patient. β_2-agonists can cause tachycardia, tremor, hypokalemia, and arrhythmias, so caution is warranted with concomitant heart disease (the cardiopulmonary axis). Antimuscarinics, though acting locally, add to the cumulative anticholinergic burden – cognitive impairment, urinary retention, glaucoma, dry mouth, constipation. The cardiovascular profile at therapeutic doses is reasonably reassuring, but the total anticholinergic load across the whole regimen must be monitored, since cumulative strong-anticholinergic use raises dementia risk.[33]

Inhaled corticosteroids: guided by eosinophils, weighing the risks in the older adult

Inhaled corticosteroids (ICS) benefit exacerbators with eosinophilia, but in the older patient must be weighed against risks that fall harder here: pneumonia (IMPACT hazard ratio 1.53 vs. LAMA/LABA; ETHOS confirmed pneumonia 3.5-4.5% with ICS vs. 2.3%),[34,35]

plus osteoporosis and fractures, cataracts, skin fragility, hyperglycemia, and candidiasis. Guide use by eosinophils and exacerbation history, and consider withdrawal when eosinophils are low and exacerbations absent; gradual withdrawal did not increase exacerbations, though FEV_1 fell somewhat more.[36]

Triple therapy (LABA/LAMA/ICS)

Two large trials support single-inhaler triple therapy: in IMPACT, it reduced moderate or severe exacerbations and hospitalizations versus dual therapies, at the cost of higher pneumonia risk;[34] in ETHOS, it outperformed both duals, with a mortality signal at the higher budesonide dose.[35] In the older adult, it is reserved for the eosinophilic exacerbator, weighing pneumonia risk; the single inhaler favors adherence.

Targeted treatments: biologics

Type 2 (eosinophilic) inflammation is a treatable trait amenable to subcutaneous monoclonals, attractive in the older patient for their tolerability as add-on therapy. Dupilumab (anti–IL-4Rα) reduced exacerbations in eosinophilic patients in BOREAS and NOTUS;[37,38] mepolizumab (anti–IL-5) did so in MATINEE with eosinophils ≥ 300 cells/μL on triple therapy.[39] They are the most refined application of treatable traits in the refractory eosinophilic exacerbator.

Other options: ensifentrine, roflumilast, and azithromycin

Ensifentrine, a nebulized dual PDE3/PDE4 inhibitor, is the first new inhaled class in over a decade, improving lung function and reducing exacerbations in ENHANCE with good tolerability,[40] though route and cost are constraints. Roflumilast (oral PDE4) helps in chronic bronchitis with exacerbations, but its gastrointestinal effects and weight loss are problematic in the frail patient. Chronic azithromycin reduces exacerbations but requires monitoring for QT prolongation, ototoxicity, and resistance.

Adherence, inhaler technique, and device selection

Device selection is decisive and often neglected, matching the patient's cognition, dexterity, and inspiratory flow: dry-powder inhalers need a flow that may be lacking in frailty; pressurized inhalers need coordination (compromised by cognitive impairment) and benefit from a spacer; soft-mist inhalers or nebulization suit the very limited patient. Simplify the regimen, recheck technique repeatedly, and remember that adherence declines with polypharmacy and cognitive impairment.

Deprescribing and polypharmacy

The counterpart of treatment is deprescribing – systematically reviewing the whole regimen to withdraw what is inappropriate, beyond de-escalating one drug. Tools such as STOPP/START help detect inappropriate drugs and omissions of beneficial treatment.[41] In COPD, this means withdrawing ICS when it adds nothing (low eosinophils, no exacerbations), reducing the anticholinergic burden, and periodically reviewing each drug against the goals of care. Simplifying improves adherence and reduces adverse events.

Relevant drug interactions in the older adult

Inhaled therapy has little systemic-interaction potential; the problem lies in oral drugs and those for comorbidities. Beyond the anticholinergic burden already noted, the main interactions are in Table 2 (QT with azithromycin, fluticasone with CYP3A4 inhibitors, and additive hypokalemia from systemic corticosteroids). One detail the table omits: theophylline – narrow-margin, metabolized by CYP1A2 – rises on smoking cessation (also relevant to clozapine and olanzapine). Cardioselective β-blockers do not worsen lung function and should not be stopped when there is a cardiovascular indication,[42] but should not be started to treat COPD, since without such an indication, they do not prevent exacerbations and may increase hospitalizations.[43]

EXACERBATIONS IN THE OLDER ADULT

Exacerbations accelerate functional decline, impair quality of life, consume resources, and carry mortality. In the older patient, they are more dangerous still, occurring on diminished physiological reserve and a background of multimorbidity.

Recognition and classification

GOLD 2026 classifies exacerbation severity by objective parameters (dyspnea intensity, respiratory

Table 2. Relevant drug interactions in the older adult with COPD

Drug (COPD)	Interacts with	Effect or risk	Action
LAMA and anticholinergic burden	Other anticholinergics (antihistamines, tricyclics, bladder antimuscarinics, antipsychotics)	Additive anticholinergic burden: cognition, falls, urinary retention, glaucoma	Sum the whole regimen's burden; deprescribe
Chronic azithromycin	Other QT-prolonging agents (antipsychotics, antidepressants, antiarrhythmics, fluoroquinolones)	Additive QT prolongation, torsade risk	Monitor QTc, K^+/Mg^{2+}; avoid risky combinations
Theophylline	Macrolides, ciprofloxacin; smoking cessation	Narrow margin; ↑ levels and toxicity; quitting smoking raises levels (CYP1A2)	Avoid if possible; monitor levels; readjust on cessation
Roflumilast	Strong CYP3A4 and 1A2 inhibitors/inducers	Changes in exposure; gastrointestinal and mood effects	Caution; monitor tolerance and weight
Systemic corticosteroid	β_2-agonists, diuretics; antidiabetics; NSAIDs	Additive hypokalemia (arrhythmia, digoxin toxicity); hyperglycemia; GI bleeding	Short courses; monitor K^+ and glycemia; gastric protection if NSAID
Fluticasone (ICS)	Strong CYP3A4 inhibitors (ritonavir, ketoconazole)	Adrenal suppression, iatrogenic Cushing	Avoid the combination or switch ICS
Non-selective β-blocker	β_2-agonists	Antagonism of the bronchodilator effect	Prefer a cardioselective agent
Cardioselective β-blocker	—	Does not worsen FEV_1; keep if there is a cardiovascular indication; do not start to treat COPD	Keep if indicated;[42] do not use for COPD without a CV indication[43]

COPD: chronic obstructive pulmonary disease; CYP: cytochrome P450; FEV_1: forced expiratory volume in the first second; GI: gastrointestinal; ICS: inhaled corticosteroid; K^+: potassium; LAMA: long-acting muscarinic antagonist; Mg^{2+}: magnesium; NSAID: non-steroidal anti-inflammatory drug; PDE: phosphodiesterase; QTc: corrected QT interval.

and heart rate, saturation, and markers such as C-reactive protein or blood gases), beyond the classic treatment-escalation criterion. In the older adult, the presentation may be atypical – confusion, falls, acute functional decline, or decompensation of a comorbidity rather than the "typical" increase in dyspnea and sputum – mandating careful differential diagnosis with pneumonia, heart failure, pulmonary embolism, and acute coronary syndrome.

The exacerbation-cardiovascular event axis

GOLD 2026 emphasizes that an exacerbation is not a purely pulmonary event: over the following days and weeks, the risk of cardiovascular events – myocardial infarction, stroke, heart failure, arrhythmias – rises significantly,[4] mainly in the first 30 days and far more after hospitalization,[44] with documented increases in myocardial infarction and stroke.[45] In the multimorbid older patient, whose cardiopulmonary axis is already strained, this means treating the exacerbation as a high-risk systemic event.

Treatment and risks of systemic corticosteroids in the older adult

Treatment relies on intensifying bronchodilators, short courses of systemic corticosteroids, antibiotics when bacterial infection is evident, and oxygen (target 88-92%) or non-invasive ventilation in hypercapnic failure. Systemic corticosteroids warrant special caution in the older adult – hyperglycemia, delirium, insomnia, fluid retention, muscle weakness, falls – so the lowest effective dose for the shortest time is preferred: in practice, oral prednisone 40 mg/day for 5 days, non-inferior to 14-day courses.[46] Higher doses or longer courses add toxicity without benefit, and corticosteroids are best reserved for moderate-to-severe exacerbations.

After the exacerbation: prevention and care transitions

The period after an exacerbation carries a high risk of relapse, readmission, and death. It is the time to optimize maintenance by the dominant trait, reinforce vaccination, start or resume rehabilitation (which reduces readmissions), reconcile medication, and address frailty. A well-planned care transition – clear instructions, medication reconciliation, early follow-up – is among the most effective ways to break the cycle of exacerbation, decline, and readmission.

ADVANCED COPD, PALLIATIVE CARE, AND ADVANCE CARE PLANNING

Advanced COPD in the older adult brings a high symptom burden (especially refractory dyspnea), progressive functional decline, recurrent exacerbations, and growing proximity to the end of life. The palliative approach should not wait for the final days but be integrated early, alongside disease-directed treatment, guided by needs rather than an exact prognosis – especially pertinent in the multimorbid older patient.

Recognizing advanced COPD and the need for a palliative approach

Several markers identify advanced disease: severe airflow limitation, frequent exacerbations and hospitalizations, refractory dyspnea despite optimized treatment, cachexia or low body mass index, frailty, and dependence. The key is to activate the palliative approach by symptomatic and functional need, without waiting for the terminal phase.

Refractory dyspnea and symptom control

When dyspnea persists despite optimized treatment, management combines non-pharmacological measures and, per guidelines, low-dose opioids.[4] In the older adult, opioids are titrated cautiously (constipation, sedation, falls), but their benefit can be notable: low-dose sustained-release oral morphine relieves refractory dyspnea[47] and improves health status without worsening $PaCO_2$.[48] Anxiety, cough, secretions, and fatigue must also be addressed.

The Latin American reality: the access abyss for opioids and palliative care

Here, the regional perspective is unavoidable. The Lancet Commission on Palliative Care and Pain Relief documented an enormous "access abyss" to the relief of suffering, with unmet need for oral morphine – low-cost and off-patent – concentrated in low- and middle-income countries.[49] Latin America has restricted opioid access owing to regulatory barriers, low consumption, fear of diversion, few trained professionals, and coverage gaps, especially rural. The consequence is that refractory dyspnea in advanced COPD is frequently undertreated. Closing this gap – strengthening palliative care and ensuring safe opioid access – is an imperative of equity and an indispensable part of geriatric COPD care in the region. The burden is also psychological and familial (anxiety, depression, caregiver distress), aggravated by out-of-pocket spending.[3]

Advance care planning

Advance care planning means timely conversations about the patient's values, goals, preferences, and treatment limits (e.g., non-invasive ventilation, intensive care, intubation), and designating a surrogate. It should start early, while cognition allows, within shared decision-making. In the Latin American context, where decisions are markedly family-centered, involve the family while respecting patient autonomy, avoiding both overtreatment and abandonment.

End of life

In the final phase, the goal is well-being: control of terminal dyspnea, deprescribing of drugs that no longer help, psychosocial and spiritual support, and caregiver attention. Continuity of care and, when available, home or inpatient palliative care allow a dignified, accompanied death – an essential part of COPD care in a rapidly aging region with unequal palliative resources.

EMERGING TECHNOLOGY AND REGIONAL CONSIDERATIONS

Emerging technology

GOLD 2026 adds a chapter on emerging technology. Telemedicine, remote monitoring, and artificial-intelligence tools for detection and risk stratification hold particular potential in the geriatric patient, but

their deployment must reckon with the digital and access divide, marked across much of Latin America.

The regional reality: unequal access

Thinking about geriatric COPD in Latin America requires recognizing that access – to quality spirometry, oscillometry, biologics, opioids for refractory dyspnea, and correctly prescribed oxygen – is profoundly unequal. Clinical knowledge is necessary but insufficient without equity of access, perhaps this review's most important regional message. Oscillometry illustrates it: effort-independent, it is a realistic alternative where quality spirometry is hardest to obtain, and widening its availability is itself a measure of equity.

CONCLUSION

COPD in the older patient is a distinct entity, defined by multimorbidity, frailty, and a life trajectory known only in its final stretch. Reading it through GETomics and the trajectome makes sense of its heterogeneity and recovers an active stance centered on what remains modifiable, and the GOLD 2026 developments cohere once coupled with comprehensive geriatric assessment. For the Latin American clinician, this reading must take root in the region's reality – accelerated aging, biomass and tuberculosis, high-altitude populations, and unequal access. Diagnosing prudently, assessing comprehensively, treating by treatable traits with the caution advanced age demands, accompanying to the end, and doing so with equity: that is the approach this review proposes.

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THE JOURNAL OF LATIN AMERICAN GERIATRIC MEDICINE REVIEW ARTICLE

Social cognition and aging: an emerging biomarch in geriatrics

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Abstract

Social cognition is the integration of various cognitive processes that enable us to interact with others in society through the exchange of non-verbal cues, such as facial expressions of emotions, voice characteristics, and body posture. This review article analyzes the scientific literature on changes in social cognition associated with normal and pathological aging, including mild cognitive impairment and dementias. The findings show a consistent pattern of deterioration in specific components of social cognition, with progression varying according to the aging trajectory. However, the review also describes significant methodological limitations in existing studies, inconsistencies in assessment instruments, and controversies regarding the independence of these deficits from general cognitive decline. The clinical implications are numerous, such as the role of social cognition in identifying early cognitive and behavioral changes that can help explain interpersonal difficulties not captured by conventional cognitive tests, strengthening the clinical differential diagnosis between normal aging, mild cognitive impairment, and major cognitive impairment in its various forms, and improving our understanding of symptoms, such as apathy, disinhibition, loss of empathy, social isolation, or vulnerability to abuse and exploitation among older adults.

Keywords: *Social cognition. Theory of mind. Executive functions. Mild cognitive impairment. Neurodegenerative disorders.*

Cognición social: indicador clínico emergente del deterioro cognitivo en pacientes geriátricos

Resumen

La cognición social es la integración de varios procesos cognitivos que nos permiten interactuar con los demás en la sociedad, mediante el intercambio de estímulos corporales como la expresión facial de emociones, caractisticas de la voz, postura corporal, etc. Este artículo de revisión realiza un análisis de la literatura científica sobre los cambios de la cognición social asociados al envejecimiento normal y patológico, incluyendo el deterioro cognitivo leve y las demencias. Los hallazgos muestran un patrón consistente de deterioro en componentes específicos de la cognición social, con una progresión diferente según la trayectoria del envejecimiento. Sin embargo, también se describe importantes limitaciones metodológicas en los estudios existentes, inconsistencias en los instrumentos de evaluación y controversias sobre la independencia de estos déficits respecto al declive cognitivo general. Las implicaciones clínicas son múltiples, como la contribución de la cognición social en la identificación de cambios cognitivo conductuales tempranos que permitan explicar dificultades interpersonales no captadas por pruebas cognitivas convencionales, fortalecer el diagnóstico clínico diferencial entre envejecimiento normal, deterioro cognitivo leve y deterioro cognitivo mayor en sus diferentes variantes. y mejorar la comprensión de síntomas como apatía, desinhibición, pérdida de empatía, aislamiento social o vulnerabilidad a abuso y explotación del adulto mayor.

Palabras clave: *Cognición social. Teoría de la mente. Funciones ejecutivas. Deterioro cognitivo leve. Trastornos neurodegenerativos.*

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INTRODUCTION

Social cognition is the integration of various cognitive processes that allow us to interact with others in society by exchanging non-verbal cues, such as facial expressions of emotions, voice characteristics, and body posture, to construct mental representations of others cognitive and emotional states,[1] which enables us to prepare an appropriate response that maintains order in interpersonal relationships and allows us to function properly in society.[2]

It represents one of the six cognitive domains included in the DSM-5 for the diagnosis of neurocognitive disorders;[3] these processes are fundamental to successful communication, requiring the proper functioning of cognitive processes, such as the perception, interpretation, and processing of information from social cues.[4]

Population aging has highlighted the need to better understand the decline and preservation of multiple functions, including those related to neurocognitive changes.[1] Social cognition, for example, is a cognitive-emotional process that also changes with age, leading to different trajectories in normal and pathological aging,[5] with consequences, such as social isolation, vulnerability to financial exploitation, increased caregiver burden, and pre-mature institutionalization.[6]

Despite this formal inclusion in the literature, social cognition is not routinely measured in geriatric assessments; unlike other cognitive domains.[1] This review examines the present evidence on social cognition in geriatrics.

LITERATURE SEARCH AND SELECTION METHODOLOGY

A critical narrative review was conducted using a systematic search of the scientific literature on social cognition in normal and pathological aging.[7] The main research question was: what is the available evidence regarding changes in social cognition during aging, and what is its utility as an emerging clinical marker in geriatrics?

The search was conducted in PubMed/MEDLINE, Scopus, Web of Science, PsycINFO, ScienceDirect, and Google Scholar. Combinations of the following terms in English and Spanish were used: "social cognition," "theory of mind," "emotion recognition," empathy, "social perception," aging, "older adults," elderly, geriatric, "mild cognitive impairment," dementia, "Alzheimer's disease," "frontotemporal dementia," "Parkinson's disease."

Articles published between 2000 and 2026, in English and Spanish, that addressed the topic of social cognition in healthy older adults, those with mild cognitive impairment, or those with neurodegenerative disorders were included. Original studies, systematic reviews, meta-analyses, instrument validation studies, and clinical trials were included.

Studies focusing exclusively on pediatric populations, adolescents, or young adults were excluded, as were investigations into non-geriatric psychiatric disorders unrelated to aging; editorials, letters to the editor, non-peer-reviewed gray literature, and duplicate studies.[8]

Study selection was conducted through a review of titles and abstracts, followed by a full reading of potentially eligible articles. Data on the author, year, country, study design, population, sample size, domain of social cognition assessed, instrument used, main findings, limitations, and clinical relevance were extracted from each study. The evidence was organized into thematic areas: conceptualization of social cognition, neurobiological bases, assessment instruments, normal aging, mild cognitive impairment, dementias, and clinical implications. The synthesis was narrative and comparative.[9]

COMPONENTS OF SOCIAL COGNITION

Social cognition is a multidimensional construct of mental processes that enable individuals to perceive, interpret, and respond appropriately to social cues from others; as such, it is organized into several domains with the following functional sequence:[2]

- Social perception: this involves the detection of relevant external social cues, such as the face, gestures, gaze, body posture, interpersonal distance, tone of voice, and social and cultural context.[5]
- Emotional processing: once we have perceived a social stimulus, the brain interprets its emotional content by recognizing facial emotions, recognizing emotional prosody, distinguishing between emotions, such as joy, sadness, anger, surprise, fear, and disgust, and assessing threat or safety.[10]
- Social cognition: the brain does not interpret an emotion in isolation but integrates it into a social context, taking into account social norms,

interpersonal relationships, culture, the environmental situation, social roles, prior emotional history, and expected behavioral rules.[5]

- Theory of mind (ToM): this refers to the brain's ability to infer the mental states and emotions of other individuals, including beliefs, desires, intentions, and knowledge. It is divided into cognitive ToM (inferring beliefs and thoughts) and affective ToM (inferring affective and emotional states).[11]
- Empathy: the ability to understand and emotionally resonate with another person's experience, making it possible to share and feel emotions with them; it consists of cognitive empathy (understanding others' emotions without experiencing them oneself) and affective empathy (sharing in and resonating with others' emotional states).[12]
- Attributional style: at this stage, the brain decides how to interpret another person's behavior, explaining why they acted in a certain way, that is, it attributes intention, blame, or hostility, and makes a neutral, benign, or negative interpretation.[13]
- Executive regulation of the social response: before responding to the received social stimulus, the response is regulated through impulse inhibition, cognitive flexibility, response planning, and evaluation of consequences.
- Decision-making and social behavioral response: the process concludes here with a verbal or nonverbal response, prosocial behavior, avoidance, confrontation, or social adaptation.[14]

NEUROBIOLOGICAL BASES

Social cognition involves structural bases and neural networks distributed across different brain regions that work together and are activated in response to specific stimuli. These include the medial prefrontal cortex, the dorsolateral prefrontal cortex, the anterior cingulate cortex, the hippocampus, the amygdala, the anterior insula, the inferior frontal gyrus, and the superior temporal sulcus.[15]

Four main neural systems are described:

- Social perception network: this comprises the lateral occipitotemporal cortex, which responds to body images; the fusiform face area, responsible for the perception of facial expressions; and the prefrontal cortex, which identifies and evaluates the order of importance of social stimuli, as well as interprets eye contact and body posture.[11]
- Empathy network: composed of structures in the anterior cingulate cortex, the amygdala, the insula, the prefrontal cortex, and somatosensory areas that support the generation of responses to emotional experiences.[16]
- Mirror neuron system: composed of the inferior frontal gyrus, the inferior parietal lobe, the fusiform face area, and the superior temporal sulcus; it is activated to simulate the actions and intentions of others.[17]
- Mentalization network: consisting of the medial prefrontal cortex, which is associated with the inference of mental states; the temporoparietal junction, which is related to the understanding and attribution of mental states; and the superior temporal sulcus, which is involved in the processing of social actions. Among the brain areas involved in decision-making, the ventromedial and dorsolateral prefrontal cortices stand out, as well as the anterior cingulate cortex.[18]

Table 1 describes the main neurobiological networks involved in social cognition and their clinical relevance.

SOCIAL COGNITION ASSESSMENT TOOLS

The assessment of social cognition in geriatrics requires instruments capable of exploring specific domains associated with each of the functional sequences. However, a significant limitation is that many tests were originally developed in young adult populations or in different cultural contexts; therefore, their application in older adults requires consideration of variables, such as age, education, language, cognitive impairment, and sociocultural context.[19]

Several instruments are available to assess social cognition in older adults across its various domains:

- Reading the mind in the eyes test (RMET): assesses the ability to perceive others' emotions and intentions based on images showing only the eyes. The test consists of 36 photos, and participants must choose one of four response options for each image. The final score ranges from 0 to 36 points, depending on the number of correct responses.[20] This test has been validated for older adults, and abbreviated versions have been developed, such as the RMET-10 (a 10-item

Table 1. Brain networks related to social cognition

Neurobiological network	Main regions	Social function	Changes due to aging or disease
Amygdala-limbic network	Amygdala; orbitofrontal cortex	Emotion recognition and detection of social salience	Impaired recognition of fear, sadness, and negative emotions
Mentalization network	Medial prefrontal cortex; temporo-parietal junction; posterior superior temporal sulcus	Inference of beliefs, intentions, and mental states	Impaired theory of mind in aging, Alzheimer's disease, and frontotemporal dementia
Empathy network	Anterior insula; anterior cingulate cortex	Emotional resonance and affective empathy	Loss of empathy, especially in frontotemporal dementia
Mirror neuron system	Inferior frontal gyrus; inferior parietal lobe; superior temporal sulcus	Understanding of actions, imitation, and social learning	Impaired interpretation of gestures, facial expressions, and social behavior

Source: Love et al.[5]

abbreviated version), which follows a normal distribution in older adult populations, with scores influenced by age, education, race, and sex.[21]

- Edinburgh social cognition test: this is a comprehensive battery that assesses affective and cognitive ToM, as well as associated behavioral changes.[22]
- Brief assessment of social skills-dementia: a screening tool designed for people with dementia that assesses emotion recognition, empathy, social reasoning, and related skills.[23]
- Mini-social cognition and emotional assessment: this is a battery of tests that assesses emotion recognition and ToM, with validity for normal aging, mild cognitive impairment, and Alzheimer's disease (AD).[24]

No single instrument comprehensively assesses all domains of social cognition, and from a geriatric perspective, the choice of instrument should depend on the clinical objective: screening, differential diagnosis, longitudinal follow-up, or research. Furthermore, results should be interpreted while taking into account the patient's overall cognitive status, educational background, language, the presence of affective symptoms, and cultural context.[25] Table 2 summarizes the most commonly used instruments for assessing social cognition in older adults.

FACTORS INFLUENCING CHANGES IN SOCIAL COGNITION DURING AGING

Social cognitive performance in older adults is influenced by multiple factors:[26]

- Demographic factors: age, sex, educational level, and socioeconomic status (approximated by national income) contribute significantly to variability in performance.[1,5] In a population-based study using the RMET-10 test, scores were significantly higher among younger individuals, those with higher education, white individuals, and those with less cognitive impairment. Women outperformed men only in the 65-74 age group.[27]
- Cognitive functions: cognitive functions and educational level consistently emerged as the primary predictors of social cognition across multiple models. Language and complex attention were the best predictors in other groups; however, not all of the variance in social cognition performance was explained by variance in cognitive domains.[18]
- Structural brain reserve: although functional magnetic resonance imaging brain networks did not show significant predictive value, head movements during scanning contributed significantly to emotion recognition, highlighting the importance of considering technical artifacts in neuroimaging studies.[28]
- Affective symptoms: RMET-10 scores were significantly higher in individuals with fewer symptoms

Table 2. Primary instruments for assessing social cognition in older adults

Instrument	Primary domain	Clinical utility	Strengths	Limitations
RMET	Theory of mind and cognitive empathy	Assesses the ability to infer mental states from eye contact	Brief, widely used, sensitive to subtle deficits	Influenced by education level, language, culture, and visual abilities
ESCoT	Cognitive and affective theory of mind, social norms	Assesses complex social situations through scenarios	Multidimensional and ecological approach	Longer administration time; limited use in older adults
Mini-SEA	Theory of mind and emotional recognition	Useful in the differential diagnosis of dementias, especially bvFTD	High sensitivity for behavioral variant frontotemporal dementia	Cross-cultural norms are still limited
BASS-D	Emotional recognition, empathy, and social reasoning	Brief screening of social cognition skills	Practical for clinical settings	Requires further validation

RMET: reading the mind in the eyes test; ESCoT: Edinburgh social cognition test; SEA: social cognition and emotional assessment; BASS-D: brief assessment of social skills–dementia; bvFTD: behavioral variant frontotemporal dementia.

of anxiety and depression, even after adjusting for demographics,[1] suggesting that affective state modulates social cognition performance.[20]

PATTERNS OF SOCIAL COGNITION ASSOCIATED WITH NORMAL AGING

Aging is associated with a loss of cortical and subcortical volume in all brain regions, particularly the frontal regions, leading to impaired performance, especially in executive functions and tasks requiring processing speed.[29]

A systematic review on facial emotion recognition in aging found that older adults show a decline, primarily for negative emotions.[30] This pattern can be explained both by structural theories and by the theory of socioemotional selectivity, which posits that older adults prioritize emotionally positive information as an adaptive strategy.[31] This implies that an older adult may easily identify a friendly or happy expression but may have more difficulty detecting subtle signs of threat, annoyance, sadness, or distrust in another person.[32]

With regard to ToM – which depends on the prefrontal cortex and influences working memory, cognitive flexibility, and inhibition – aging is associated with slight changes, particularly in complex tasks, such as interpreting irony, understanding double meanings, and recognizing false beliefs.[33]

Both affective and cognitive empathy do not show uniform changes; with aging, emotional sensitivity toward others increases, while cognitive empathy shows difficulty in understanding complex social situations.[32]

Social perception undergoes changes over the years, with greater difficulty in picking up on social cues, leading older adults to misinterpret conversations and intentions.[33]

Social emotional regulation in older adults shows better emotional regulation than in younger people, as they tend to focus on positive emotions and avoid unnecessary conflicts, which leads them to try to maintain important emotional bonds.[34]

PATTERNS OF SOCIAL COGNITION ASSOCIATED WITH COGNITIVE DECLINE

Mild cognitive impairment is associated with significant deficits in social cognition, although these are of lesser magnitude than in established dementias. Deficits in social cognition do not manifest uniformly across all neurodegenerative diseases.[35] AD, Parkinson's disease, and frontotemporal dementia can impair ToM, emotional recognition, empathy, and social reasoning, but with varying intensity, timing of onset, and diagnostic relevance.[36] Table 3 summarizes the most relevant clinical patterns of social cognition impairment in these disorders.

A meta-analysis of 17 studies found that mild cognitive impairment is associated with significant impairments in ToM (d = 0.63) and facial emotion recognition

Table 3. Profile of social cognition impairment by neurodegenerative disorder

Domain	Alzheimer's disease	Parkinson's disease	Behavioral variant of frontotemporal dementia
Theory of mind	Mild or moderate impairment, most evident in complex tasks	May be impaired even without dementia	Severe and early impairment
Emotional recognition	Decreased recognition of negative emotions	Impaired recognition of facial emotions	Global and profound impairment
Empathy	Gradual decline, secondary to cognitive impairment	Decreased affective empathy	Marked loss of affective and cognitive empathy
Social perception	Early subtle deficits with progression	Impaired interpretation of social cues	Severe impairment in interpersonal behavior
Moral/social reasoning	Preserved in early stages	Impaired complex social judgment	Markedly impaired
Clinical significance	Contributes to isolation and caregiver burden	Associated with executive dysfunction, apathy, and behavioral changes	A central clinical feature useful for differential diagnosis

Source: Setién-Suero et al.[38]

(d = 0.58). Recognition of fear and sadness was particularly affected, while recognition of disgust, happiness, and surprise showed no significant differences. Sociocognitive deficits were more severe in multidomain mild cognitive impairment.[37]

A more recent meta-analysis that included 28 cross-sectional studies with 2,368 participants found that individuals with mild cognitive impairment exhibited better emotion recognition (Cohen's d = 0.69) and ToM (d = 0.74) compared to those with AD dementia. These findings suggest a progressive decline in social cognition along the continuum from mild cognitive impairment to major cognitive impairment.[37]

AD

AD presents a pattern characterized by social cognition deficits that are less evident at the onset, with impairments in ToM observed primarily in complex tasks.[38]

Emotional processing shows that the recognition of negative emotions is frequently impaired; deficits in social perception also emerge with complex tasks, and attributional bias is observed, particularly when inferring age.[37]

Deficits in social cognition in AD are often secondary to other cognitive declines rather than being primary impairments of social cognition. However, social cognition appears to be partially independent of other cognitive domains, which justifies its inclusion in diagnostic batteries for a complementary approach to dementia.[38]

FRONTOTEMPORAL DEMENTIA

Frontotemporal dementias encompass several neurodegenerative diseases; the most common is the frontal variant, which is characterized by changes in social and interpersonal behavior and a lack of awareness of the disease itself. The behavioral variant of frontotemporal dementia presents the most severe and early-onset profile of social cognitive impairment.[39]

In this condition, loss of empathy, disinhibition, inappropriate social behavior, and difficulty interpreting intentions or social norms are the main manifestations of the clinical picture.[40] Empathic deficits are considered a diagnostic criterion for this condition and may lead to changes in patients' daily lives, particularly in decision-making and judgment.[41] Moral judgments are primarily assessed through moral dilemma stories; therefore, patients with frontotemporal dementia are unable to make decisions, acting without considering the moral and emotional dimensions of their actions.[39]

It appears that all social cognition processes are affected in behavioral variant frontotemporal dementia, leading to an inability for patients to communicate with those around them; therefore, addressing social cognition disorders is essential in these patients.[42]

PARKINSON'S DISEASE

In Parkinson's disease, there is degeneration of dopaminergic neurons in the substantia nigra, the striatum, the thalamus, and the subthalamic nuclei. Within its broad spectrum of symptoms, most patients experience executive dysfunction, and approximately 80% develop dementia.[43] However, social deficits may appear even before the development of major cognitive impairment, particularly in relation to ToM, emotional recognition, and affective empathy; these changes may be modulated by executive dysfunction, apathy, depressive symptoms, and alterations in fronto-striatal circuits.[44]

DISCUSSION

This review describes how social cognition is one of the neurocognitive domains of great importance in geriatrics, both because of its inclusion in neurocognitive disorders and because of its association with interpersonal functioning, social behavior, autonomy, and quality of life in older adults. Unlike other cognitive domains commonly assessed, social cognition is an under-explored area in geriatric practice; it would be of great relevance given the behavioral changes and variations, difficulties in social interaction, loss of empathy, errors in emotional interpretation, and increased psychosocial vulnerability present in aging across its various trajectories.

One key point to highlight in this review is that social cognition should not be understood as a single cognitive function, but rather as a multidimensional construct composed of several sequential and interrelated processes, including social perception, emotional recognition, social knowledge, ToM, empathy, attribute style, executive regulation of social responses, and social decision-making. This helps us understand that the social changes observed in older adults do not stem from a single area but rather from various neural networks that may be compromised depending on age, cognitive reserve, affective state, educational attainment, sociocultural context, and the presence of neurodegenerative diseases.

The reviewed evidence shows that, in normal aging, changes in social cognition are heterogeneous. Some components, such as emotional regulation and orientation toward positive stimuli, may remain relatively intact or even exhibit adaptive patterns. Other domains, such as the recognition of negative emotions and ToM in complex tasks, typically show an age-related decline. These changes result from the interaction between neurobiological changes in frontotemporal and limbic networks and motivational mechanisms characteristic of aging, such as the prioritization of emotionally significant relationships and the regulation of negative affective experiences. Therefore, social aging should not be interpreted exclusively as deterioration, but rather as a reorganization of cognitive, emotional, and motivational abilities.

In mild cognitive impairment, the literature points to the presence of significant alterations in ToM and emotional recognition, though to a lesser extent than in established dementias. This finding is clinically relevant; as it allows us to consider the possibility that social cognition may serve as a complementary marker in the early stages of cognitive decline, especially in subgroups of patients for whom memory impairments are not the predominant symptom. However, the diagnostic utility of these deficits still requires further longitudinal studies to determine how socio-cognitive alterations predict progression to dementia, functional decline, or an increased caregiver burden.

Neurodegenerative disorders exhibit distinct patterns of impairment in social cognition. In behavioral variant frontotemporal dementia, empathy, ToM, social judgment, and the regulation of interpersonal behavior constitute a primary and early clinical feature, with very high diagnostic value in the differential diagnosis. In AD, changes are typically more subtle in the early stages and become more evident as impairment progresses, particularly in tasks requiring greater executive function. In Parkinson's disease, difficulties with emotional recognition, affective empathy, and ToM may appear even in the absence of major cognitive impairment, due to fronto-striatal dysfunction, affective symptoms, apathy, and executive impairment. This variability in changes allows us to emphasize that social cognition provides distinct clinical information depending on the neurodegenerative profile.

From a neurobiological perspective, social cognition depends on distributed networks that include the amygdala, the anterior insula, the anterior cingulate cortex, the medial prefrontal cortex, the orbitofrontal cortex, the temporoparietal junction, the superior temporal sulcus, and regions associated with the mirror neuron system. The vulnerability of these networks during aging and in neurodegenerative diseases provides a basis for explaining the observed changes in social behavior.

With regard to clinical assessment, this review shows that there are various instruments for exploring specific domains of social cognition; however, none of them comprehensively assesses all components, and many have limitations related to educational level, language, cultural context, and sensitivity and specificity for the geriatric population, which represents a barrier to their systematic incorporation into comprehensive geriatric assessment. Consequently, the choice of instrument depends on the clinical objective: screening, differential diagnosis, longitudinal follow-up, behavioral characterization, or research.

Assessing social cognition in older adults can help identify early cognitive and behavioral changes, explain interpersonal difficulties not captured by conventional cognitive tests, guide psychoeducational interventions for caregivers, and improve understanding of symptoms, such as apathy, disinhibition, loss of empathy, social isolation, or vulnerability to abuse and exploitation. Furthermore, its incorporation can strengthen the clinical differential diagnosis between normal aging, mild cognitive impairment, and major cognitive impairment in its various forms.

Future research should focus on longitudinal studies that evaluate the predictive value of social cognition in the progression from mild cognitive impairment to dementia, as well as its relationship with functionality, institutionalization, caregiver burden, and quality of life. Cross-cultural validation of brief, ecological instruments applicable in geriatric practice is also required. Finally, it will be necessary to develop intervention studies that explore the possibility of preserving or rehabilitating specific components of social cognition through social-cognitive training, psychoeducational interventions, behavioral approaches, and neuromodulation strategies.

CONCLUSION

Social cognition represents an emerging and underappreciated clinical domain in the assessment of older adults. These aspects can change during normal aging and become specifically impaired in mild cognitive impairment and neurodegenerative diseases.

The patterns of impairment are not uniform. While in behavioral variant frontotemporal dementia, social cognitive impairments constitute core and early manifestations, in AD they tend to be more subtle in the initial stages and progress alongside global cognitive decline. In Parkinson's disease, sociocognitive changes may appear even before dementia and be associated with executive dysfunction.

This review highlights that social cognition should not be viewed solely as a secondary consequence of general cognitive decline, but rather as a partially independent domain with potential diagnostic, prognostic, and therapeutic value. Its assessment could complement conventional cognitive tests, improve understanding of behavioral and interpersonal changes, and guide intervention strategies targeting both patients and their caregivers.

In conclusion, integrating the assessment of social cognition into comprehensive geriatric evaluation could broaden our understanding of cognitive decline at an earlier stage, moving beyond memory to incorporate emotional, behavioral, and interpersonal dimensions that are essential for older adults' autonomy and quality of life.

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CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

ETHICAL CONSIDERATIONS

Protection of human subjects and animals. The authors declare that no experiments on humans or animals were performed for this research.

Confidentiality, informed consent, and ethical approval. This study does not involve personal patient data, medical records, or biological samples, and does not require ethical approval. SAGER guidelines do not apply.

Declaration on the use of artificial intelligence. The authors declare that no generative artificial intelligence was used in the writing or creation of the content of this manuscript.

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