Short report

Personalised intervention for people with depression and severe COPD

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Summary

Chronic obstructive pulmonary disease (COPD) is often complicated by depression and exemplifies the challenge in managing chronic illnesses that require active patient participation in care. In a clinical trial (NCT00151372), we compared a novel personalised intervention for depression and COPD (PID-C) targeting treatment adherence with treatment as usual (TAU). In 138 patients with major depression and severe COPD, PID-C led to a higher remission rate and a greater reduction in depressive symptoms and in dyspnoea-related disability than TAU over 28 weeks and 6 months after the last session. If replicated, PID-C may serve as a care model for patients with both depression and medical illnesses with a deteriorating course.

Declarations of interest

G.S.A. has received grant support from Forest Pharmaceuticals; has consulted to Hoffman-LaRoche, Lilly, Pfizer and Otsuka; and has served on speakers bureaux of AstraZeneca, Avenir, Forest, Merck, Novartis and Sunovion.

Method

Participants were recruited from consecutive admissions to an acute in-patient pulmonary rehabilitation unit. They signed consent approved by the Weill-Cornell Institutional Review Board. The diagnosis of COPD was made by a pulmonologist according to American Thoracic Society Guidelines after examination, spirometry and other tests. Participants met criteria for unipolar major depression (SCID/DSM-IV), and had a score of 14 or more on the 17-item Hamilton Rating Scale for Depression (HRSD). Patients with other psychiatric diagnoses (except anxiety disorders) or severe cognitive impairment (i.e. Mini-Mental State Examination score ≤ 20) were excluded. Participants were randomised into PID-C or TAU in blocks of five.

Participants in the PID-C group had their first session of PID-C (30 min) at discharge and the remainder in their own practice cases. The intervention targets patient-specific barriers to non-adherence in seven domains, i.e. misconceptions about COPD and depression, misunderstanding about their regimen, misattribution of depressive symptoms, hopelessness, overestimation of exercise effort, dissatisfaction with care and practical barriers. The care managers telephoned the patients’ physicians and informed them of the patients’ status and adherence. Physicians’ recommendations for depression and COPD were not influenced by PID-C managers. For participants in the TAU group, at discharge their own physicians received a letter informing them of the diagnosis of depression.

The primary outcomes were: (a) remission of depression (HRSD ≤7); (b) reduction of depressive symptoms; and (c) dyspnoea-related disability quantified with the Pulmonary Functional Status and Dyspnea Questionnaire – Modified (PFSDQ-M), with questions on degree of performance and frequency in ten activities influenced by dyspnoea. The PFSDQ-M has significant correlations with pulmonary function tests and partial pressure of oxygen (PO2). These instruments were administered by trained raters and not the care managers. Intent-to-treat analyses were performed. Cox’s proportional hazards survival analysis was used to study remission. To study depressive symptoms and dyspnoea, profiles of HRSD and PFSDQ-M from discharge to 28 and from 28 to 52 weeks were compared between PID-C and TAU using mixed-effects models. The models included time-trend parameter(s), group, and time x intervention interaction. The robustness of these models was tested by comparing them with the same models after multiple imputation, using a Markov Chain Monte Carlo approach.

Results

Pulmonary patients (n = 898) were screened and 138 met criteria and provided signed consent (see online Fig. DS1 for CONSORT diagram). There were no significant clinical differences among participants randomised to PID-C or TAU. During the intervention phase 12 (18%) in the PID-C and 12 (17%) in the TAU group died. Other attrition was 25% (n = 17) in the PID-C and 17% (n = 12) in the TAU group. There were no significant differences in demographics, depression and disability between those who dropped out and those who completed the study.

By the time of discharge, 88 participants had failed to remit (HRSD >7). The PID-C group had a higher remission rate (HRSD ≤7) than TAU participants (Wald χ² = 5.78, d.f. = 1, P = 0.016, hazard ratio (HR) = 2.18; number needed to treat:...
The PID-C group experienced greater reduction in dyspnoea than TAU (F(1,196) = 5.40; P = 0.021); effect size at 28 weeks was 0.53 (95% CI 0.09–0.97). The PID-C group experienced greater reduction in dyspnoea-related disability (PFSDQ-M) than the TAU group (treatment × time: F(1,197) = 4.11; P = 0.044); effect size at 28 weeks was 0.40 (95% CI −0.01 to 0.87) (Fig. 1).

Figure 1(a) Remission of depression after discharge from rehabilitation hospital and (b) course of dyspnoea-related disability in older adults with major depression and chronic obstructive pulmonary disease (COPD) randomised to personalised intervention for depression and COPD (PID-C) or treatment as usual (TAU).

Remission of depression: 17-item Hamilton Rating Scale for Depression (HRSD) ≤7.

PFSDQ-M: Pulmonary Functional Status and Dyspnea Questionnaire – Modified.

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Disclosure

We found that PID-C led to a higher remission rate of depression (1 more remission every 3.83 patients), and also reduced depressive symptoms and dyspnoea-related disability more than TAU in community residing patients with major depression and severe COPD. These benefits lasted 6 months after the intervention end. Even though COPD has a deteriorating course, dyspnoea-related disability did not worsen in the PID-C group over 1 year. If replicated, PID-C may serve as a management model for those with both depression and a medical illness with a deteriorating course, who often neglect their own care.

3.83, Fig. 1). Mixed-effects modelling showed that the PID-C group had a greater decline in HRSD than those in the TAU group between discharge and 28 weeks (treatment × time: F(1,196) = 5.40; P = 0.021); effect size at 28 weeks was 0.53 (95% CI 0.09–0.97). The PID-C group experienced greater reduction in dyspnoea-related disability (PFSDQ-M) than the TAU group (treatment × time: F(1,197) = 4.11; P = 0.044); effect size at 28 weeks was 0.40 (95% CI −0.01 to 0.87) (Fig. 1).

Mixed-effects modelling showed that the PID-C group had greater decline in HRSD than the TAU group during the follow-up phase (treatment: F(1,185) = −2.41; P = 0.018); PID-C: least square mean at 28 weeks 9.12 (s.e. = 0.96) and at 52 weeks 9.44 (s.e. = 1.06); TAU: least square mean at 28 weeks 12.24 (s.e. = 0.87) and at 52 weeks 11.43 (s.e. = 1.02). The advantage of PID-C over TAU on the PFSDQ-M was maintained even after intervention ended (treatment × time: t = −1.61, d.f. = 57.5, P = 0.113, Fig. 1). Comparison of least square means at 52 weeks favoured PID-C (t1,28 = −3.11; P = 0.002). All comparisons cited above were similar to imputed model analyses.

In conclusion, a personalised intervention offered by care managers over a period of 28 weeks increased remission rates, and improved depressive symptoms and dyspnoea-related disability more than TAU. These benefits were sustained for an additional 6 months. A next step may be to deliver and study PID-C in a group format and facilitate its dissemination. This intervention may serve as a care model for people with both depression and a medical illness with a deteriorating course, who often neglect their own care.

Discussion

We thank Timothy E. Clark and Susan Friedman, LCSW for their contributions to this study.

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References

Online supplement

**Personalised intervention for depression and COPD (PID-C)**

**Session 1 (rehabilitation hospital)**

Introduction of the role of care manager

- Describe the intervention and explain how it might help.

Assessment of barriers to treatment

*Use this guide to identify causes of barriers to treatment in each individual patient.*

- Misconceptions about COPD and depression.
- Misunderstanding about treatment and about the actual regimen.
- Misattribution of depressive symptoms.
- Hopelessness.
- Overestimation of the energy needed to perform daily exercises.
- Dissatisfaction with prior treatment or aftercare arrangements.
- Logistic barriers to treatment, for example scheduling visits and access to care, transportation, finances.

**Education**

- Brief discussion of facts about depression and its impact on the care of COPD.

**Sessions 2–9 (at home)**

Ongoing assessment

- Depressive symptoms, dyspnoea-related disability.
- Treatment recommendations (rehabilitative, medical, psychiatric) and barriers to engagement.

**Address barriers to treatment**

*Focus on barriers to treatment pertinent to the individual patient.*

- Misconceptions about COPD and depression: address incorrect facts about COPD and depression, recognise and address stigma.
- Misunderstanding about the actual regimen: discuss the role of prescribed treatment and exercise in reducing dyspnoea and disability and in preventing exacerbations.
- Misattribution of depressive symptoms: identify likely contributors to symptoms and clarify the role of prescribed antidepressant treatment in reducing them.
- Hopelessness: identify hopelessness as a symptom of depression that fuels poor expectations about treatment. Discuss the role of antidepressant treatment and exercise in improving function and in conferring a feeling of empowerment. Offer support.
- Overestimation of the energy needed to perform daily exercises: describe in realistic terms what needs to be done, when and how.
- Dissatisfaction with aftercare: help patient develop a plan to address concerns (for example coach patient to express their concerns and ask question of health professionals).
- Logistic barriers: help patients develop concrete strategies to address practical issues (for example identify ways to attend appointments; devise reminders for taking medications and conducting exercises; enlist help of family members and social services).

**Collaboration with physicians**

Inform the physicians about any significant changes in the patient's status as well as any problems with treatment adherence, and engage them in addressing them.

*The manual is available on request from the authors.*
Contacted by the Study Team \( (n = 898) \)

Excluded at in-person assessment \( (n = 751) \)
- Not depressed \( (n = 588) \)
- Other diagnoses \( (n = 87) \)
- Refused \( (n = 31) \)
- Other exclusions \( (n = 28) \)
- Received other treatment \( (n = 17) \)

Met selection criteria after in-person assessment \( (n = 147) \)

Failed to complete the baseline assessment \( (n = 9) \)

Randomised at admission to rehabilitation \( (n = 138) \)

Allocated to intervention \( (n = 67) \)
- Exit during the treatment phase \( (n = 29) \)
  - Died \( (n = 12) \)
  - Too sick medically \( (n = 3) \)
  - Admitted to nursing home \( (n = 3) \)
  - Cannot be located \( (n = 5) \)
  - Refused \( (n = 4) \)
- Rated at end of treatment \( (28\ weeks) \) \( (n = 38) \)
- Rated at 52 weeks \( (n = 27) \)

Allocated to usual care \( (n = 71) \)
- Exit during the treatment phase \( (n = 24) \)
  - Died \( (n = 12) \)
  - Too sick medically \( (n = 3) \)
  - Admitted to nursing home \( (n = 3) \)
  - Cannot be located \( (n = 4) \)
  - Refused \( (n = 4) \)
  - Other \( (n = 2) \)
- Rated at end of treatment \( (28\ weeks) \) \( (n = 46) \)
- Rated at 52 weeks \( (n = 29) \)

Exited during the treatment phase \( (n = 29) \)
- Died \( (n = 12) \)
- Too sick medically \( (n = 3) \)
- Admitted to nursing home \( (n = 3) \)
- Cannot be located \( (n = 5) \)
- Refused \( (n = 4) \)
- Other \( (n = 4) \)

Exited during the follow-up phase \( (n = 11) \)
- Died \( (n = 5) \)
- Too sick medically \( (n = 1) \)
- Went to nursing home \( (n = 1) \)
- Cannot be located \( (n = 4) \)
- Refused \( (n = 1) \)

Exited during the follow-up phase \( (n = 17) \)
- Died \( (n = 5) \)
- Too sick medically \( (n = 1) \)
- Went to nursing home \( (n = 1) \)
- Cannot be located \( (n = 8) \)
- Refused \( (n = 2) \)

Fig. DS1 CONSORT diagram: participant progress through the phases of the randomised trial.
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