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Given the unique situation of the COVID-19 surge during the conduct of this survey, the results may not reflect patient satisfaction during ordinary times.

Figure 1 and table 1.

Figure 1. Survey results*

Table 1
Clinical Characteristics (N=117 subjects)

| Characteristic          | N   | %   | SD  |
|-------------------------|-----|-----|-----|
| Age                     | 59.8 years |     | 14.79 |
| Sex (male)              | 62  | 53  |     |
| HF LVEF                 | 79  | 68  |     |
| Ischemic etiology       | 32  | 27  |     |
| Atrial Fibrillation     | 31  | 26  |     |
| Diabetes                | 59  | 50  |     |
| Hypertension            | 89  | 76  |     |
| ICD                     | 49  | 42  |     |
| S-Creatinine            | 1.37 mg/dl | 0.80  |     |
| LVEF (mean)             | 27.48 % | 10.06 |     |
| LVVIDD                  | 6.08 cm | 1.03  |     |

HF-EF: Heart failure reduced ejection fraction, ICD: implantable cardiac defibrillator, LVEF: left ventricular ejection fraction, LVVIDD: left ventricular internal diastolic dimension

*calculated for HF-EF subjects only (n=79)

Research

P006. Relationship of Heart Failure Patient Caregivers Mutuality and Preparedness to Caregiving Role Strain and Burden during COVID-19
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Background: The hospitalization rates among Heart Failure (HF) patients has increased from 23% in 2000 to 29% in 2010 necessitating the efforts to improve care and reduce cost (CDC, 2014). This is causing an increase burden on the health care system, families, and the society. At discharge, patients are educated on self-care, which is a non-pharmacological approach towards patients managing their own disease state. Self-care is a difficult concept for most Heart Failure patients to master because it requires adapting to new life-style changes. The caring burden increases as patient’s advance in the disease state. HF patients rely on informal care givers who are family and friends (Hooker, Schmiege, Trivedi, Amoyal & Beleman, 2019).

The sudden transition to the Heart failure patient’s caregiver role can be a social, financial, emotional, and physical strain that effects effective role adaptation. It is important to consider mutuality and preparedness when preparing care givers to their new role. Caregiver mutuality and preparedness can have mediating effects on caregiver role strain and burden over time (Schumacher, Stewart, & Archbold, 2007).

Methods: The purpose of this cross-sectional correlational study was to investigate the relationship between Heart failure patient’s caregiver mutuality and preparedness to caregiving to role strain and burden. Two hypotheses were tested in this study: 1) There will be a negative relationship between Heart failure patient’s caregiver mutuality and preparedness to caregivers role strain and burden 2) There will be a negative relationship between mutuality and preparedness to caregivers role strain and burden. The Roy Adaptation Model guided this research. The sample consisted of 195 adult Heart failure patient caregivers who participated via Amazon Mechanical Turk (M Turk) a crowd sourcing marketplace for survey participation and data collection. Predictor measures included mutuality and preparedness. Mutuality was measured by the Mutuality Scale and preparedness was measured by the Preparedness for Caregiving Scale. The outcome measure, role strain, was measured by the caregiver’s perception of financial, physical, and social strain, and burden was measured with the Zarit burden scale.

Results: This study demonstrated that there was a statistically significant negative relationship between caregiver mutuality and preparedness to role strain, r (195) =.058, p < .001 and a statistically significant negative relationship between caregiver mutuality and preparedness to burden, r(195) =.071, p < .001. These findings indicate that mutuality and preparedness are important predictors of caregiver role strain and burden. The Heart failure certified nurse is positioned to translate the evidence of caregiver role research to the assessment, planning, and evaluation of interventions that can assist the informal caregiver during the dynamic phases of caregiver role adaptation.

Conclusion: Assessing the caregiver’s preparation for caregiving, in addition to caregiver mutuality, is an important step in individualizing interventions that will have a positive effect on the role transition to caregiver. Furthermore, individual interventions based on the demand of caregiving will assist in the preparation of the caregiver during the transition to the community setting.

This output includes descriptive on the variables, custom tables that display the set of responses for each of your composite variables and a few regressions to explore your states hypotheses.

• Regression (multivariate): Mutuality and Preparedness vs Strain
  ◦ R2=.058 (low) and p=.001 (signif)
  ◦ Coefficients:
    □ Mutuality: -.216 (p=.042) — a negative impact (decreases) on strain (not much, but significant)
    □ Preparedness: .435 (p=.000) — a positive impact (increases) on strain

• Regression (bivariate): Preparedness vs Strain
  ◦ R2=.042 (low) and p=.002 (signif)
  ◦ Coefficient: .275 (p=.002) — a positive impact (increases) on strain

• Regression (bivariate): Mutuality vs Strain
  ◦ R2=.004, p=.600 NOTE: Mutuality not statistically significant on its own. Interplay with Preparedness

• Regression (bivariate): Mutuality and Preparedness vs Burden
  ◦ R2=.071 (low), p=.001 (signif)
  ◦ Coefficients:
    □ Mutuality: -.427 (p=.000) — negative impact (decreases) perceived Burden
    □ Preparedness: .475 (p=.000) — positive impact (increases) perceived Burden
○ Regression: Mutualty vs Burden – NOT SIGNIFICANT
○ Regression: Preparedness vs Burden – NOT SIGNIFICANT
• Regression: Burden vs Strain
  ○ R2 = 0.312 (stronger impact than those above), p < 0.000 (signif)
  ○ Coefficient for Burden = .515 (p = 0.000) (For each one point increase in the Burden score, there is a half point increase in the Strain score. Remember that these scores only range from 0 to 4, so this is a heavy hit

Research

P007. Genotypic and Phenotypic Differences and Similarities Among Patients With Transthyretin Amyloidosis or Other Inherited Cardiovascular Diseases: Insights From a Genetic Testing Program
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Background: Hereditary transthyretin amyloidosis (hATTR or ATTRv [variant]) is a progressive and fatal disease caused by mutations in the transthyretin (TTR) gene. These mutations destabilize protein folding, resulting in amyloid deposits and causing multisystem dysfunction such as cardiomyopathy, whose etiology may be attributed to traditional causes of cardiovascular diseases. Genetic testing was recently added to the diagnostic armamentarium for ATTR with cardiomyopathy. Heart failure nurses, whether registered nurses or advanced practice nurses, can have a pivotal role in appropriately diagnosing hATTR as the underlying cause of heart failure.

Objective: A molecular diagnostic program will help improve differential diagnosis and describe prevalence and characteristics of patients with TTR mutations versus patients with mutations associated with other inherited cardiovascular conditions.

Methods: Data from patients enrolled in the hATTR Compass program, which provides confidential genetic testing to patients in the United States (including Puerto Rico) and Canada with possible hATTR with polyneuropathy or with a family history of hATTR, were analyzed. DNA next-generation sequencing was performed using a panel of 92 genes associated with inherited cardiovascular conditions.

Results: A total of 978 patients under the care of cardiologists were referred for testing using this panel; 74 patients were positive for TTR mutations and 52 were positive for other non-TTR cardiovascular pathogenic mutations. The most common TTR mutation was p.V142I (V122I). Most patients (66.2%) with a TTR mutation did not have a family history of hATTR. Of patients with non-TTR mutations, 16 had mutations in the MYBPC3 locus, associated with cardiomyopathy. Patients with TTR mutations were older than those with non-TTR mutations (mean age, 67 vs 53 years). Both groups had similar proportions of heart disease (89% TTR vs 90% other cardiovascular diseases). Some key indicators of hATTR were more prevalent in patients with non-TTR versus TTR mutations: autonomic (21% vs 14%), motor (19% vs 12%), and gastrointestinal dysfunction (17% vs 8%, respectively); however, bilateral carpal tunnel syndrome (0% vs 26%) and sensory dysfunction (15% vs 28%) were more prevalent in patients with TTR mutations. A limitation of this analysis was that symptoms may have been underreported because of the simplified, voluntary nature of participation and data collection. More patients with TTR mutations had other diagnostic tests (eg, pyrophosphate imaging, biopsy) than those with non-TTR mutations (34% vs 15%).

Conclusion: Despite newer diagnostic methods such as pyrophosphate imaging, hATTR is commonly undiagnosed. Because hATTR can progress rapidly and be fatal, it is imperative that an accurate diagnosis be made early to institute appropriate therapy; genetic testing is key for obtaining an accurate diagnosis. Heart failure nurses are uniquely positioned to recognize the multiple symptoms that should raise clinical suspicion and help in the early diagnosis of hATTR.

Research

P008. Referral and Diagnosis of Hereditary Transthyretin Amyloidosis by Heart Failure Nursing Specialists in the United States: Insights From a Genetic Testing Program
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Background: Hereditary transthyretin amyloidosis (hATTR or ATTRv [variant]) is a progressive and fatal disease caused by mutations in the transthyretin gene (TTR) that result in the deposition of misfolded TTR protein in major organs and systems, leading to multisystem dysfunction. Patients often experience a mixed phenotype of both cardiomyopathy and polyneuropathy. Early diagnosis, which can be facilitated with genetic testing, is key to achieving optimal patient outcomes.

Objective: Describe the number of patients with TTR mutations and their demographics in comparison with patients with mutations associated with other inherited cardiovascular diseases that can mimic the symptoms of hATTR, in a selected sample of patients presenting with symptoms of heart failure.

Methods: This analysis collected demographic and clinical data from patients enrolled in the hATTR Compass program, a confidential genetic testing program offered in the United States (including Puerto Rico) and Canada for patients suspected of having hATTR with cardiomyopathy or with a family history of hATTR. DNA next-generation sequencing was performed using a panel of 92 genes associated with inherited cardiovascular conditions. Symptoms reported may be underrepresented because of limitations of data collection and program participation.

Results: Among 142 participating institutions in the United States, 321 patients were referred for genetic testing by heart failure nursing specialists. Of this group, 17 had TTR mutations and 36 had mutations associated with other non-TTR related inherited cardiomyopathies. More patients in the non-TTR mutation group were female compared with the TTR mutation patients (58% and 47%, respectively). Non-TTR mutation patients were younger than TTR mutation patients, on average (55 and 70 years, respectively). Most patients in both the non-TTR and TTR mutation groups (97% and 94%, respectively) did not have or did not know of a family history of hATTR. The most common non-TTR cardiomyopathy-related mutation was TTN, which is associated with dilated cardiomyopathy. The most common TTR mutation was p.V142I, which is typically associated with a predominant cardiomyopathy phenotype. Compared with TTR mutation patients, a higher proportion of non-TTR mutation patients reported sensory dysfunction, motor dysfunction, and autonomic dysfunction each. Heart disease was very common in both the non-TTR and TTR mutation patient groups (Table 1).

Conclusion: Patients with hATTR can present with both polyneuropathy and cardiomyopathy symptoms. Diagnosis of hATTR is challenging, as many patients do not have a known family history of hATTR, and symptoms of hATTR can overlap with other inherited cardiovascular diseases. It is critical to recognize the multiple symptoms that