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The real world evidence of heart failure: A model of epidemiological research using of administrative big databases

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Aims: Patients with heart failure (HF) randomized in controlled trials are generally selected and do not fully represent the "real world". The purpose of this presentation is to better describe the characteristics of HF analyzing administrative data of a population of nearly 2,500,000 subjects.

Methods: Data came from the ARNO Observatory including in-habitants of 5 Local Health Units of the Italian National Health Service (INHS). Patients were selected when discharged for HF (January 1, 2008 - December 31, 2012). Clinical characteristics, pharmacological treatments, rate and reasons for re-hospitalization and direct costs for the INHS occurring during 1 year follow-up (FU) were described.

Results: Of the 2,456,739 subjects included in the database, 54,059 (2.2%) were hospitalized for HF: 41,413 were discharged alive and prescribed on HF treatments. Mean age was 78 ± 11 years, females accounted for 51.4%. Just 26.6% were managed in a cardiology setting. The more frequent co-morbidities were diabetes (30.7%), COPD (30.5%) and depression (21%). ACE-inhibitors/Angiotensin Receptor Blockers, Beta-Blockers and Mineralocorticoid antagonists were prescribed in 65.8, 49.7 and 42.1% of patients. During 1-year FU at least one re-hospitalization occurred in 56.6% of patients, 49% of them were due to non-cardiovascular causes. INHS's direct cost per patient per year was $11,867 \in$ of which 76% related to hospitalizations.

Conclusions: Real world evidence provides a description of patients' characteristics and treatment patterns that are very different from those reported by randomized clinical trials. Costs for the INHS are mainly driven by hospitalizations which are often due to non-cardiovascular reasons.

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ATF3 expression in cardiomyocytes preserves homeostasis in the heart and controls peripheral glucose tolerance

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O besity and type 2 diabetes (T2D) trigger a harmful stress-induced cardiac remodeling process known as cardiomyopathy. These diseases represent a serious and widespread health problem in the Western world, however, the underlying molecular basis is not clear. ATF3 is an "immediate early" transcription factor whose expression is highly and transiently induced in response to multiple stressors. All cellular stresses that play a role in T2D-induced cardiomyopathy, namely, metabolic stress, oxidative stress, ER stress and inflammation, are inducers of ATF3 transcription. Here we show that mice with cardiac-specific ATF3 deficiency (ATF3-cKO) exhibit severe cardiac fibrosis, higher levels of heart hypertrophic markers, increased inflammation and worse cardiac function, as compared to wild-type mice in response to a high-fat diet (HFD). These results demonstrate that ATF3 has a protective role, dampening HFD-induced cardiac remodeling. Remarkably, HFD-fed ATF3-cKO mice display increased hyperglycemia and poor glucose tolerance, despite higher blood insulin levels, as compared to HFD-fed wild-type mice. This is the first indication that heart tissue plays a role in regulating blood glucose levels, similar to skeletal muscle. Collectively, our results suggest that cardiac-specific ATF3 expression exerts both local and systemic effects related to T2D-induced cardiomyopathy.

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