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Human Biology

Week 8 Clinical Scenario

Scenario (my answers below in bold):

Grace was a sweet and kindly 70 year old woman who had developed diffuse atherosclerosis causing narrowing of her coronary arteries. This caused her heart muscle to become very weak from lack of oxygen and other nutrients, due to poor blood flow through the arteries supplying the heart muscle {the coronary arteries} with these nutrients. She never had an actual heart attack {no death of cardiac tissue} and did not report any chest pain seeming like angina. She was one of the, unfortunately, not infrequent people who do not get any chest discomfort when her heart is 'hurting' during activity or exercise. This happens more often in females for unknown reasons. She had what is termed 'silent angina'. She had no problems with the function of the valves in her heart. Over a period of time, she developed swelling of her ankles and lower legs and would get more short of breath with very little activity.

Her diagnosis was congestive heart failure due to atherosclerosis. Congestive heart failure is a 'condition' and not a disease itself. CHF results from disease. Her disease was atherosclerosis with narrowed coronary arteries which causes most {about 95%} of the cases of congestive heart failure. Unusual viral infections which target and damage the heart muscle account for many of the rest, especially if they are young when they get their illness.

She was placed on a low table salt {sodium chloride} diet and a group of medicines that consisted of a diuretic, supplemental potassium chloride because of being on the diuretic, a medicine that relaxed the smooth muscles of the large veins and arteries of her body, and a medicine that inhibited the formation of a hormone which her body was making too much of as a result of her weak heart. She did as well as can be expected for a couple years. She was very comfortable except occasions she would rapidly develop severe shortness of breath and go to the Emergency Room where we were able to get her comfortable again. These likely were episodes of 'angina' but were without her having any symptom of chest pain. This is termed 'silent angina'. She ultimately died peacefully during sleep at home, probably of a sudden electrical heart rhythm problem where her heart just suddenly stopped beating.

Grace was alive at a time before many of the visualization tests of the heart's arteries and function were available and before many of the newer cardiac non-invasive procedures had been developed. There is a newer approach to this 'silent angina' problem that was not acceptably done at the time but has since been recognized as useful. Today it is likely Grace would have been managed differently by the doctors and may have had fewer sudden episodes of shortness of breath {she never had a lot} and a few extra years, but from what I saw, Grace had a wonderful rich full life, just as it was. More on this in the answer.

The interaction of heart muscle function, blood pressure regulation by the heart and arteries, and the kidney's functions in regulating blood volume plus pressure are the focus here. The central problem in the physiology that causes CHF is the heart can no longer generate a normal blood pressure and other organ systems react to this low blood pressure in an otherwise normal way. The only organ that 'knows' how to create a proper reaction is the brain. The kidney and other organs react as they normally would. But this normal physiology causes problems because the 'expected' blood pressure could not be developed by the weak heart, so homeostasis cannot be re-established.

Recall the various ways and organs that maintain blood pressure and consider the kidney's contribution to this and the artery muscle's contribution {big hint: what does the hormone, renin, do?}. Knowing the normal physiology gives us clues to the development of abnormal signs {i.e. measured weight and ankle swelling} and symptoms {i.e. shortness of breath}.

The questions to lead you through understanding this are: What do the kidneys do if the blood pressure is low? What hormones released by the kidney are involved? What different processes do these hormones stimulate in the body? Why was she retaining too much fluid and having ankle swelling?

What could the arterial blood vessels do if the blood pressure is low? Would this help or hinder an already weak heart? With the heart not pumping forward into the aorta as well as it would when normal, would you expect the pressure in her vena cava {the big vein returning blood from the body to the right atrium} to be higher, lower, or the same as when her heart was healthy?

When blood pressure is too low, filtration cannot occur in the glomerulus. To rectify this, the enzyme renin is secreted by the juxtaglomerular apparatus between the afferent arteriole and the distal convoluted tube in the nephrons of the kidneys. Renin then causes the release of the hormone aldosterone, which promotes excretion of potassium ions and reabsorption of sodium ions. Since salt is always reabsorbed before water, the renin-aldosterone sequence helps restore proper fluid balance in the kidneys. The secretion of antidiuretic hormones can also help raise blood pressure by increasing the amount of water in the blood (which consequently increases the total volume of blood). Release of these hormones, while occurring in healthy individuals, for sure, is not intended to be continuous. The ongoing low blood pressure caused by congestive heart failure, then, would place a constant strain on the kidneys because they are always being asked to respond to and mitigate the effects of lower-than-normal blood pressure.

In Grace's case, it is possible that dysfunction in the kidneys could have exacerbated fluid retention, but the primary cause of her edema in the ankles was probably blood backing up in her extremities due to her low blood pressure. The backing up of blood would have increased pressure in blood vessels and led more fluid to diffuse into the tissues, causing to swelling.
Grace's shortness of breath may also have been related fluid retention to pulmonary edema, or the buildup of fluid in her lungs (medlineplus.gov/ency/article/000140.htm).

In the heart, it would make sense for the arterial blood vessels to constrict to increase blood pressure. In Grace's case, this would problematic because artherosclerosis had already narrowed her coronary arteries and constriction could limit blood flow too much or perhaps even close an artery completely. With less blood flow in the coronary arteries, less oxygen and fewer nutrients would be available for her heart, causing further weakness. I also suspect--although I have not been able to find a reference for this--that a heart already damaged by congestive heart failure may not be able to withstand any significant increase in blood pressure, if it were even possible to achieve.

I would imagine that the blood pressure throughout the pulmonary and systemic circuits is low in cases of congestive heart failure like Grace's, because the heart itself is not pumping the way it should. That would mean that blood pressure in the vena cava--where blood is returning to the heart from the veins--was also lower for Grace when she developed congestive heart failure than when her heart was functioning properly.