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4/9/1994 12:00:00 AM

Discharge Summary

Unsigned

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Report Status :

Unsigned

ADMISSION DATE :

04-09-94

DISCHARGE DATE :

05-26-94

CHIEF COMPLAINT :

Cean Leach is a 38 year old male who is admitted in transfer from the Morehegron Valley Hospital in Ville , Virginia for further evaluation and management of accelerating chest pains syndrome .

Mr. Leach has a long and complex history of coronary artery disease .

He was without known cardiovascular illness until he suffered an acute anterior myocardial infarction in August of 1988 .

He was treated at that time with TPA .

A subsequent cardiac catheterization revealed a 90% lesion in his proximal left anterior descending coronary artery and minor disease elsewhere in his coronary distribution .

His ejection fraction was in the mid-50's.

Because of angina following that catheterization , he ultimately underwent percutaneous transluminal coronary angioplasty of his left anterior descending coronary artery .

He did well until he developed recurrent angina in March of 1991 at which time repeat catheterization demonstrated restenosis of his left anterior descending and he underwent a second PTCA with good results .

He again did well until approximately late 1992 .

Since that time , he has had multiple admissions to George'sgot Medical Center with recurrent chest pain .

He has ruled in for small R Q wave myocardial infarctions on many but not all of these occasions .

He has had a progressive decline in his left ventricular ejection fraction as measured by non-invasive means with an echo ejection fraction in the low 40's and radionuclide ejection fraction estimated in August of 1994 at 31% .

He was admitted to George'sgot Medical Center with a typical episode of this discomfort in October of 1994 .

He was transferred at that time to the Ph University Of Medical Center .

That was his first Ph University Of Medical Center admission .

Because of Mr. Leach 's history and the fact that multiple cardiac catheterizations prior to that transfer had failed to demonstrate fixed epicardial coronary disease , it was decided that he should undergo provocative testing to evaluate whether or not he had coronary artery spasm and / or impaired coronary flow reserve .

His angiograms from Morehegron Valley Hospital from his four prior catheterizations were reviewed and the patient was gradually tapered off all of his vasoactive medications .

He was taken to the Catheter Laboratory on October 13 , 1994 .

There he had a right heart catheterization with unremarkable intercardiac pressures .

Repeat coronary angiography once again failed to demonstrate any significant obstructive lesions in either his dominant right coronary artery or his left coronary system .

He then underwent a Doppler Coronary Flow study which demonstrated impaired coro

nary flow reserve .

Specifically , his coronary flow maximally increased by a factor of 2-2.5 whereas a normal response is considered to a 3-4 fold increasing in coronary flow to maximal stimulus .

Following the coronary flow studies , he was given greater doses of intracoronary ergonovine to assess his susceptibility to coronary artery spasm .

He had a normal response to ergonovine that is he had diffuse narrowing of his coronary arteries without induction of focal spasm .

However , he did develop typical anginal discomfort which was easily reversed with intracoronary nitroglycerin .

It was felt that the time that this study was most consistent with a syndrome of impaired coronary flow reserve or microvascular angina .

He was treated with increasing doses of two calcium channel blockers and gradually ambulated in the hall without discomfort and was discharged to home .

Following that admission in October he had a symptom free interval which lasted a couple of months .

Starting in the October of 1994 , he once again started having recurrences of his typical anginal discomfort .

This occurred at rest and with exercise .

He had multiple admissions to the Moreheggon Valley Hospital once again ruling in for a small R Q wave myocardial infarctions on a number of these occasions .

His medications were adjusted many times without adequate interruption of the pattern of increasingly frequent and increasingly severe episodes of pain .

His most recent admission to the Moreheggon Valley Hospital is on April 5 , of 1994 .

He had a prolonged episode of typical anginal pain and rule in for a small Q wave myocardial infarction .

Because of the refractory nature of his chest pain and the failure of multiple drug combinations to control his symptoms , he was transferred to Ph University Of Medical Center on 4-9-94 .

His medications that had been used individually or in combination include beta blockers and Lodapine cardiazem , nifedipine and coumadin , aspirin , cyclopamine and serotonin antagonists .

There is no history of drug abuse or smoking .

Of note , his brother had a myocardial infarction in his late 20's .

There is no other family history of premature coronary disease or history of stroke or deep venous thrombosis suggestive of hypercoagulable state .

Of note , his prior work-up has also included screening test for hypercoagulability with normal levels of protein S and C and an unrevealing right ventricular endomyocardial biopsy .

His medications on admission included Isordil , Lovastatin , aspirin , heparin and Lodapine , Cardizem and Lasix .

PHYSICAL EXAMINATION :

He looked generally well .

His blood pressure 160/90 .

His heart rate was 88 .

His jugular venous pressure was normal .

His chest was clear and his carotids were brisk and his cardiac examination revealed a soft systolic flow murmur .

His abdomen was obese with no palpable organomegaly .

His femoral pulses were 1+ and equal without bruits .

He had trace peripheral edema and excellent peripheral pulses .

His initial laboratory values included a sodium of 138 .

Potassium of 3.2 .

Normal chloride and CO2 .

His BUN was 12 and his creatinine was 1.1 .

Glucose was 155 although subsequent determinations were normal .

His fasting cholesterol was 174 with an HDL of 35 and an LDH of 97 .

His liver function tests were normal with the exception of his LDH which was mildly elevated to 222 .

His hematocrit was approximately 39 with an unremarkable differential and normal platelet count .

His sedimentation rate was 32 and was 40 on repeat testing .

HOSPITAL COURSE :

the primary concern upon Mr. Leach 's transfer was that despite the fact that he had multiple previous catheterizations over the prior two years that he may have redeveloped obstructive coronary artery disease .

It was ; therefore , felt appropriate to start his repeat work-up by repeating his cardiac catheterization and coronary angiography .

To that end , he was taken to the Cardiac Catheterization Laboratory on 4-10-94 , there he underwent right and left heart catheterization , coronary angiography and left ventriculography .

The right heart catheter which was done using a Swan Ganz catheter advanced to 8 French sheath and right femoral vein revealed a mean right atrial pressure 3 mm of mercury and a pulmonary capillary wedge pressure of 12 mm of mercury .

The left ventricular pressure was 105/12 .

Coronary angiography revealed a picture which was essentially unchanged from October of 1994 .

That is there was no obstructive large vessel or coronary artery disease although there were some diffuse luminal irregularities .

Left ventriculography revealed a large area of antero lateral and apical hypokinesis to akinesis with an estimated ejection fraction of 35% .

Impression at the time of his catheterization was that the precise etiology of his chest pain remained rather enigmatic .

He was ; therefore , seen in consultation by Dr. Lato Tikfreierm of the Hematology Service to evaluate him further for a possible hypocoagulable condition versus primary and secondary platelet abnormality which could explain his recurrent coronary events .

After an extensive evaluation including in vitro testing and flow cytometry of Mr. Leach 's platelets , it was ultimately determined that he did in fact have a primary platelet defect .

Specifically , his platelets were not affected by aspirin and showed spontaneous hyperaggregability .

This was utilized in making some changes in Mr. Leach 's regimen .

Specifically his aspirin was discontinued and he was put back on ticlopidine at a higher than ordinary dose at 500 mg. b.i.d.

Repeat in vitro testing of Mr. Leach 's platelets suggested that although they remained refractory to aspirin , they did demonstrate reduced hyperaggregability spontaneously which was felt to be possible reflective of the ticlopidine effect .

While in the hospital , Mr. Leach continued to nearly daily episodes of chest pain .

These were generally spontaneous in nature and lasted anywhere from several minutes to nearly an hour duration .

There were typically improved but not completely eradicated with sublingual nitroglycerin and generally required i.v. narcotics for complete pain relief .

Because of the ongoing nature of his pain .

Multiple changes and additions were made to his drug regimen .

He was tried on theophylline and imipramine without real change in his chest pain syndrome .

He was tried on a variety of different combinations of calcium channel blockers including amlodipine and diltiazem and lodipine and verapamil .

Amlodipine and bepridil all without marked change in his clinical syndrome .

The rest of his hypercoagulable work-up revealed that he had nearly normal levels of clotting factors , specifically , his antithrombin 3 of level was 111 with a reference range of 80-120 .

Protein C was 85 with a reference range of 70-140 .

Protein S was 114 with a reference range of 70-140 .

Plasminogen was slightly high at 151 with upper limit of normal being 130 .

Factor 8 was likewise slightly high at 249 with a reference range of 50-200 .

Thrombin time and reptilase time were normal .
It was felt that these results were non-specific and may represent just an acute phase reactants .
His anti-cardiolipin antibodies both IGM and IGG were within normal range .
His APC resistance was normal .
He was seen in consultation by both Rheumatology and Endocrinology to evaluate the possibility of primary connective tissue disease and / or possibly a pheochromocytoma .
Rheumatology felt that there was no good evidence of a primary vasculitic process as the source of his presumed small vessel coronary disease and no further rheumatologic work-up was undertaken .
He had multiple 24 hour urine tests performed to evaluate the question of pheochromocytoma , these demonstrated mild elevations of some urinary catecholamines , but it was felt that this could well represent a reaction to the complex medical regimen that he was having as well as the recurrent episodes of pain themselves .
It was not felt that he had good evidence of pheochromocytoma .
His complement levels were normal and he had no evidence of hepatitis , B serologies which also measured to see if there was some chronic inflammatory process .

Because of the persistent nature of his pain and the inability to control it adequately on constantly changing a quite complex medical regimen , it was suggested that a sympathetic blockade be performed as a method of controlling his chest pain syndrome .

To evaluate the efficacy of this prior to committing him to a permanent blockade procedure , a temporary sympathetic blockade was performed using a local anesthetic and stellate ganglion injection .

Prior to performing that an exercise tolerance test was performed on May 13 . That study demonstrated that the patient was able to exercise for ten minutes of a standard protocol and stopped because of fatigue ; although he had no significant ST changes with exercise , he did experience his typical anginal chest discomfort which was ultimately relieved of the combination of sublingual nitroglycerin and i.v. morphine sulfate .

On the basis of that test , he underwent stellate block the following day . That was performed in the pain clinic using 1% lidocaine to the skin and 10 cc of Bupivacaine under fluoroscopic guidance at the level of T1 . This produced a good sympathetic block with evidence of a rise in temperature in the left arm and ptosis and myosis of the left eye .

He was then taken directly from the pain clinic to the exercise laboratory and repeat exercise testing was done .

He once again exercised for ten minutes achieved the same rate pressure product and had no chest discomfort .

This encouraged his care takers to pursue a more permanent sympathetic blockade .

After extended discussions with pain service , Mr. Leach was brought back down to the pain clinic on October 27 .

There a catheter was placed at the level of T1 under fluoroscopic guidance .

Once again Narcaine was injected to induce a sympathetic block .

The catheter was left in place for approximately 24 hours with recurrent injections maintained the sympathetic block and during that time he had no recurrent angina .

After extensive discussions with the patient , the pain service and thoracic surgery , it was felt appropriate to proceed with a surgical sympathectomy to relieve him of his intractable chest pain syndrome .

Therefore , on October 27 , the patient underwent left thorascopic thoracic sympathectomy by Dr. Aalv Sctroi .

He tolerated that procedure well and had no recurrent anginal pain throughout the rest of his hospitalization .

He had incisional pain which was well-controlled with a combination of Percocet and morphine sulfate and progressively ambulated and was discharged to home in good

ood condition free of recurrent angina on May 27 , 1995 .

DISCHARGE MEDICATIONS :

Ticlopodine 500 mg. p.o.b.i.d. Lovastatin 20 mg. p.o.q.d. Lasix 80 mg. q.d. Kayc
iel 60 meq b.i.d. Cimetidine 400 mg. b.i.d. Isordil 20 mg. t.i.d. and Lodopine 1
0 mg. q.d. a combination of Percocet , Morphine Sulfate elixir p.o. as needed .

DISCHARGE DIAGNOSIS :

1. Unstable angina secondary to syndrome X .
2. Hypertension and hyperlipidemia as well as epicardial coronary artery disease

PRINCIPAL PROCEDURES :

Left thorascopic sympathectomy on May 21 and cardiac catheterization and coronar
y angiograph and left ventriculography on April 10 , 1994 .

Treadmill exercise testing X2 on May 13 and May 14 .

Left stellate ganglion sympathetic block on 5/14/94 and placement of a low cathe
ter with sympathetic block on October 27 .

TRI I. MNONKOTE , M.D.

TR :

cp / bmot

DD :

06-02-94

TD :

06/02/94

CC :

Dr. Lato Tikfreiern of Pediatric Hematology .

Dr. Taarnna Lanceloilneg of the Cardiac Unit and Dr. Aalv Sctroi of Thoracic Sur
gery .

Dr. Center of Endocrinology .

Dr. Conrellie Koterudes , Medical House Officer .

Dr. Sta Center of the Pain Service .

Dr. Li Rhalttland c / o George'sdgot Medical Center , Aurie

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