D:/Nsurg/Vol. 13, No. 2, Jul. - Dec. 2009/Nsurg-12.doc (A) P. 83 – 86

CASE REPORT

Basal Ganglia Infarct in Young Female Patient: in Consideration with D.V.T and Paradoxical Emboli

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ABSTRACT:

Twenty four years old female presented on the 20th postnatal day with painful swelling of her limbs (more in the lower limbs) along with headache, nausea, and right sided weakness. On the basis of the history, relevant physical examination and investigations (C.T scan brain and echocardiography) she was diagnosed to have a paradoxical emboli from D.V.T (deep venous thrombosis) of her legs that reached her brain through patent foramen oval causing a basal ganglia infarct. Based on her serum albumin and ascetic albumin, her diagnosis was made which was in the favor of nephrotic syndrome. She was heparinized and anti-platelets aggregating therapy was initiated for her deep venous thrombosis. She was advised to get physiotherapy for the weakness she had on her right side. Because of timely diagnosis and early treatment her weakness had a gross improvement and she was able to start moving her limbs by herself at the time of hospital discharge.

INTRODUCTION

Foramen ovalis: In fetus the foramen oval is held open by the pressure gradient between the right and the left atria. The right atrial pressure which is more than that of the left atria pushes the flexible septum aside permitting the right to left flow of oxygenated blood from the inferior vena cava into the left atrium.

Patent foramen ovalis: The remnant of the foramen oval is termed fossa ovalis. If the foramen oval is completely covered but not sealed, it is called probe patent or P.F.O (patent foramen ovale), indicating that the foramen can be opened by reversal of the intra-atrial pressure gradient intra-cardiac catheterization.

Approx. 20-34% of the normal human heart has P.F.O.[1] Defect typically ranges from 1-10 mm in maximum potential diameter. A patent foramen ovale is not considered an atrial septal defect (A.S.D) because no septal tissue is missing. As a result no intra-atrial shunting can occur as long as left atrial pressure exceeds right atrial pressure and the flap valve remnant of septum premium of foramen Oval is competent. However, an elevation of the right atrial pressure can cause right to left intra-atrial shunting through patent foramen ovale (P.F.O). Intermittent increase in the right atrial pressure occurs in the normal individuals during early ventricular systole and

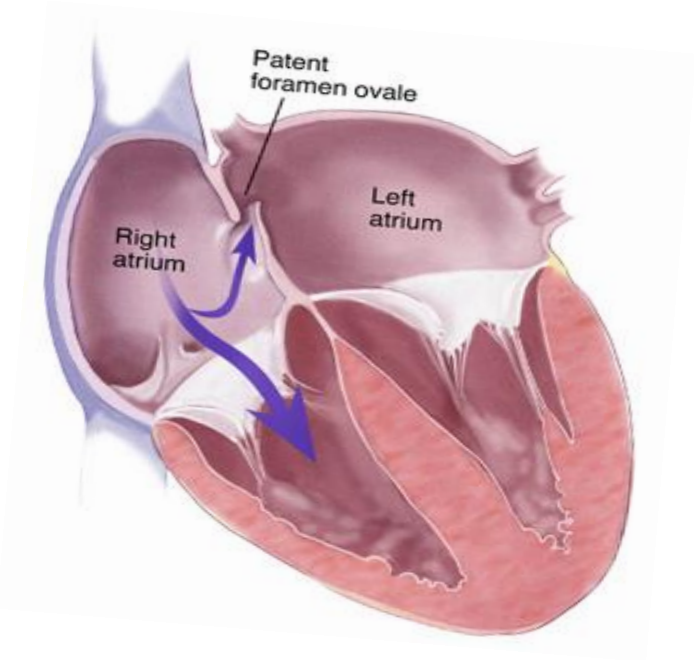

Fig. 1:

with the decrease intra-thoracic pressure of inspiration.

Clinical importance of the patent foramen ovale (P.F.O) lies in its association with the paradoxical emboli and stroke.[2-4] Paradoxical emboli occur when embolus arising in the systemic venous system or the right atrium crosses the patent foramen ovale (P.F.O) during the right to left shunting and entering the systemic arterial circulation.[5]

Diagramatic Presentation of Patent Foramen Oval in Human Heart: (Taken from www.nmtmedical.com/ patient heart 2 main).

Case Report

A young 24 years old female patient primigravida, having 35 weeks of gestational amenorrhea noticed swelling in all limbs and a disproportionate increase in abdominal girth. She also developed nausea and headache. As she was pregnant she went to a local general practitioner, where symptomatic treatment was given. She did not improve and visited a gynecologist at the district hospital with complaints of shortness of breath, swelling of her left leg, headache nausea and right sided weakness.

The patient was admitted in the hospital and was labeled as having deep venous thrombosis in the left leg; she was put on heparin 10,000 units 8 hourly along with antibiotics. On her second day of admission, she suddenly developed respiratory distress along with a low blood pressure and a weak thready pulse; she was started on oxygen inhalation and was given aminophyline and hydrocortisone (500mg) each as an IV infusion.

A call was given to the medicine. She was advised renal function test along with urine analysis and a chest x-ray. The results of the reports revealed hemoglobin of 5.5mg/dl, a blood urea of 60mg/dl and serum creatinine of 1.8mg/dl.

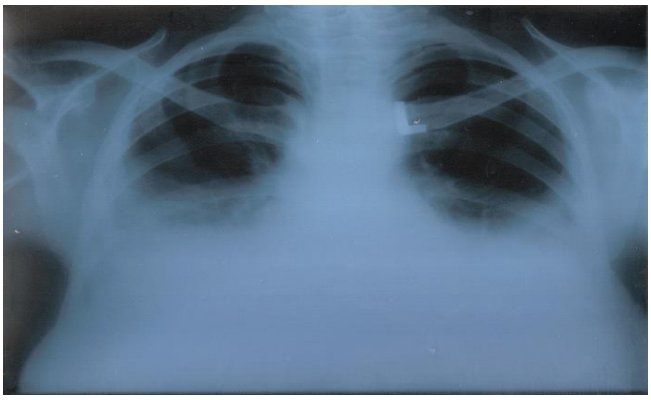

Fig. 2: *An X- Ray Chest of the Patient Showing Bilateral Pleural Effusion.*

One unit of whole blood transfused and a low dose of dopamine support was started. When the patient improved, she was sent for a chest radiograph the next morning (Fig. 2) while at the radiology department, she went into labor and was soon shifted to the labor room.

The patient delivered a healthy live male baby. The placenta was expelled; there was no tear and no post partum hemorrhage. She was kept in the ward for 2 days where she kept feeding her baby. She was discharged on tablet warfain 2.5mg/day, she was advised to periodically check her P.T/I.N.R.

Two weeks later, she again presented with generalized body swelling, ascites, palpitations, easy fatigability and right sided weakness. On physical examination, her pulse was 86 beat per minute, B.P. was 130/90 mm of Hg and was a febrile. Central nervous system examination revealed right sided hemi paresis and up going right planter.

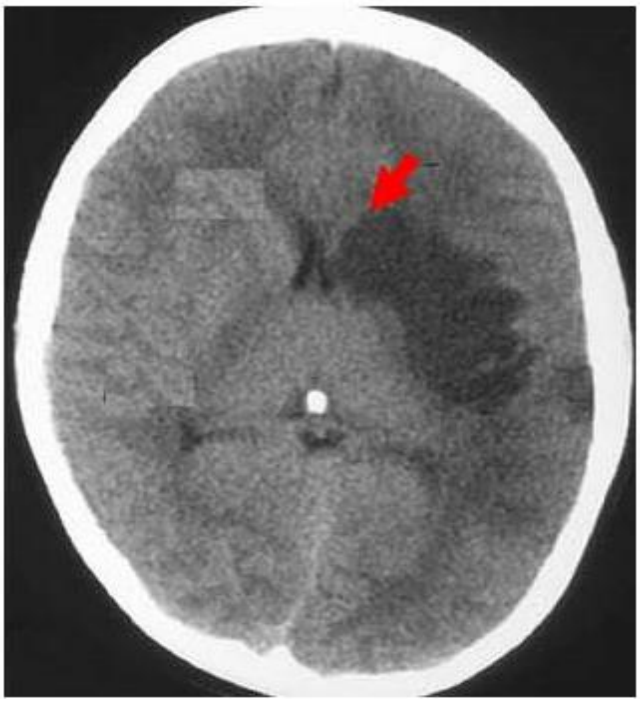

Fig. 3: *C.T Scan Brain Plain of the Patient Showing Left Basal Ganglia Infarct).*

The examination also revealed distended abdomen, positive shifted dullness and diminished bowel sounds. Her chest examination revealed bilateral

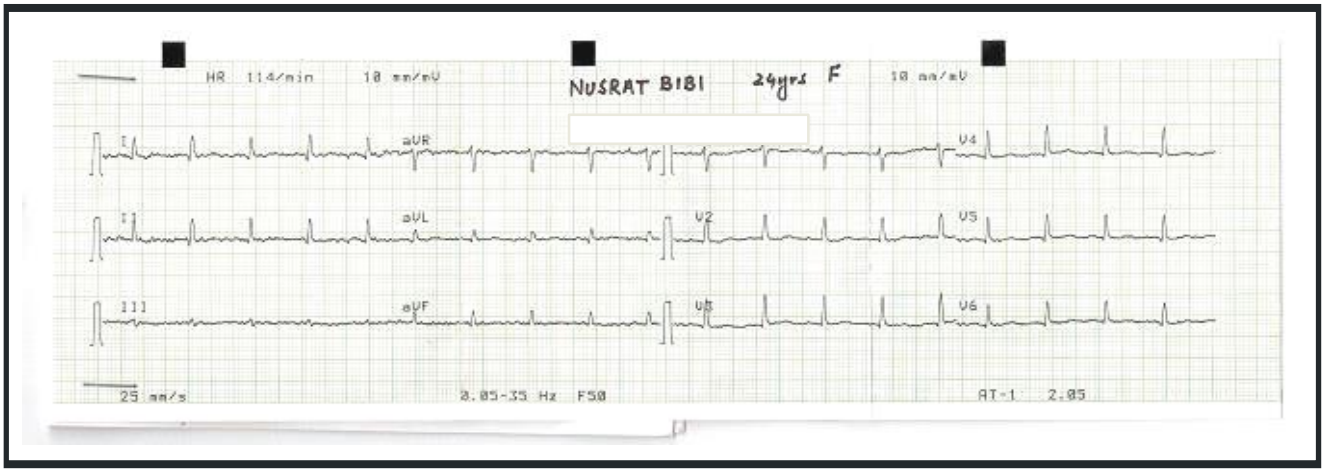

Fig. 4: *E.K.G of the Same Patient with no Acute Changes.*

crypitations and decrease breath sounds, J.V.P. was not raised and there was no carotid bruit; Her I.N.R. and prothrombin time was repeated and her Doppler ultra-sound of the left leg was done which revealed D.V.T. The patient was started on ceftriaxone 1gm intravenously 12 hourly, injection heparin 10,000 units subcutaneously 8 hourly and tablet warfarin 2.5 mg/daily.

She was advised to have a C.T scan of the brain which showed a basal ganglia infarct on the left side (Fig. 3).

An electrocardiogram (E.K.G) was performed to evaluate any changes in the activity of the heart over time however no acute changes was seen on the EKG report (Fig. 4). To rule out any abnormal leaking of blood through the valves and to observe the cardiac output, echocardiography was performed (data no sho-wn) which revealed the patent foramen ovale. It thus became obvious that this could be paradoxical emboli which got dislodged and went all the way to the right side of the heart and due to the increase in the right atrial pressure caused by the intra-thoracic pressure the emboli went to the left atria and to the brain causing an infarct.

She was put on anti-platelet aggregating therapy (Disprin 75mg and clopidogrel 75mg orally). For the abdominal swelling her ascetic diagnostic tap was sent to the laboratory. The report of which turned out to be transudate (Table. 1). After this her serum albumin was performed which turned out to be 1.57 g/dl. Her Serum-ascites albumin gradient **(**SAAG**)** value was more than 1.2 g/dl. Her urine routine examination showed +3 proteins and 24 hours urinary protein of 2450 mg/ 24 hrs.

These investigations also favored that the patient was having nephrotic syndrome, she was advised to restrict protein dietary intake, and was referred to the nephrologists for further management.

Table 1:

Patient Lab Reports	
Protein content	0.5 mg/dl
Glucose	60 mg/dl
Total cells	36 mg/dl
Differential cells	Nil
Lymphocytes	87%
Mesothelial cells	4%
Plasma cells	1%
Neutrophil	8%
Malignant cells	Not seen
Z.N stain:	No A.F.B seen
Gram staining	No gram positive / no gram negative bacteria seen

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