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PARANEOPLASTIČKI SINDROM KAO MOGUĆI UZROK PLUĆNE TROMBOEMBOLIJE KOD PACIJENTKINJE SA NEFROTSKIM SINDROMOM

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SAŽETAK: Prikaz slučaja: Pacijentkinja dobi 59 godina primljena je zbog gušenja, oticanja potkolenica i opšte slabosti. Prethodno je lečena nekoliko godina zbog fokalno segmentne glomeruloskleroze sa nefrotskim sindromom imunosupresivnom terapijom. Očekivani terapijski odgovor nije postignut. Po prijemu laboratorijski su utvrđeni: hipoalbuminemija, hiperlipidemija i proteinurija nefrotskog ranga. Na RTG pluća opisana je obostrana pleuralna efuzija zbog koje je urađena pleuralna punkcija kojom je evakuisano 800 mL tečnosti. Urade se tumorski markeri, ehosonografija dojki i mamografija, a po preporuci onkologa i scintigrafija kostiju. Na mamografiji opisani su mikrokalcifikati obostrano, a scintigrafija kostiju pokazuje patološku akumulaciju radiofarmaka u petom torakalnom pršljenu i korpusu sternuma, te III i IV rebru levo. Petog dana hospitalizacije dolazi do pogoršanja opšteg stanja, hipotenzije, tahikardije i stenokardije kao i porasta D-dimera. Na EKG-u sinusni ritam, frekvenca 80/min, niska voltaža u standardnim i unipolarnim odvodima. Po preporuci kardiologa uradi se CT po programu za plućnu tromboemboliju (PTE) koja je pokazala submasivnu PTE. Primjenjena je terapija niskomolekularnim heparinom, oksigenoterapija uz dopaminergike, bronhodilatatore, infuzije humanih albumina i plazme i tranzitorno lečenje hipervolemije hemodijalizama. Pacijentkinja je zbog multiorganske disfunkcije bila hospitalizovana 61 dan. Magnetna rezonanca dojki nije urađena zbog lošeg opšteg stanja bolesnice. Najverovatnije se radilo o karcinomu dojke sa sekundarnim depozitima koji je kasno prepoznat. PTE, kao vjerovatna posledica paraneoplastičkog nefrotskog sindroma, pravovremeno je dijagnostikovana i lečena.
Ključne reči: nefrotski sindrom; plućna tromboembolija; paraneoplastični sindrom

UVOD

Plućna tromboembolija je vaskularno oboljenje nastalo kao komplikacija venske tromboze i otkidanja tromba koji cirkulacijom dospeva do pluća. Od stepena okluzije i broja zahvaćenih plućnih arterija (masivnosti embolije) zavisi i klinčka slika. Godišnja učestalost 2-3/ 1000 stanovnika. Najčešće su embolizacije iz proksimalne dubokea venska tromboza ekstremiteta (DVT) - 40%, vene kave inferior VCI - 10-20%, distalna DVT - 20-30%, a retko iz gornjih ekstemiteti usled centralnog venskog katetera (CVK). Faktori rizika: traume, ortopedske operacije (naročito kuka i kolena), velike abdominalne, torakalne, ginekološke operacije, operacije vena, kardiovaskularna oboljenja praćena srčanom dekompenzacijom i aritmijama, septična stanja, dugotrajna imobilizacija, porođaj, autoimuna oboljenja, kao i maligna oboljenja (karcinom pankreasa, dojke, prostate i bronha).

PRIKAZ SLUČAJA

Korišten materijal iz istorije bolesti pacijentkinje, medicinski podaci iz bolničkog informacionog sistema KIS - UKC Republike Srpske, otpusno pismo pacijentkinje iz UKC Republike Srpske.

Pacijentkinja je imala pozitivnu porodičnu anamnezu na maligne bolesti i značajno je izgubila na telesnoj težini.

Objektivni fizikalni nalaz: kahektične građe. Cor: Srčana akcija ritmična, ubrzana, tonovi tiši, bez šumova, TA 120/70 mmHg. Pulmo: Auskulatorno nad plućima obostrano oslabljen disajni šum. Donji ekstremiteti (DE): Obostano pretibijalni edemi.

Laboratorijski nalazi:

- Hematološki parametri: Leukociti 14,3 x 10^9/L; Eritrociti 4,43 x 10^{12}/L; Hemoglobin 137 g/L, Trombociti 528 x 10^9/L;
- Biohemijski osnovni parametri: enzimi jetre: AST 38 U/L ALT 26 U/L J, LDH

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Vol. 45 (2020) No. 3 TIMOK MEDICAL GAZETTE Case report

PARANEOPLASTIC SYNDROME AS A POSSIBLE CAUSE OF PULMONARY THROMBOEMBOLISM IN A FEMALE PATIENT WITH NEPHROTIC SYNDROME -CASE REPORT

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SUMMARY: A 59-year-old female patient was admitted to the hospital due to suffocation, lower leg swelling, and general weakness. She had been treated previously with immunosuppressive therapy for several years because of focal segmental glomerulosclerosis with nephrotic syndrome. The expected therapeutic response was not accomplished. Upon admission, the following were determined in the laboratory: hypoalbuminemia, hyperlipidemia and nephrotic range proteinuria. X-ray of the lungs showed bilateral pleural effusion, because of which a pleural puncture was performed and which drained 800 mL of fluid. Tumor markers test, breast echosonography and mammography were performed, along with bone scintigraphy, which was done upon the recommendation of an oncologist. Mammography described microcalcifications bilaterally while bone scintigraphy showed pathological accumulation of radiopharmaceuticals in the V thoracic vertebra and sternum corpus, and III and IV ribs on the left. On the fifth day of hospitalization, there was a deterioration of patient's general condition with hypotension, tachycardia and angina, as well as an increase in D-dimer. On the ECG sinus rhythm, f 80 / min, low voltage in standard and unipolar leads. Upon the recommendation of a cardiologist, CT was performed according to the program for pulmonary thromboembolism (PTE), which showed submassive PTE. Low molecular weight heparin therapy was used, along with oxygen therapy with dopaminergics, bronchodilators, human albumin and plasma infusions, statins and transient treatment of hypervolemia by means of hemodialysis. The patient was hospitalized for 61 days due to multiorgan dysfunction. Breast magnetic resonance imaging was not performed due to the poor general condition of the patient. Most likely it was breast cancer with secondary deposits, which was recognized late. PTE, as a probable consequence of paraneoplastic nephrotic syndrome, was diagnosed and treated in a timely manner.

Key words: nephrotic syndrome; pulmonary thromboembolism; paraneoplastic syndrome

INTRODUCTION

Pulmonary thromboembolism is a vascular disease that occurs as a complication of venous thrombosis and a thrombus breaking loose and reaching the lungs through circulation. The clinical picture also depends on the degree of occlusion and the number of affected pulmonary arteries (massiveness of the embolism). Annual frequency is 2-3 / 1000 inhabitants. The most common embolizations are proximal DVT 40%, VCS - 10-20%, distal DVT - 20-30%, upper extremities (CVC). Risk factors: trauma, orthopedic surgery (particularly hip and knee), major abdominal, thoracic, gynecological surgeries, vein surgery, cardiovascular diseases accompanied by cardiac decompensation and arrhythmias, septic conditions, long-term immobilization, childbirth, autoimmune diseases, as well as malignant diseases (pancreatic, breast, prostate and bronchus cancer).

CASE REPORT

Material used from the patient's medical history, medical data from the hospital information system KIS-UCC of Republika Srpska, patient's letter of discharge from UCC of Republika Srpska.

Results: The patient had a positive family history of malignant diseases and a significant weight loss. Objectively of cachectic appearance. Cor: Heart action rhythmic, fast, tones quieter, no noise, TA 120 / 70mmHg. Pulmo: Ausculatory over the lungs bilaterally diminished respiratory murmur. DE: Mutual pretibial edema.

Laboratory findings:

- Haematologic parameters: Leukocytes $14,3x10^9$/L0; Erythrocytes $4,43x10^{12}$/L;

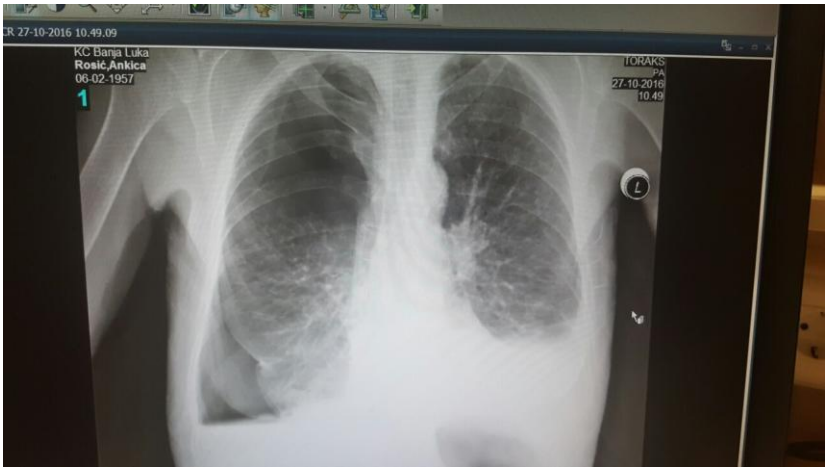
300 U/L, ukupni proteini seruma: 41 g/L, albumini 21 g/l, holesterol 4,6 mmol/l, trigliceridi 3,9 mmol/L

- Kardiološki biomarkeri: CK 84 U/L; CK-MB 19 U/L; TnT 69,7 ng/L; D-dimer 8,93 ng/l;
- Azotne materije i klirens kreatinina: urea 11,9 mmol/L, kreatinin 101 μmol/l (eGFR po forumuli Cockcroft: 41,7 ml/min, MDRD 51,7 ml/min), mokraćna kiselina 317 μmol/l
- elektroliti seruma i parametri acidobaznog statusa: K 3,7, Ca 2,12, Na 140, Cl 100, P 0,94, ASTRUP: pH 7,482, cHCO3 26,8, ABE 3,6.
- Analiza urina: albumin++++, eritrociti 3-4, leukociti 6-10; BIURET 4,3 g/24h
- Tumorski markeri: CA 125 586, CA 15-3 98, CA 19-9: 1,2, CYFRA 21-1 2,8, CEA 9,4, NSE 9,4, HE4 241,5, ROMA 90,7%.
- Hormonski status: TSH 2,75, FT4 17,75, Tireoglobulin 41,91, kalcitonin 0,694.

Zaključujemo da je pacijentkinja imala trombocitozu, bubreznu insuficijenciju III stepena, uredan mineralni status, hipoproteinemiju i hiperlipidemiju, proteinuriju nefrotskog ranga, metaboličku alkalozu i povišene tumorske markere za dojku i genitalni trakt.
Ultrazvučni ginekološki nalaz je bio uredan.
Petog dana hospitalizacije pacijentkinji se naglo pogoršava kliničko stanje. Kao tegobe navodi gušenje, stezanje u grudnom košu i suhi kašalj. Objektivno dispnoična u miru uz prisutnu centralnu cijanozu, akcija srca tahikardična, tonovi tiši, bez šumova, TA 80/60mmHg. Na EKG-u sinus ritam, frekvenca 80/min, niska voltaža u standardnim i unipolarnim odvodima. Primenjena je oksigenoterapija, Dobutamin 5mcg/kg/min (250mg Dobutamina u 250mL 0,9%NaCl-a), Clexane 0,6 ml 1x1 s.c i uzeta krv za D dimer i kardiospecifične enzime.

RTG snimak srca i pluća otkriva obostrano pleuralni izliv (efuzija pleure) do nivoa V rebra kao i inkapsulirani izliv u projekciji donjeg plućnog polja desno (Slika 1.)

Slika 1. RTG snimak srca i pluća: obostrana efuzija pleure do nivoa V rebra, inkapsulirani izliv u projekciji donjeg plućnog polja desno



Kompjuterizovana tomografija toraksa (CT) po programu za PTE prikazao je submasivnu tromboemboliju desne plućne arterije (Slika 2.). Diferencira se defekt u lumenu- tromboembolija grane desne plućne arterije za gornji rezanj sa ekstenzijom u segmentne grane za anteriorni segment kao i nepotpuna tromboza intermedijalne grane desne plućne arterije. Širina stabla plućne arterije 23mm, desne plućne arterije 21mm, leve plućne arterije 18mm. U prikazanom parenhimu nema konsolidacije ni infiltracije. Posterobazalno pleuralni izliv desno širine oko 5cm, levo do 6cm sa posledičnim kompresivnim atelektazama bazalnih segmenta donjih plućnih reznjeva.

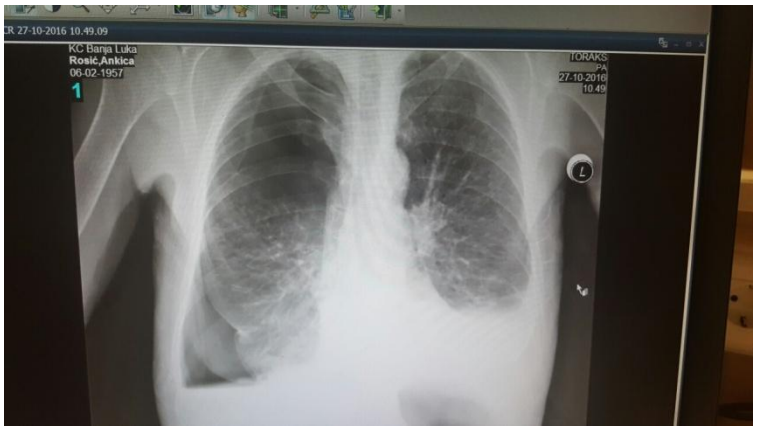
Haemoglobin 137 g/L, Thrombocytes 528 x 10^9/L;

- Byochemical basic parameter: AST 38 U/L, ALT 26 U/L, LDH 300 U/L;, total serum proteins 41 g/L, albumins 21 g/l, cholesterol 4,6 mmol/l, triglycerids 3,9mmol//L,
- Cardio-byomarkers: CK 84 U/L; CK-MB 19 U/L; TnT 69,7; D-dimer 8,93;
- Blood Nitrogen substances and creatinine clearance: urea 11,9mmol/L, creatinin 101 ɥmol/l (eGFR by formula Cockcroft: 41,7ml/min, MDRD 51,7 ml/1,73m^2/min), uric acid 317 ɥmol/l,
- Serum electrolytes and acid-base status parameters: K 3,7, Ca 2,12, Na 140, Cl 100, P 0,94, ASTRUP: pH 7,482, cHCO3 26,8, ABE 3,6.
- Urine analysis: albumin+++, erythrocytes. 3-4; leukocytes: 6-10; BIURET 4,3 g/24h
- Tumor markers: CA 125: 586, CA 15-3: 98, CA 19-9: 1,2, CYFRA 21-1 2,8, CEA: 9,4, NSE: 9,4, HE4 241,5: , ROMA 90,7%.
- Hormone status: TSH 2,75; FT4 17,75; Thyroglobulin: 41,91; calcitonin 0,694;

We conclude that the patient had thrombocytosis, grade III renal insufficiency, normal mineral status, hypoproteinemia and hyperlipidemia, nephrotic range proteinuria, metabolic alkalosis, and elevated tumor markers for the breast and genital tract. Gynaecology ultrasound findings were normal. On the fifth day of hospitalization, the patient's clinical condition sharply deteriorated. as problems, she reported suffocation, chest tightness and dry cough. Objectively dyspnoeic at rest with central cyanosis present, tachycardic heartbeat, quieter tones, no murmur, TA 80 / 60mmHg. ECG showed sinus rhythm, f 80 / min, low voltage in standard and unipolar leads. Oxygen therapy was administered, Dobutamine 5mcg / kg / min (250mg Dobutamine in 250mL 0.9% NaCl), Clexane 0.6 ml 1x1 s.c; blood was taken for D dimer and cardiospecific enzymes.

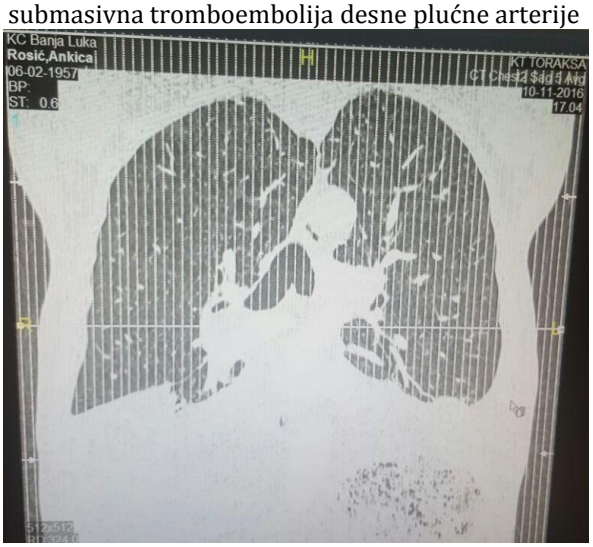
Chest X-ray reveals bilateral pleural effusion (pleural effusion) to the level of the V rib as well as an encapsulated effusion in the projection of the lower lung field to the right (Figure 1).

Figure 1. Chest X-ray (X-ray of the heart and lungs) shows bilateral pleural effusion up to V rib as well as encapsulated effusion in the projection of the lower lung area to the right

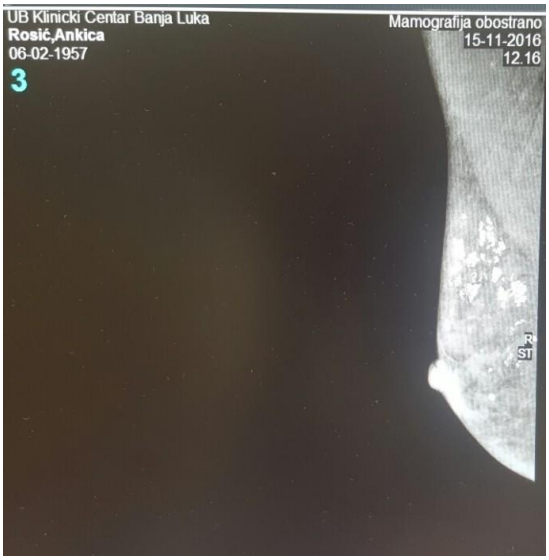


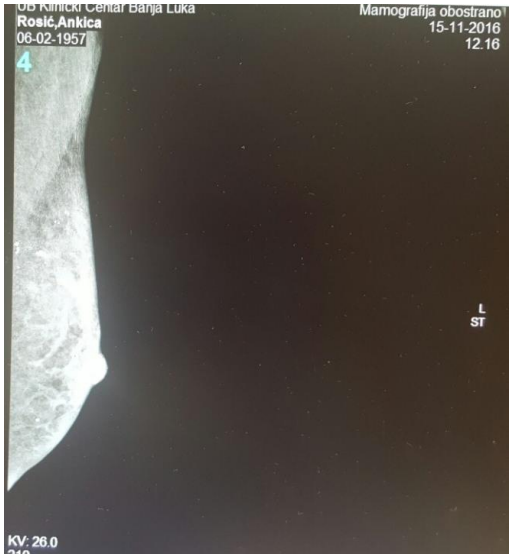
Computed tomography of the thorax (CT) according to the PTE program showed submassive thromboembolism of the right pulmonary artery (Figure 2). The defect in the lumen is differentiated - thromboembolism of the branch of the right pulmonary artery for the upper lung lobus with extension into segmental branches for the anterior segment as well as incomplete thrombosis of the intermediate branch of the right pulmonary artery. Pulmonary artery tree width 23mm, right pulmonary artery 21mm, left pulmonary artery 18mm. There is no consolidation or infiltration in the parenchyma shown. Posterobasal pleural effusion right about 5 cm wide, left up to 6 cm with consequent compressive atelectasis of the basal segments of the lower lung lobes.

Slika 2. Kompjuterizovana tomografija toraksa (CT) po programu za PTE CT toraksa po protokolu za PTE: submasivna tromboembolija desne plućne arterije



Slika 3. i 4. Nativna mamografija obe dojke i aksilarne regije: Prepektoralno obostrano, izraženije u desnoj dojci se uočavaju multiple intraduktalne segmentne kalcifikacije koje u potpunosti ispunjavaju duktuse. Između ovih amorfnih kalcifikacija se uočavaju multiple pojedinačne mikrokalcifikacije suspektno malignih karakteristika

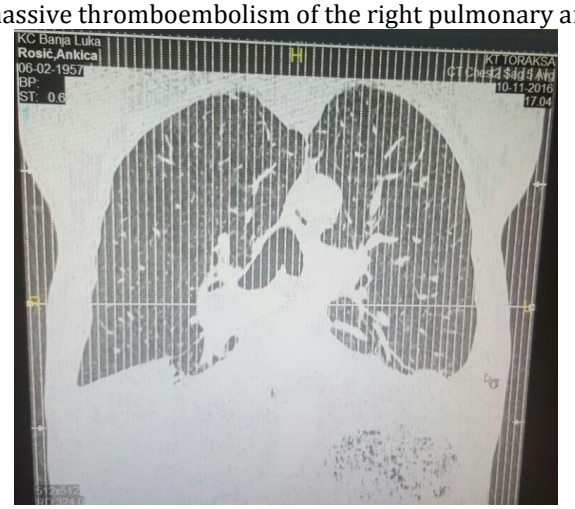




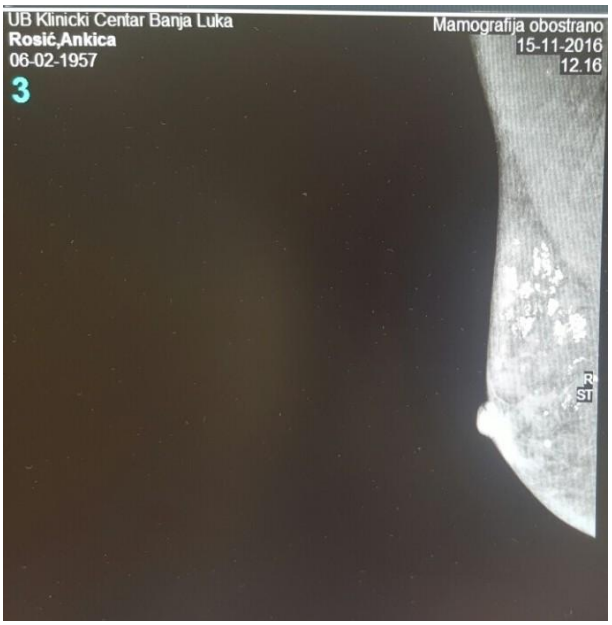
DISKUSIJA:

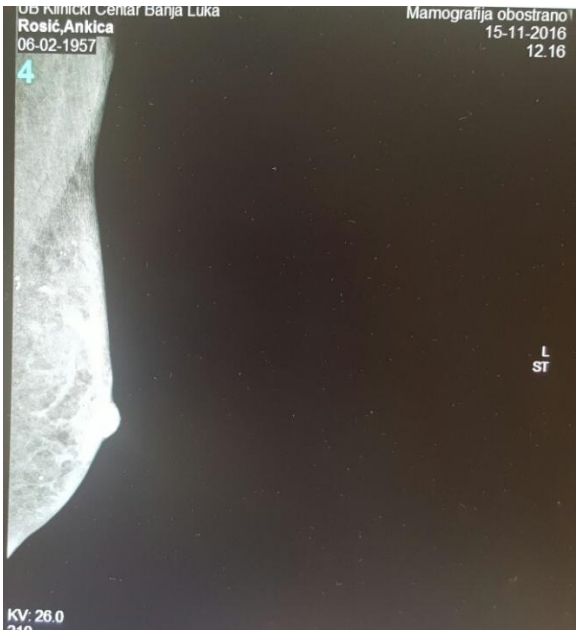
Maligni tumori deluju na organizam lokalno: tumorskom masom, infiltrativnim rastom, destrukcijom lokalnog tkiva, kompresijom, nekrozom, krvarenjem, sekundarnim infekcijama. Sistemski lučenjem hormona i drugih supstanci deluju na udaljene organe i sisteme ili troše gradivne i energetske materije. Paraneoplastični sindrom je grupa kliničkih poremećaja udružena sa malignom bolešću koji nisu posledica direktnih fizičkih efekata primarnog tumora ili metastatske bolesti [1]. Javlja se kod 10-20% onkoloških bolesnika, pre svega kod sitnoćelijskog karcinoma pluća, dojke, jajnika, malignih limfoma a kliničke manifestacije su različite. Nije vezan za veličinu primarnog tumora, može nastati kasno u evoluciji bolesti ili biti prvi znak recidiva bolesti. Precizan mehanizam nastanka paraneoplastičnog sindroma nije jasan,

Figure 2. Chest CT according to the PTE protocol:
submassive thromboembolism of the right pulmonary artery



Figures 3. and 4. Native mammography of both breasts and the axillary region: Bilaterally prepectoral and more pronounced in the right breast, multiple intraductal segmental calcifications are observed, completely filling the ducts. Among these amorphous calcifications, multiple individual microcalcifications of suspected malignant characteristics are observed





DISCUSSION:

Malignant tumors affect the body locally: by their mass, by infiltrative growth, by destruction of local tissue, by compression, through necrosis, bleeding, secondary infections. By systemically secreting hormones and other substances, they affect distant organs and systems or consume building material and energy. Paraneoplastic syndrome is a group of clinical disorders associated with a malignant disease and which are not a result of direct physical effects of the primary tumor or a metastatic disease [1]. It exists in 10-20% of patients, primarily in small cell lung, breast, ovarian cancer, and in malignant lymphomas; clinical manifestations differ. It is not related to the size of the primary tumor, it may occur late in the evolution of the disease or it may be the first sign of disease

pretpostavlja se da je vezana za produkciju biološki aktivnih supstanci od strane tumora (polipeptidni hormoni i citokini) ili produkcijom antitela. Paraneoplastični sindrom obuhvata nespecifične metaboličke i endokrine manifestacije tumora.

Simptomi i znaci paraneoplastičnog sindroma mogu biti:

- **Sistemski:** anoreksija, kaheksija, gubitak u telesnoj masi, temperature, ortostatska hipotenzija.
- **Kožni:** stečena palmoplantarna keratodermija, pemfigus vulgaris, pruritus.
- **Neurološki:** periferna neuropatija, encefalopatija, nekrotizirajuća mijelopatija, retinopatija udružena sa karcinomom, gubitak vida, visceralna neuropatija.
- **Endokrini i metabolički**: nemetastatska hiperkalcemija, lučenje paratireoidnom sličnog hormona (češće kod skvamoznog, mikrocelularni karcinom 10%), Sy. Cushing, hiperkorticizam (mikrocelularni karcinom 1,6-4,5%), sindrom neadekvatnog lučenja antidiuretičkog hormona, ginekomastija i galaktoreja, preterano lučenje gonadotropnog hormona, karcinoid sindrom, hipertireoidizam, hiper i hipoglikemija, hipofosfatemija, hipourikemija.
- **Renalni:** glomerulonefritis, tubulointersticijumska bolest. Hematološki: anemija, leukocitoza i eozinofilija, leukemoidna reakcija, trombocitoza I trombocitopenijska purpura.
- **Koagulopatije:** hiperkoagulabilnost, Troussean-ov sindrom (češći kod adenokarcinoma), tromboflebitis, diseminovana intravaskularna koagulopatija.
- **Kolageno vaskularni:** dermatomiozitis, polimiozitis, vaskulitis, sistemski eritemski lupus.
- **Koštano-zglobni**: batičasti prsti, plućna hipertrofična osteoartropatija (češće kod adenokarcinoma).

U literaturi se opisuje nefrotski sindrom kao direktan uzročnik tromboembolije usled gubitka antitrombotičnih faktora urinom i povećane produkcije protrombotičkog faktora u jetri [2].
Hemodijalizni bolesnici imaju dva puta veću učestalost plućne tromboembolije od bolesnika bez bubrežnog oboljenja, a hemodijalizni bolesnici imaju veću učestalost PTE od bolesnika na peritoneumskoj dijalizi][3. Tome doprinosi infekcija vaskularnog pristupa, septično stanje i upotreba privremenih i trajnih centralnih venskih katetera za hemodijalizu.

ZAKLJUČAK:

Učestalost plućne tromboembolije je potcenjena zbog nepouzdane kliničke slike, dijagnoze i nedovoljno preciznih testova za potrdu klinički suspektne PTE. Potreban je multidisciplanaran pristup lečenju jer je realna incidenca deset puta veća od procenjene. Kod prikazane pacijentkinje se najverovatnije radilo o karcinomu dojke sa sekundarnim depozitima koji je kasno prepoznat. PTE kao vjerovatna posledica paraneoplastičkog nefrotskog sindroma je pravovremeno dijagnostikovana i lečena.
Dobrom saradnjom kardiologa, nefrologa, onkologa i pulmologa je moguć je pravovremeno otrivanje osnovne bolesti, bolje preživljavanje i postizanje boljeg kvaliteta zivota pacijenata.

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recurrence. The exact mechanism of occurrence of paraneoplastic syndrome is not clear, it is assumed to be related to the production of biologically active substances by tumors (polypeptide hormones and cytokines) or the production of antibodies. Paraneoplastic syndrome includes nonspecific metabolic and endocrine manifestations of a tumor.

Symptoms and signs of paraneoplastic syndrome may be:

- **Systemic**: anorexia, cachexia, weight loss, fever, orthostatic hypotension.
- **Dermal**: acquired palmoplantar keratoderma, pemphigus vulgaris, pruritus.
- **Neurological**: peripheral neuropathy, encephalopathy, necrotizing myelopathy, cancer-associated retinopathy, vision loss, visceral neuropathy.
- **Endocrinal and metabolic**: nonmetastatic hypercalcemia, secretion of parathyroid-like hormone (more common in squamous cell carcinoma, microcellular carcinoma 10%), Sy. Cushing, hypercorticism (microcellular carcinoma 1.6-4.5%), the syndrome of inappropriate secretion of antidiuretic hormone, gynecomastia and galactorrhea, excessive secretion of gonadotropic hormone, carcinoid syndrome, hyperthyroidism, hyper and hypoglycemia, hypophosphatemia, hypouricemia.
- **Renal**: glomerulonephritis, tubulointerstitial disease.
- **Haematological**: anemia, leukocytosis and eosinophilia, leukemoid reaction, thrombocytosis and thrombocytopenic purpura.
- **Coagulopathies**: hypercoagulability, Troussean syndrome (more common in adenocarcinoma), thrombophlebitis, disseminated intravascular coagulopathy.
- **Collagen vascular**: dermatomyositis, polymyositis, vasculitis, systemic lupus erythematosus.
- **Bone and joint**: digiti hyppocratic, pulmonary hypertrophic osteoarthropathy (more common in adenocarcinoma).

The literature gives the nephrotic syndrome as a direct cause of thromboembolism due to loss of antithrombotic factors through urine and increased production of prothrombotic factor in the liver [2].
Hemodialysis patients have twice the incidence of pulmonary thromboembolism than patients without renal disease, and hemodialysis patients have a higher incidence of PTE than patients on peritoneal dialysis [3]. Vascular access infection, septic condition and use of temporary and permanent central venous catheters for hemodialysis contribute to this.

CONCLUSION:

The incidence of pulmonary thromboembolism has been underestimated due to unreliable clinical picture, diagnosis, and insufficiently accurate tests which would confirm clinically suspected PTE. What is required here is a multidisciplinary approach to the treatment because the real incidence is ten times higher than estimated. The patient most likely had breast cancer with secondary deposits but one which was detected late. PTE as a probable consequence of paraneoplastic nephrotic syndrome was timely diagnosed and treated.
Timely detection of the underlying disease, better survival and patients' better quality of life are possible through good cooperation of cardiologists, nephrologists, oncologists and pulmonologists.

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