CASE REPORTS

Refer to: Santiago SM Jr, Klaustermeyer WB, Brockman G: Pulmonary thromboembolism in erythema multiforme. West J Med 126:505-506, Jun 1977

Pulmonary Thromboembolism in Erythema Multiforme

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ERYTHEMA MULTIFORME is an acute inflammatory disease with an extremely wide range of clinical presentation. Signs may vary from a few scattered lesions of the skin and mucous membranes to a generalized bullous eruption with severe systemic manifestations.[1] The disease is commonly associated with a number of unrelated conditions. These include bacterial, viral, mycoplasmal, protozoan and mycotic infections; drug reactions; vaccination; contact dermatitis; collagen vascular disease; radiation therapy; internal malignancy, and hormonal changes during pregnancy and menstruation.[2,3] In addition to the cutaneous features of erythema multiforme, there may also be involvement of the respiratory tract, heart, kidneys, gastrointestinal tract and central nervous system.[3] Pulmonary manifestations include tracheobronchitis, pneumonitis, pulmonary consolidation, pleural effusion and hilar adenopathy.[1,4-8] In the following report the case of a patient with recurrent erythema multiforme and pulmonary thromboembolism is described.

Report of a Case

A 29-year-old black man had a history of recurrent episodes of erythema multiforme since 1964 requiring high-dose corticosteroid therapy and frequent admissions to hospital. A typical episode consisted of a maculopapular and vesicular eruption involving the skin of the extremities and mucous membranes of the oral cavity and glans penis accompanied by difficulty in speaking and swallowing. Numerous efforts to identify specific etiologic factors have been nonproductive.

In September 1973 a recurrence of erythema multiforme was accompanied by severe right-sided pleuritic chest pain, shortness of breath and a nonproductive cough. Diminished breath sounds were noted at the right base and a right-sided pleural effusion was seen on roentgenograms of the chest. Thoracentesis yielded a minimal amount of serosanguinous fluid that was sterile on culture. The patient refused further diagnostic studies and gradually improved over a period of several weeks.

The patient was readmitted on February 26, 1976, to the Wadsworth Veterans Administration Hospital in Los Angeles with a five-day history of right-sided pleuritic chest pain, shortness of breath and an acute exacerbation of erythema multiforme. On physical examination, temperature was 38.3°C (101°F); pulse rate, 104 beats per minute; respiratory rate, 22 per minute, and blood pressure, 110/90 mm of mercury. Vesicles and erosions were noted on the lips, buccal mucosa, tongue, hands and feet. Increased fremitus in the right mid-posterior portion of the chest and accentuation of the second pulmonic heart sound were noted on auscultation. Laboratory findings included a hemoglobin level of 14 grams per 100 ml, leukocyte count of 13,300 cells per cu mm and no abnormalities on an SMA-12 (Sequential Multiple Analysis). Arterial blood gas studies done on admission showed an oxygen pressure of 76 mm of mercury, a carbon dioxide pressure of 35 mm of mercury and a pH of 7.43. An infiltrate was noted in the right mid-lung field on roentgenograms of the chest and a perfusion lung scan with technetium Tc 99m-labeled macroaggregated albumin (^{99m}Tc-MAA) showed perfusion defects in the right upper and lower lobes and lingula. On pulmonary angiograms a filling defect was noted in the right main pulmonary artery with absence of flow to the right upper lobe (Figure 1). The patient was treated with heparin and maintenance prednisone (40 mg per day) was continued. Clini-

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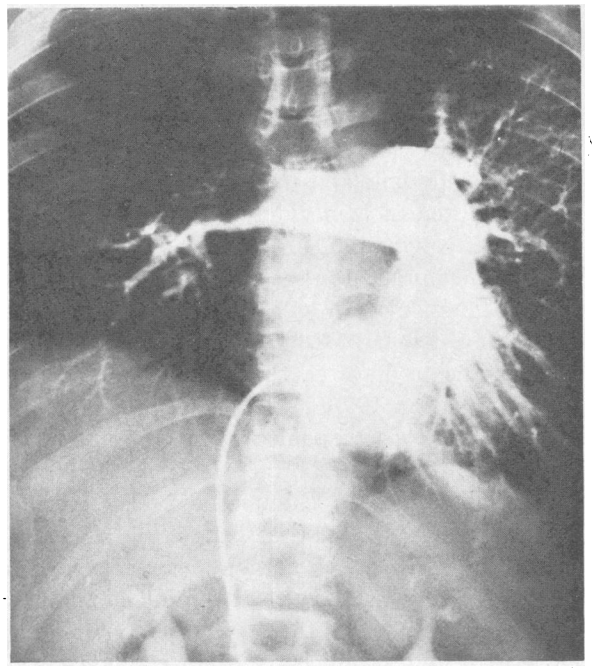

Figure 1.—Pulmonary angiogram showing filling defect in right main pulmonary artery and absence of flow to the right upper lobe.

cal improvement occurred in two days with reduction of the fever and chest pain. Radionuclide venograms (using ^{99m}Tc-MAA) of the lower extremities done a week after admission were normal. The skin and mucous membrane lesions gradually resolved and the patient was discharged on a regimen of sodium warfarin (Coumadin®) and prednisone after two weeks in hospital.

Discussion

The precise pathogenesis of erythema multiforme has not been firmly established. Its occurrence after exposure to a variety of unrelated agents may suggest an immune mechanism and histopathologic findings of allergic vasculitis would tend to support this.[9] Vasculitis may account for some of the reported manifestations of erythema multiforme including glomerulonephritis and microangiopathic hemolytic anemia.[10,11] The pulmonary vascular occlusion in our patient may likewise be a manifestation of the vasculitis of erythema multiforme. He had one documented episode of pulmonary thromboembolism that occurred coincidentally with an exacerbation of erythema multiforme. A previous bout of acute erythema multiforme in September 1973 was also accompanied by features suggestive of pulmonary thromboembolism (pleuritic chest pain, dyspnea and serosanguinous pleural effusion). To our knowledge, pulmonary thromboembolism has not been reported in erythema multiforme. It is possible that some of the previously reported cases of "pneumonitis" in erythema multiforme are secondary to pulmonary thromboembolic disease.

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Refer to: Seltzer RA: Pneumoperitoneum following intermittent positive pressure breathing. West J Med 126:506-509, Jun 1977

Pneumoperitoneum Following Intermittent Positive Pressure Breathing

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THERE ARE SEVERAL reports of pneumoperitoneum secondary to progressive pneumomediastinum in neonates with respiratory distress syndrome or chronic pulmonary disease.[1-4] Recently, this phenomenon has been described in three adults receiving mechanical ventilation, in two of whom exploratory laparotomy was done for suspected perforated viscus.[5] The following report is the

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