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REFERENCES

1. STARLING, E. H.: *J. Physiol.*, 19: 312, 1896.
2. WELLS, H. S., YOUMANS, J. B. AND MILLER, D. G., JR.: *J. Clin. Invest.*, 12: 1103, 1933.
3. WEECH, A. A.: *Intern. Clin.*, 2: 223, 1936.
4. SHWACHMAN, N. AND GAHM, N.: Studies in cystic fibrosis, In: Year Book of Pediatrics, 1957-1958, edited by S. S. Gellis, Year Book Publishers, Inc., Chicago, Ill., 1957, p. 190.
5. TREVARROW, V. et al.: *J. Lab. & Clin. Med.*, 27: 471, 1942.
6. NELSON, W. E., ed.: Textbook of pediatrics, 6th ed., W. B. Saunders Company, Philadelphia, 1954, pp. 308, 309, 316.
7. LUMB, G. AND BEAUTYMAN, W.: *J. Path. & Bact.*, 64: 679, 1952.
8. ILLINGWORTH, R. S. AND FINCH, E.: *Arch. Dis. Childhood*, 29: 507, 1954.
9. BILLE, B. S. V. AND VAHLQUIST, B.: *Acta pædiat.*, 44: 435, 1955.
10. MYERS, W. K. AND TAYLOR, F. H. L.: *J. A. M. A.*, 101: 198, 1933.
11. COPE, C. L. AND GOADBY, H. K.: *Lancet*, 1: 1038, 1935.
12. BINGER, M. W. AND KEITH, N. M.: *J. A. M. A.*, 109: 1, 1937.
13. JOHANSEN, A. H.: *Acta path. et microbiol. scandinav.* (suppl.), 37: 272, 1938.
14. RYTAND, D. A.: *Arch. Int. Med.*, 69: 251, 1942.
15. MESSINGER, M. J.: *Canad. M. A. J.*, 55: 381, 1946.
16. ALBRIGHT, F., BARTTER, F. C. AND FORBES, A. P.: *Tr. A. Am. Physicians*, 62: 204, 1949.
17. HOMBURGER, F. AND PETERMANN, M. L.: *Blood*, 4: 1085, 1949.
18. THOMPSON, W. H., MCQUARRIE, I. AND BELL, E. T.: *J. Pediat.*, 9: 604, 1936.
19. SCHICK, B. AND GREENBAUM, J. W.: *Ibid.*, 27: 241, 1945.
20. HERTZOG, F. V. AND FAUST, O. A.: *Ibid.*, 36: 641, 1950.
21. WYNGAARDEN, J. B. et al.: *Pediatrics*, 9: 729, 1952.
22. ILLINGWORTH, R. S. AND FINCH, E.: *Arch. Dis. Childhood*, 29: 513, 1954.
23. FRIED, C. T. AND HENLEY, W. L.: *Pediatrics*, 14: 59, 1954.
24. STURGEON, P. AND BRUBAKER, C.: *A.M.A. Am. J. Dis. Child.*, 92: 254, 1956.
25. LAHEY, M. E. AND SCHUBERT, W. K.: New deficiency syndromes occurring in infancy, In: Bulletin of the 26th annual meeting of the Society of Pediatric Research, Buck Falls, Penn., May 7 and 8, 1956, Report No. 25.
26. ULSTROM, R. A., SMITH, N. J. AND HEIMLICH, E. M.: *A.M.A. Am. J. Dis. Child.*, 92: 219, 1956.
27. SCHWARTZ, M. AND THOMSEN, B.: *Brit. M. J.*, 1: 14, 1957.
28. ALTMANN, A. AND MURRAY, J. F.: *South African J. M. Sc.*, 13: 91, 1948.
29. TALBOT, N. B., CRAWFORD, J. D. AND COOK, C. D.: *New England J. Med.*, 256: 1080, 1957.
30. HARPER, H. A.: Review of physiological chemistry, 6th ed., Lange Medical Publications, Los Altos, Cal., 1957, p. 324.

RÉSUMÉ

Il s'agit d'un garçon de 4 ans de nationalité italienne, présentant une hypoprotéinémie temporaire acquise non expliquée avec œdème et anémie hypochrome microcytaire.

A l'admission, de l'œdème, de l'oligurie, de l'hépatomégalie et un certain degré d'ascite furent constatés; ces symptômes sont apparus à la suite d'une amygdalite aiguë survenue une semaine auparavant.

Hypertension, atteinte rénale, protéinurie et hématurie étaient présentes à un degré minime à l'admission, mais disparurent peu de temps après.

Le taux de protéines plasmatiques revint à la normale au bout de trois semaines, grâce à une infusion de sang total et une de plasma, et à l'administration de cortisone.

Actuellement cet enfant dix mois après la guérison de cette maladie jouit d'une excellente santé, avec une diète ordinaire. L'hémoglobine et le taux de protéines sanguines demeurent normaux.

Un examen du taux de protéines sanguines fut pratiqué chez les autres membres de sa famille et le résultat fut normal.

Il semble s'agir d'un cas inusité de perturbation du métabolisme des protéines plasmatiques. Plusieurs diagnostics furent envisagés entre autre, l'œdème de carence nutritionnelle, la dysfonction pancréatique, la cirrhose hépatique, la péricardite constrictive, la néphrite aiguë, la néphrose, mais cependant l'hypoprotéinémie idiopathique acquise semblait être le plus plausible.

GINGIVITIS CAUSED BY
HISTOPLASMA CAPSULATUM

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THIS CASE OF HISTOPLASMOSIS is being reported because it demonstrates again the presence of the disease in Quebec.⁵

The patient, a painter 57 years of age who had lived all his life in the Montreal area, was admitted to a local hospital in February 1954. He complained of headaches and gastro-intestinal symptoms associated with fresh and altered blood in the stool. These signs and symptoms had been present for almost ten years, during which time the patient had not worked at all.

Examination revealed the following pertinent facts.

Blood Wassermann was negative. Examination of urine did not reveal anything of note. Hæmoglobin was 113%. White blood cell count was 12,750 per c.mm. with a normal differential. The details of the physical examinations are not available, except that the patient's blood pressure, at this time, was 100/80 mm. Hg.

Chest plate showed a diffuse shadow on both upper halves of the lungs. This shadow was more evident on the right side. There was an opacity in the apex area of the right lung with a dense nodule, 2 cm. in diameter, located in the subclavicular area. No diagnosis of these lesions was made. A barium meal demonstrated an incompletely healed duodenal ulcer.

The patient was treated for peptic ulcer and nervous depression, and left the hospital three weeks later. His condition was said to have improved.

He saw his family physician in January 1956 for complaints of vomiting and increased weakness. He was then raising large amounts of sputum and stated that he had not taken any appreciable amount of food during the previous two weeks. Examination of the oral cavity revealed the presence of a mucosal lesion which was said to have been present for six to eight weeks. The physician thought this lesion to be a neoplastic growth and sent the patient to the Institut du Radium.

On admission, an ulcer, 2 cm. long and 1 cm. wide, was found at the right side of the upper gum (Fig. 1). A biopsy was taken on which the Institut d'Anatomie Pathologique de l'Université de Montréal reported as follows:

"The biopsy of the mucous membrane of the gum is very small and badly traumatized. The portions which lend themselves to microscopic examination are minute. The cutis below the rete Malpighii of the gum is infiltrated with inflammatory elements, mainly mononucleated cells, histiocytes and plasmacytes. Some multinucleated giant cells with marginal or peripheral nuclei are present. These cells are scattered without any order and without formation of groups suggesting tuberculous follicles.

"The cytoplasm of these giant cells is more or less filled with oval inclusions of equal size. These inclusions have well-defined outlines. Some appear as vacuoles without stainable content whereas other ones show

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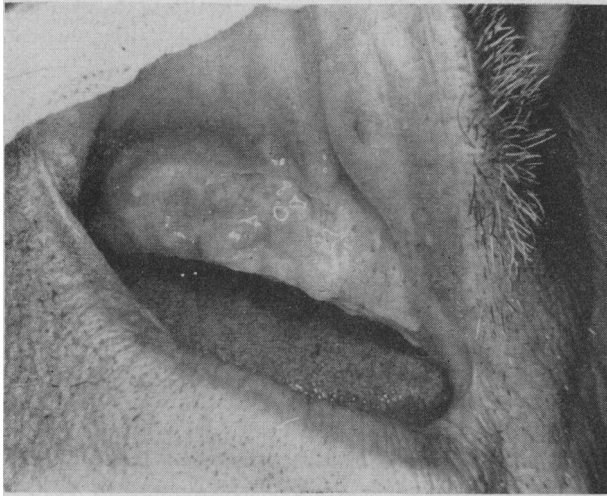


Fig. 1

minute basophilic granulation. All these inclusions are stained deep red by the Gridley technique. All these features allow one to regard the inclusions as parasites belonging to the *Histoplasma* group."

In the light of this report, a new biopsy was taken and inoculated on different media. Growth at room temperature became visible after five days. Microscopic examination of these cultures obtained on Sabouraud glucose agar seven days after inoculation showed the presence of many round and oval chlamydospores diagnostic of *Histoplasma capsulatum*.

The patient was then discharged from the Institut du Radium and entered another hospital in April 1956. Surgical removal of the mucosal lesion was considered; however, permission was not obtained and the patient left the hospital one week later. He was seen at the Outdoor Clinic on April 27 and May 22, 1956. He had lost weight and his blood pressure had declined from 125/80 to 92/80 during the time between these two visits. His skin had become bronze-coloured. The patient died at home on July 10, 1956. No autopsy was performed.

In retrospect, the question may be raised whether or not the lesion in the mouth, leading to the diagnosis of histoplasmosis, might have been associated with a generalized form of this disease as has often been reported.¹⁻¹² The diagnosis of generalized histoplasmosis could also explain a number of signs and symptoms observed in this patient.

Since no search for *Histoplasma capsulatum* in other clinical specimens was made and autopsy was not performed, this question remains unanswered.

REFERENCES

1. BAUM, G. L. *et al.*: *A.M.A. Arch. Dermat.*, 76: 4, 1957.
2. CURTIS, C. A. AND GREKIN, J. N.: *J. A. M. A.*, 134: 1217, 1947.
3. GAMMELL, E. B. AND BRECKENRIDGE, R. L.: *Ann. Otol. Rhin. & Laryng.*, 58: 249, 1949.
4. HULSE, W. F.: *A.M.A. Arch. Otolaryng.*, 54: 65, 1951.
5. MANKIEWICZ, E., BLANK, F. AND RUBIN, J. H.: *Canad. M. A. J.*, 71: 386, 1954.
6. MURRAY, J. F. AND BRANDT, F. A.: *Am. J. Path.*, 27: 783, 1951.
7. POLES, F. C. AND LAVERTINE, J. D. O'D.: *Thorax*, 9: 233, 1954.
8. ROBERTS, S. E. AND FORMAN, F. S.: *Ann. Otol. Rhin. & Laryng.*, 59: 809, 1950.
9. SCHULZ, D. M.: *Am. J. Clin. Path.*, 24: 11, 1954.
10. SIMSON, F. W. AND BARNETSON, J.: *J. Path. & Bact.*, 54: 299, 1942.
11. VAN PERNIS, P. A., BENSON, M. E. AND HOLINGER, P. H.: *Ann. Int. Med.*, 18: 384, 1943.
12. WEED, L. A. AND PARKHILL, E. M.: *Am. J. Clin. Path.*, 18: 130, 1948.

RUPTURED ANEURYSM OF THE SINUS OF VALSALVA*

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ANEURYSMS of the sinuses of Valsalva are a rare cardiac lesion and in the majority of instances are due to a congenital defect, but occasionally may result from luetic aortitis or possibly bacterial endocarditis. Rupture of these aneurysms into the right side of the heart until recently has been a pathological diagnosis, but now it is realized that such a rupture produces a characteristic clinical picture, which is being recognized with increasing frequency. The importance of early recognition of a ruptured sinus aneurysm lies in the fact that without operation the outlook is grave, whereas with the technique of open heart surgery, made possible by either the heart-lung pump¹ or hypothermia,² the lesions can be corrected and the patient returned to a normal life. Herein is reported a case of rupture of a congenital aneurysm of the right coronary sinus of Valsalva into the right ventricle. The correction of this under hypothermia represents one of the early surgical successes in this condition and probably the first instance in which this condition has been corrected in this country.

PATHOLOGICAL AND CLINICAL ASPECTS OF THE CONDITION

The following deals only with the congenital aneurysms of the sinuses of Valsalva. Edwards and Burchell³ have demonstrated pathologically that the defect leading to the development of a congenital aneurysm of a sinus of Valsalva is a lack of continuity between the aortic media and the annulus fibrosus of the aortic valve (Fig. 1). With the impact of the aortic pressure, the weakened base of the sinus develops an aneurysmal sac which usually points either into the right atrium or the right ventricle. In a review of the world's literature, Sawyers,³ in 1957, was able to collect 37 autopsy-proven cases in which the aneurysm had ruptured. In 26 of these (70%) the aneurysm arose from the right coronary sinus of Valsalva and of the 26, 19 (70%) ruptured into the right ventricle. Three of the 26 aneurysms ruptured into the right atrium, one into the left ventricle, one into the pulmonary artery, one into the pericardium, and one into both the right atrium and the right ventricle. Eleven cases arose from the non-coronary sinus of Valsalva and all of these ruptured into the right atrium. No case has been reported of rupture of a congenital aneurysm of the left coronary sinus of Valsalva.

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