Underrecognition of chylomicronemia as a cause of acute pancreatitis

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Objective: To determine how often chylomicronemia is considered by admitting physicians as a possible cause of acute pancreatitis.
Design: Retrospective hospital chart review.
Setting: Tertiary care teaching hospital in an urban centre with a referral population of 1 million.
Patients: All patients admitted with acute pancreatitis from Jan. 1, 1985, to Dec. 31, 1987. Episodes of pancreatitis were divided into two groups: those for which a cause was known after history taking, physical examination and laboratory investigation at the time of admission (group 1) and those for which a cause was unknown after full examination (group 2).
Results: There were 319 episodes of chylomicronemia in 162 patients. The cause of the pancreatitis was known after examination at the time of admission in 239 (75%) of the episodes; there was hypertriglyceridemia in 7 (3%). No cause was identified after examination in the other 80 episodes (25%); chylomicronemia was considered in 18 cases (29%) and was found in 6 (33%) of them (mean serum triglyceride level 34.4 mmol/L). Of the remaining 62 episodes in group 2, 10 (16%) were later found to be caused by chylomicronemia (mean serum triglyceride level 22.6 mmol/L). Among the 80 episodes in group 2 at least one medical condition associated with chylomicronemia was present in 24. In only 7 (29%) of the 24 was chylomicronemia considered; in 6 the mean serum triglyceride level was 19.7 mmol/L.
Conclusions: Although the overall detection rate of chylomicronemia was low, its presence in patients without other etiologic factors after examination may have been much higher. Consideration of chylomicronemia in this subgroup at the time of presentation may increase diagnostic yield and help prevent further occurrences of pancreatitis.

Objectif : Préciser à quelle fréquence l'hyperchylomicronémie est envisagée par les médecins de l'admission en tant que cause possible de pancréatite aiguë.
Conception : Étude rétrospective des dossiers d'hospitalisation.
Cadre : Hôpital d'enseignement de soins tertiaires d'un centre urbain avec une population consultante de 1 million de personnes.
Patients : Tous les patients admis du 1er janvier 1985 au 31 décembre 1987 et souffrant d'une pancréatite aiguë. Les crises de pancréatite ont été réparties en deux groupes : celles dont la cause était connue après établissement des antécédents médicaux, l'examen physique et les analyses de laboratoire lors de l'admission (groupe 1) et celles dont la cause était inconnue après examen complet (groupe 2).
Résultats : On a relevé 319 crises d'hyperchylomicronémie chez 162 patients. La cause

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de la pancréatite était connue après l'examen au moment de l'admission dans 239 (75 %) des cas; on a constaté une hypertriglycéridémie chez 7 patients (3 %). Dans les 80 autres crises (25 %), aucune cause n'a été dégagée après examen; l'hyperchylomicronémie a été envisagée dans 18 cas (29 %) et observées dans 6 (33 %) d'entre eux (taux sérique moyen de triglycérides de 34,4 mmol/L). Parmi les 62 autres crises dans le groupe 2, on a découvert ultérieurement que 10 (16 %) avaient pour cause l'hyperchylomicronémie (taux sérique moyen de triglycérides de 22,6 mmol/L). Dans les 80 crises du groupe 2, au moins une pathologie médicale associée à l'hyperchylomicronémie était présente dans 24 cas. Chez 7 seulement (29 %) de ces 24 cas, on a envisagé l'hyperchylomicronémie; dans 6 cas, le taux sérique moyen de triglycérides était de 19,7 mmol/L.

Conclusions : Même si le taux global de dépistage de l'hyperchylomicronémie était faible, sa présence chez les patients sans autre facteur étiologique après examen était peut-être beaucoup plus élevée. Envisager l'existence de l'hyperchylomicronémie dans ce sous-groupe au moment de la présentation peut augmenter l'efficacité du diagnostic et aider à prévenir d'autres cas de pancréatite.

The incidence rate of acute pancreatitis is estimated to be between 10 and 30 per 100 000 population.[1] The clinical diagnosis relies on a combination of history taking, physical examination and laboratory investigation. Abdominal pain radiating to the back as well as high serum and urine levels of amylase point toward acute pancreatitis.[2] The causes of pancreatitis are numerous, alcohol abuse, biliary lithiasis and obstruction by tumour being the most common. When these factors are absent, other possible causes include drugs, viral infection and chylomicronemia.[3,4]

Acute pancreatitis is associated with pain, severe illness and death. Recent epidemiologic reports have shown that despite advances in clinical medicine the death rate of 20% has not changed since the 1940s.[1] Therefore, if patients at risk could be identified and the causative conditions treated, the incidence of pancreatitis and the corresponding death rate might decrease.

The proportion of cases of acute pancreatitis caused by chylomicronemia is unknown. Estimates have varied from 4% to 53%.[5] We suspect that chylomicronemia is not considered often enough. We performed a retrospective study to estimate how frequently chylomicronemia was considered by physicians as a cause of pancreatitis, especially in the absence of alcoholism and features of pancreatic duct obstruction and in the presence of secondary causes of chylomicronemia.

Methods

All medical records at the Ottawa Civic Hospital with the diagnostic code for pancreatitis were reviewed. The hospital is a tertiary care teaching hospital with a referral population of 1 million. The period under review was Jan. 1, 1985, to Dec. 31, 1987.

Several patients had more than one episode of acute pancreatitis. Since we wanted to determine the frequency of consideration of chylomicronemia in all presentations, we counted episodes rather than patients.

Verification of diagnosis

An episode was included if the following criteria were documented: (a) history of an acute onset of constant epigastric pain radiating to the back; (b) history of nausea or vomiting associated with the pain; (c) abdominal tenderness on physical examination; (d) presence of at least two of high serum level of amylase, high urine level of amylase, high serum level of lipase and low serum level of calcium; and (e) ultrasonographic or computed tomographic evidence of pancreatic inflammation.

Classification of episodes

After carefully examining the charts of all verified episodes of pancreatitis we were able to classify the episodes into two groups. In group 1 the physician was aware of at least one etiologic factor (e.g., history of alcohol abuse or biliary obstructive disease) during the initial evaluation. Group 2 comprised episodes in which the cause was unidentified even after the history taking and physical examination.

Analysis of consideration of chylomicronemia

All episodes were examined for evidence of consideration of chylomicronemia. This was considered to have occurred if the physician had mentioned chylomicronemia as a possible cause in the admitting note or had ordered the serum triglyceride level to be measured on admission. The laboratory at the Ottawa Civic Hospital does not include such measurement in the screening biochemical profile, so its ordering was considered evidence that chylomicronemia had been considered a cause. A serum

triglyceride level of more than 10.0 mmol/L was strongly supportive of a diagnosis of chylomicronemia.

Group 2 was of greatest interest, because in the absence of an obvious etiologic factor the admitting physician would be reasonably expected to investigate other causes. Episodes in group 2 were examined for the presence of conditions that predispose to chylomicronemia (diabetes mellitus, obesity, chronic alcohol consumption, hypothyroidism and nephrotic syndrome) to determine whether any of them had prompted the physician to think of chylomicronemia.

Results

We reviewed 162 charts documenting 319 verified episodes of acute pancreatitis. An obvious etiologic factor was identified in 239 (75%) of the episodes (in 117 patients); a history of alcohol abuse was documented in 176 episodes, a history of biliary lithiasis in 55, a history of elevated serum triglyceride levels in 7 and cancer of the pancreas in 1. Apart from the 7 episodes in which hypertriglyceridemia was documented, chylomicronemia was considered as a possible cause of the pancreatitis in 13 episodes but was not found in any of them.

In 80 (25%) of the episodes (in 45 patients) no cause was identified initially. Chylomicronemia was considered in 18 cases and found in 6. In these six episodes the mean serum triglyceride level on admission was 34.4 (extremes 2.9 and 66.0) mmol/L. Of the 62 episodes in which chylomicronemia was not considered on admission, the condition was subsequently diagnosed during the hospital stay in 10. The mean serum triglyceride level 4 days after admission in these 10 episodes was 22.6 (extremes 8.7 and 32.7) mmol/L. Of the 80 episodes in this group at least one medical condition predisposing to chylomicronemia was documented in 24. In only 7 of the 24 was chylomicronemia considered; in 6 of the 7 the mean serum triglyceride level was 19.7 (extremes 8.9 and 26.7) mmol/L.

Discussion

Of the 319 episodes 23 (7%) were due to chylomicronemia. This incidence rate is similar to that in another series.[5] In our study it should be considered the minimum, since chylomicronemia was considered at the time of admission in only 31 (10%) of 312 episodes and may have been missed completely throughout the hospital stay.

It is important to consider chylomicronemia at the time of admission, because chylomicrons clear from the blood within a few days when a patient is not fed, which is standard practice in the treatment of acute pancreatitis. The serum triglyceride level might be only slightly elevated or even normal a few days after admission, and this would result in the incorrect conclusion that chylomicronemia was not responsible for the pancreatitis.

In group 1 it may be argued that it is unreasonable to consider chylomicronemia in the presence of another etiologic factor. However, in group 2 no etiologic factor was readily apparent, and yet chylomicronemia was considered in only 18 (22%) of 80 episodes at the time of admission. The significance of this figure becomes more apparent when one realizes that chylomicronemia was subsequently diagnosed in 33% of the episodes in which it had been suspected and in 16% of the remaining episodes in which it had not been considered; perhaps it was present in several more cases but was missed. The obvious clustering of chylomicronemia in group 2 is not surprising; the other causes of pancreatitis such as alcohol abuse and biliary obstruction reveal themselves more readily, and thus their absence should increase one's suspicion of chylomicronemia.

The presence of one or more medical conditions known to predispose to chylomicronemia did not seem to have a strong prompting effect on the admitting physician. The rate of consideration was only 29% (7 of 24 episodes), which is only slightly higher than the overall rate of 22% (18 of 80). This suggests that the general awareness of chylomicronemia is low and that the inclusion of this condition in the differential diagnosis of acute pancreatitis is infrequent.

Our data indicate that there might be room for clinical judgement on whether to look for chylomicronemia in the presence of another etiologic factor. However, chylomicronemia can easily be detected without cost to the health care system: one simply needs to stand the serum or plasma overnight in the refrigerator. Once diagnosed, chylomicronemia can be controlled through diet and drug therapy in most cases, and further episodes of pancreatitis can be prevented; this would save expensive hospital care. Thus, we believe that chylomicronemia should be considered in all cases of pancreatitis.

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